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Trajectories of lung recovery following COVID-19 in a prospective multicentric cohort study: Impact of sleep apnea and its treatment by continuous positive airway pressure

Sébastien Baillieul[a], Lucile Sesé[b,c], Laurent Boyer[d], Bruno Chenuel[e,f], Philippe Romand[g], Carole Planes[b], Geneviève Derumeaux[d], Sam Bayat[h], Marie Destors[a], Thi Hong Van Ngo[a], François Arbib[i], Mathias Poussel[e,f], Edem Allado[e,f], Thomas Gille[b], Jean-Louis Pépin[a*] and Renaud Tamisier[a*]

[a]Grenoble Alpes University, HP2 Laboratory, INSERM U1300, Grenoble Alpes University Hospital, Grenoble, France; [b]Univ. Sorbonne Paris Nord, UFR SMBH Bobigny, INSERM, Hôpital Avicenne, HUPSSD, AP-HP, Bobigny, France; [c]Department of Public Health, Aarhus University, Aarhus, Denmark; [d]Service d'Explorations fonctionnelles, Univ. Créteil, INSERM, CHU Hôpital Henri Mondor, AP-HP, Créteil, France; [e]CHRU-Nancy, Explorations Fonctionnelles Respiratoires-Centre Universitaire de Médecine du Sport et Activité Physique Adaptée, Nancy, France; [f]UR DevAH, Université de Lorraine, Nancy, France; [g]Service de pneumologie, hôpitaux du Leman, Thonon-Les-Bains, France; [h]Department of Pulmonology and Physiology, Grenoble Alpes University, ESRF, INSERM UA07, Grenoble Alpes University Hospital, Grenoble, France; [i]SHU Pneumologie-Physiologie, Grenoble Alpes University Hospital, Grenoble, France

ABSTRACT

Background: Obstructive sleep apnoea (OSA) is associated with more severe forms of COVID-19. We investigated the impact of OSA and its treatment on lung recovery trajectories over the year following acute care for different COVID-19 severities.

Methods: Prospective multicentric (5 centres) cohort study, recruiting from June 2020 to April 2022. All patients with a non-fatal pulmonary presentation of COVID-19 were eligible. OSA diagnosis (apnoea-hypopnoea index [AHI] >5/h) was performed at inclusion. Pulmonary function tests, including haemoglobin-corrected diffusing capacity of the lung for carbon monoxide (DLCO-Hb, expressed as % predicted), the primary endpoint, were conducted at 3 months and repeated at 6 and 12 months post-COVID-19 diagnosis only for patients with persisting abnormal values (conditional monitoring approach). Mixed models with subjects as a random effect were performed to assess the impact of OSA severity and treatment on DLCO-Hb recovery trajectories.

Results: Three hundred and eighteen patients were included in the cohort, 102 (32.1%) requiring outpatient care, and 216 (67.9%) requiring hospitalisation. OSA was not present in 75 (23.6%) patients, newly diagnosed or not treated in 203 (63.8%) and treated by continuous positive airway pressure in 40 (12.6%). 53.3% of tested patients presented a DLCO-Hb ≥80% of predicted values at 3 months. This proportion was significantly lower in non-treated OSA patients at 6 months ($p < 0.01$), but not at 12 months ($p = 0.07$), indicating comparable recovery. In multivariable analysis, OSA severity and its treatment but not COVID-19 severity significantly influenced the evolution of DLCO-Hb.

Conclusion: OSA severity and treatment affect the initial trajectories of lung recovery following COVID-19.

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KEYWORDS
Sleep apnoea, obstructive; COVID-19; respiratory function tests; continuous positive airway pressure

Introduction

The emergence of the severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2), and the resulting coronavirus disease 2019 (COVID-19) pandemic has led to unprecedented challenges to global public health,[1] with an estimated more than 600 million cases worldwide and at least 6.6 million deaths.[2] Beyond acute mortality,[3,4] it is estimated that 7%–54% of COVID-19 survivors may develop post-acute sequelae of SARS-CoV-2 infection,[5] a broad condition covering multiple and various associations of symptoms or sub-phenotypes.[6] Despite progress and improvements in acute respiratory care, accumulating evidence suggests that COVID-19 may exert long-lasting effects on lung function, with COVID-19 survivors requiring prolonged follow-up due to delayed pulmonary

CONTACT Renaud Tamisier rtamisier@chu-grenoble.fr
*Co-senior authors.

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recovery with persistent symptoms and functional impairment.[2] Pulmonary sequelae include remaining and relapsing symptoms, such as cough and shortness of breath,[7] as well as functional and structural pulmonary impairments, including chest imaging abnormalities in more than half of COVID-19 survivors[5] and abnormal gas exchange.[2,8] Age, sex, and comorbidities have been identified as modulating the risk of post-acute sequelae of SARS-CoV-2 infection.[5,7,9] Among these comorbidities, obstructive sleep apnoea (OSA) has been identified as playing a crucial role in COVID-19 outcomes.[9–14]

OSA is a major public health problem, affecting nearly one billion people worldwide,[15] and is considered an independent risk factor for several cardiovascular and metabolic comorbidities.[16] The repeated partial (hypopnoea) or complete (apnoea) collapses of upper airways occurring during sleep lead to a cascade of detrimental consequences, such as intermittent hypoxia, sleep fragmentation, autonomic activation, and systemic inflammation, which underlies the pathophysiological consequences of OSA.[17]

Beyond the association between OSA and an increased risk for fatal COVID-19,[10–12] OSA is growingly acknowledged as a risk factor for the development of post-acute sequelae of SARS-CoV-2 infection.[9,13,14,18] Mandel HL. et al., based on the analysis of electronic health record data from multiple research networks (n = 1,783,940), showed that adults with pre-existing OSA had significantly elevated odds of probable post-acute sequelae of SARS-CoV-2 infection.[9] Focusing on lung function, Labarca G. et al. identified significantly higher rates of altered diffusing capacity of the lungs for carbon monoxide (DLCO) one year after COVID-19 infection among COVID-19 survivors with versus without pre-existing comorbid OSA.[14] If available evidence consistently shows a negative impact of OSA on COVID-19 outcomes even one year after diagnosis, the impact of OSA treatment by continuous positive airway pressure (CPAP), the first-line treatment of OSA, has never been assessed.

We aimed to investigate the impact of OSA and its treatment on lung recovery trajectories over the year following acute care for different COVID-19 severities.

Materials and methods

Study design

'COVID-19: Respiratory and Sleep Follow-up' (Co-SURVIVORS) is a French prospective multicentric cohort study, recruiting from June 2020 to April 2022. All recruiting centres were public universities and general hospitals located in France (Alpes-Leman, Bobigny, Créteil, Grenoble, and Nancy), with Grenoble Alpes University Hospital as the coordinating centre.

The study was approved by an independent ethics committee (Comité de Protection des Personnes Ouest VI, CPP1285 HPS2 IDRCB: 2020-A01181-38, RIPH: 20.04.29.32621) and registered on ClinicalTrials.gov (NCT04406324 – Date of registration: 2020/05/28). This study was conducted following applicable good clinical practice requirements in Europe, French law, ICH E6 recommendations, and the ethical principles of the Helsinki Declaration. External data quality control was performed systematically for the following criteria: informed consent and case report forms. All included individuals signed written informed consent forms before enrolment in the study.

Study participants

All patients with a non-fatal pulmonary presentation of COVID-19 were eligible, whatever the initial severity, from outpatient care to intensive care unit (ICU) hospitalisation.

Patients were recruited by an investigating doctor from each centre, depending on initial severity as follows: i) by phone call for patients who have been positively tested for COVID-19 at the centre and who have benefitted from outpatient care; ii) on discharge from hospital for patients hospitalised in a conventional care unit (infectious medicine department, COVID unit, pneumology department); iii) on discharge from hospital for patients admitted to ICU.

Patients were included in the cohort if they were aged >18 years, 3 months (±30 days) from the date of the positive diagnosis for SARS-CoV-2 (confirmed by RT-PCR in the presence of respiratory symptoms), and were affiliated with a French social and health insurance system or equivalent. Pregnant or

breastfeeding women, individuals with unstable psychiatric disorders, dementia, or an unstable medical condition, prisoners or patients who require protection by the law, and individuals with a diagnosis of infection by other pathogens than SARS-COVID-19 were not included, as well as those who declined to participate.

Assessments

Clinical assessment

All patients underwent a medical examination at inclusion performed by a senior pulmonologist. Anthropometric parameters were measured (height, weight, BMI calculation [kg/m^2]). Demographic parameters, comorbidities, past medical history, tobacco and alcohol consumption, and current medications and clinical manifestations during the acute phase of COVID-19 were collected in the hospital patient's medical record and completed with the patient's interview. Self-questionnaires included dyspnoea assessments (subjective assessment of dyspnoea through a visual analogic scale and modified Medical Research Council dyspnoea scale), sleep characteristics and scores (subjective sleep duration, Epworth sleepiness scale, Berlin Score, Pichot fatigue and depression scales,[19] International Restless Legs Syndrome severity scale, and in case of insomnia complaints, the Insomnia Severity Index). (See Supplementary Material)

Patients were further stratified according to their highest grade on the WHO seven-grade severity-scale COVID-19, based on the analysis of medical records, comorbidities, and medications, as follows: outpatients (grades 1–2); simple hospitalisation (not requiring supplemental oxygen: grade 3); hospitalisation for acute respiratory failure (requiring supplemental oxygen or Ambient air PaO2 <70 mmHg: grade 4); and hospitalisation in an ICU (requiring high-flow oxygen, non-invasive or invasive ventilation: grades 5–6).[20]

Pulmonary function tests

Pulmonary function tests were performed from inclusion to up to 12 months post-COVID-19 diagnosis. These tests included spirometry, full-body plethysmography, and diffusing capacity of the lungs for carbon monoxide (DLCO) performed according to the current recommendations of the European Respiratory Society (ERS) and the American Thoracic Society (ATS).[21] In addition, arterial blood gases were levied at rest (in a sitting position for at least 5 min). The following parameters were analysed: partial pressure of oxygen, partial pressure of carbon dioxide, arterial saturation in oxygen, bicarbonates, pH, lactates, and haemoglobin concentration. Haemoglobin-corrected DLCO (DLCO-Hb, expressed as % predicted), the primary endpoint as one of the most sensitive respiratory function variables in COVID disease, was calculated.[2,14] Impaired diffusion capacity was defined as DLCO-Hb <80% of the predicted values.[2]

To monitor lung recovery trajectories, pulmonary function tests were performed as close as possible of the 3-, 6- and 12-months visits post-COVID-19 diagnosis. Based on a conditional prolonged monitoring approach, pulmonary function tests were repeated during follow-up only for patients with persisting abnormal values, including impaired lung function (e.g., forced vital capacity [FVC] <80% predicted and/or diffusing capacity of the lung for carbon monoxide [DLCO] <70% predicted) and/or significant dyspnoea or pulmonary auscultation abnormality at the previous assessment.

Sleep examination

All centres are well-established sleep units with trained personnel for diagnosis, treatment, and follow-up of patients with sleep disorders. OSA status and OSA treatment status were defined at the time of COVID-19 diagnosis. OSA patients already treated by CPAP at the time of COVID-19 benefitted from therapeutic management and follow-up in compliance with international recommendations for good clinical practice. CPAP was selected as the initial treatment approach, and all CPAP-treated patients were followed by home care providers, applying the same standardised procedures, including regular mandatory home visits by technicians or nurses. The mean CPAP adherence was assessed during follow-up visits and defined as the mean usage from treatment initiation to follow-up evaluation based on CPAP devices data. Adherence to CPAP was defined as a mean nightly usage of at least 4 hours per night.

In unknown OSA status patients at the time of COVID-19 diagnosis, OSA diagnosis was performed between 3 and 6 months post COVID-19 diagnosis with polysomnography (PSG) or respiratory polygraphy (PG) performed either as an inpatient in the sleep laboratory or as an outpatient at home (see Supplementary

Material). PSG and PG were performed in room air, without supplemental O_2 for all patients. For the present analysis, OSA diagnosis was considered for an apnoea-hypopnoea index (AHI) ≥5/h. This enabled us to categorise patient with unknown OSA status appropriately.

Patients were stratified into 3 groups based on AHI and CPAP adherence at the time of COVID-19 diagnosis: No OSA (AHI < 5/h), Untreated OSA (AHI ≥ 5/h and no CPAP or CPAP adherence <4 hours/night) and Treated OSA (AHI ≥ 5/h and CPAP adherence ≥4 hours/night, including only patients treated with sufficient adherence at the time of COVID-19 diagnosis).

Statistical analysis

STROBE (Strengthening the Reporting of Observational Studies in Epidemiology) guidelines were applied (www.strobe-statement.org). Data are described using a median with interquartile range (IQR) for quantitative data and frequency and percentage for categorical variables. A comparison between 3 groups (No OSA, Untreated OSA, and Treated OSA) was performed using the Chi-squared test for comparisons of qualitative variables and a non-parametric Wilcoxon test for the quantitative variables. To account for multiple comparisons, a Bonferroni correction was used.

To investigate the evolution of DLCO-Hb after 3, 6, and 12 months of COVID-19 diagnosis, and due to our conditional monitoring approach, we applied domain-specific imputation, with last observation carried forward method, meaning that values identified as normal at 3 or 6 months were retained for subsequent timepoints. A mixed model with subjects as a random effect, including the interaction between COVID-19 groups and time, was performed. The different models with linear, quadratic, and cubic time effects have been studied to find the best model according to BIC and the normality of residuals. The mixed model with a quadratic time effect was retained due to its higher performance. The univariable analysis helped in identifying the key adjusted variables. The threshold of the univariable models was fixed at 20% for the multivariable analysis.

For all data aside from pulmonary function tests, a simple imputation method was carried out in case the missing data are few (<5%) by replacing the missing values with the median for quantitative variables and with the most frequent values for qualitative variables. A multiple imputation method with 5 datasets was used in this study for variables with missing data >5%. Variables for which the percentage of missing data observed was greater than 20% have not been retained for analysis. The significance threshold for statistical tests was set at 5%. Statistical analysis has been performed using SAS (V.9.4, SAS Institute).

Results

Study population

Three hundred and eighteen patients were prospectively included (see flowchart in Figure 1) in the cohort at 3 months post-COVID-19 diagnostic (median [Q1; Q3] delay = 3 [3; 4] months). One hundred and two (32.1%) patients required outpatient care, and 216 (67.9%) required hospitalisation (monitoring = 27 [8.5%], acute respiratory failure = 82 [25.8%], and intensive care unit (ICU) for invasive mechanical ventilation = 107 [33.7%]). Patients hospitalised for acute respiratory failure or requiring ICU hospitalisation were predominantly in the Untreated OSA and Treated OSA groups (Table 1). The characteristics of included patients according to COVID-19 severity are presented in Supplementary Table S1, and in Table 2 for OSA groups. Treated OSA patients were significantly older, predominantly males, mostly obese, and presented a higher burden of comorbidities, especially regarding cardio- and cerebrovascular diseases. There was no difference between the three groups regarding the prevalence of sleep comorbidities. Of the 63 (16.1%) patients presenting a known OSA status at the time of inclusion, 40 (63.5%) met the criteria for adequate CPAP adherence (>4 hours per night).

Pulmonary functional assessment at three months (Table 2)

Among the 245 patients tested at 3 months (median [Q1; Q3]) delay = 3 [3; 4] months) post-COVID-19 diagnostic, 53.3% of tested patients presented a DLCO-Hb ≥80% of predicted values, of which 72.4% of

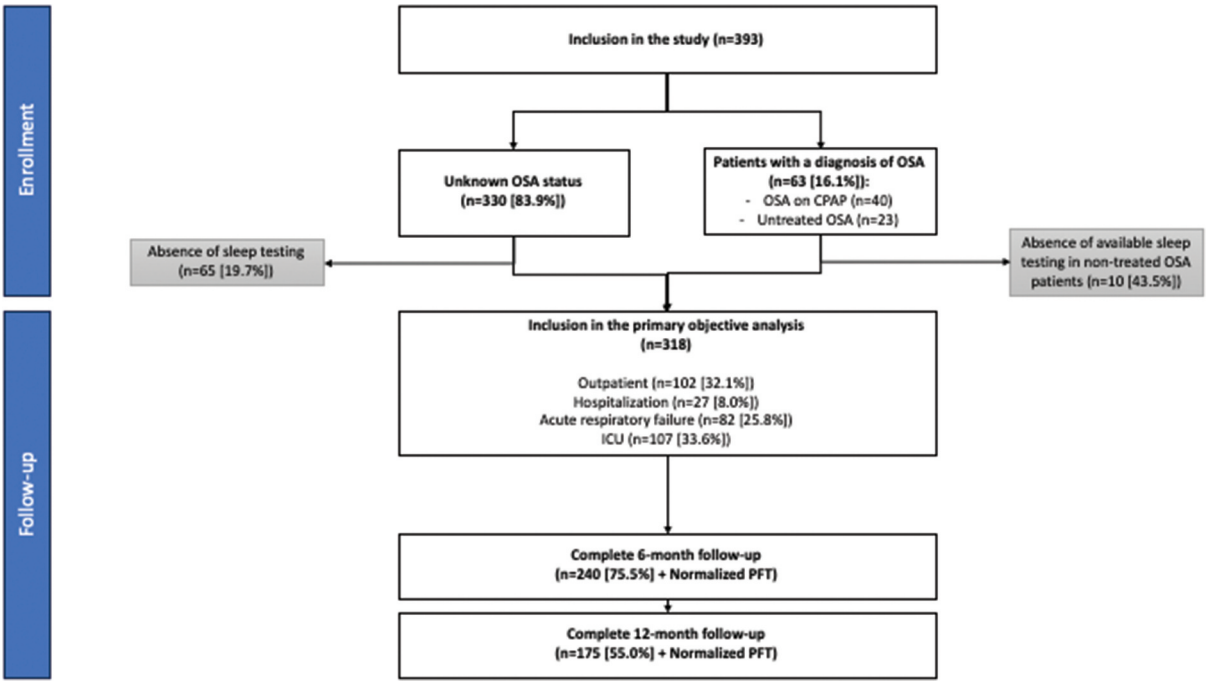

Figure 1. Study flow-chart. Abbreviations: CPAP: continuous positive airway pressure; ICU: intensive care unit; PFT: pulmonary function tests; OSA: obstructive sleep apnoea.

Table 1. Contingency table of COVID-19 acute care according to sleep apnoea status.

	No OSA *N* = 75 (23.6%)	Untreated OSA *N* = 203 (63.8%)	Treated OSA *N* = 40 (12. 6%)
COVID-19 outpatient *N* = 102 (32.1%)	42 (56.0%)	51 (25.1%)	9 (22.5%)
COVID-19 requiring hospitalisation *N* = 27 (8.5%)	11 (14.7%)	14 (6.9%)	2 (5.0%)
COVID-19 with acute respiratory failure *N* = 82 (25.8%)	7 (9.3%)	63 (31.0%)	12 (30.0%)
COVID-19 requiring ICU *N* = 107 (33.6%)	15 (20.0%)	75 (37.0%)	17 (42.5%)

Abbreviations: COVID-19: coronavirus disease 2019; ICU: intensive care unit; OSA: obstructive sleep apnoea.

patients in the outpatient group, compared to only 55.0%, 52.6%, and 34.2% in the hospitalisation group, hospitalisation for acute respiratory failure group, and hospitalisation in an ICU group, respectively (overall $p < 0.001$). The trajectories of recovery of DLCO-Hb according to initial COVID-19 severity over the year following COVID-19 acute care are presented in Figure 2. There was no difference in DLCO-Hb values between OSA groups (No OSA = 85.4 [71.5; 90.8] *vs*. Untreated OSA = 79.2 [56.6; 96.0] and Treated OSA = 86.3 [65.3; 102.8], $p = 0.25$), and the proportion of patients with DLCO-Hb ≥80% was comparable between the three groups. The other parameters that were significantly impaired in Untreated and Treated OSA patients were vital capacity, forced vital capacity, residual volume, and total lung capacity.

Sequential pulmonary functional assessment: Trajectories of lung recovery (Tables 3 and 4, Figures 2 and 3)

At 6 months (median [Q1; Q3]) delay = 7 [6; 7] months), 240 (75.5%) of the included patients performed the pulmonary functional assessments (No OSA (n [%]) = 59 [24.6%], Untreated OSA = 149 [62.1%]; Treated OSA = 32[13.3%]). At 12 months (12[12; 12] months), 175 (55.0%) of the included patients performed the pulmonary functional assessments (No OSA (n [%]) = 37 [21.1%], Untreated OSA = 106 [60.6%]; Treated OSA = 32 [18.3%]).

Table 2. Baseline population characteristics according to sleep apnoea severity.

	All patients N = 318	No OSA N = 75 (23.58%)	Untreated OSA N = 203 (63.84%)	Treated OSA N = 40 (12.58%)	Overall *p* value
Gender					
Female	114 (35.8)	48 (64)	61 (30)	5 (12.5)	**<0.001**
Male	204 (64.2)	27 (36)	142 (70)	35 (87.5)	
Age (years)	62 [52.0; 70.0]	55 [41.0; 65.0]	63.0 [55.0; 71.0] ($)	67.5 [58.0; 74.0] ($)	**<0.001**
BMI (kg/m^2)	27.8 [24.9; 31.5]	24.6 [22.2; 29.2]	28.1 [25.4; 31.6] ($)	31.0 [27.7; 33.6] ($*)	**<0.001**
Smoking status					
Current smoking	7 (2.2)	2 (2.7)	3 (1.5)	2 (5.0)	0.709
Former smoking	129 (40.6)	29 (38.7)	84 (41.4)	16 (40.0)	
No smoking	182 (57.2)	44 (58.7)	116 (57.1)	22 (55.0)	
Dyspnoea (visual analogic scale, 0–10)	6.0 [4.0; 8.0]	7.0 [4.0; 9.0]	6.0 [4.0; 8.0]	7.0 [5.0; 9.0]	0.193
MRC					
0	272 (86.1)	63 (85.1)	179 (88.6)	30 (75)	0.066
1	25 (7.9)	3 (4.1)	15 (7.4)	7 (17.5)	
2	14 (4.4)	5 (6.8)	6 (3.0)	3 (7.5)	
3	4 (1.3)	2 (2.7)	2 (1.0)	0 (0.0)	
4	1 (0.3)	1 (1.4)	0 (0.0)	0 (0.0)	
MEDICAL HISTORY					
Cardiovascular diseases (n (%))	141 (44.3)	16 (21.3)	94 (46.3) ($)	31 (77.5) ($*)	**<0.001**
Atherosclerosis	5 (1.6)	1 (1.3)	3 (1.5)	1 (2.5)	0.877
Hypertension	104 (32.7)	14 (18.7)	66 (32.5) ($)	24 (60) ($*)	**<0.001**
Coronary insufficiency	22 (6.9)	2 (2.7)	16 (7.9)	4 (10)	0.224
Myocardial infarction	8 (2.5)	1 (1.3)	6 (3)	1 (2.5)	0.745
Arrhythmias	18 (5.7)	1 (1.3)	14 (6.9)	3 (7.5)	0.177
Cerebrovascular diseases (n (%))	15 (4.7)	4 (5.3)	7 (3.4)	4 (10.0)	0.195
Respiratory diseases (n (%))	137 (43.1)	29 (38.7)	68 (33.5)	40 (100.0) ($*)	**<0.001**
Asthma	7 (2.2)	4 (5.3)	2 (1.0)	1 (2.5)	0.089
COPD	10 (3.1)	3 (4.0)	4 (2.0)	3 (7.5)	0.166
Diabetes (n (%))	53 (16.7)	7 (9.3)	36 (17.7)	10 (25.0)	0.079
Type 1 diabetes	2 (0.6)	0 (0)	1 (0.5)	0 (0.0)	0.565
Type 2 diabetes	52 (16.4)	7 (9.3)	35 (17.2)	10 (25.0)	0.082
Renal failure (n (%))	8 (2.5)	0 (0.0)	7 (3.4)	1 (2.5)	0.265
Cancer history (n (%))	17 (5.3)	4 (5.3)	10 (4.9)	3 (7.5)	0.804
SLEEP CHARACTERISTICS					
Average subjective sleep duration (hours)	6.5 [6.0; 7.5]	6.5 [6.0; 7.0]	6.5 [6.0; 7.5]	6.5 [6.0; 7.0]	0.755
Berlin status -high-risk category (n (%))	98 (30.9)	19 (25.3)	65 (32)	14 (35.9)	0.435
Epworth Sleepiness Scale (/24)	7.0 [4.0; 11.0]	8.0 [3.5; 12.0]	6.0 [4.0; 10.0]	6.0 [4.0; 12.0]	0.321
Asthenia Score (Pichot,/32)	9.0 [4.0; 18.0]	10.5 [5.0; 20.0]	9.0 [3.0; 17.0]	8.0 [3.0; 17.0]	0.251
Depression Score (Pichot,/13)	2.0 [0.0; 6.0]	3.0 [0.0; 6.0]	1.0 [0.0; 5.0]	2.0 [0.0; 5.0]	0.317

(*Continued*)

Table 2. (Continued).

	All patients N = 318	No OSA N = 75 (23.58%)	Untreated OSA N = 203 (63.84%)	Treated OSA N = 40 (12.58%)	Overall *p* value
RLS score (Number of symptoms,/4)	0.0 [0.0; 2.0]	0.0 [0.0; 2.0]	0.0 [0.0; 2.0]	0.0 [0.0; 2.0]	0.722
International Restless Legs Syndrome severity scale (/40)	15.5 [11.0; 21.0]	16.0 [12.0; 21.5]	15.5 [10.0; 20.0]	18.0 [12.0; 27.0]	0.435
Insomnia Severity Index (/28)	15.0 [12.0; 19.0]	18.0 [13.0; 21.0]	15.0 [10.0; 19.0]	15.0 [11.0; 17.0]	0.139
POLYSOMNOGRAPHY					
Total sleep time (min)	384 [327.0; 424.0]	377 [325.0; 413.0]	387.0 [333.0; 427.0]	384.0 [356.0; 437.0]	0.600
Wake after sleep onset (%TST)	14.6 [8.6; 28.0]	11.5 [7.1; 22.6]	15.2 [9.1; 29.5]	23.8 [12.3; 36.3]	**0.030**
Stage 1 sleep (N1, %TST)	8.8 [5.3; 13.4]	6.1 [4.4; 10.0]	9.5 [5.7; 14.4] ($)	8.5 [6.2; 15.3]	**0.003**
Stage 2 sleep (N2, %TST)	52.9 [45.3; 61.6]	51.4 [45.8; 58.1]	53.7 [44.7; 61.9]	59.4 [46.6; 65.3]	0.310
Slow Wave Sleep (N3, %TST)	14.4 [6.0; 20.6]	18.3 [10.9; 23.6]	13.3 [4.1; 19.4] ($)	9.5 [5.2; 13.2] ($)	**0.001**
REM Sleep (%TST)	21.9 [17.3; 26.7]	22.8 [18.6; 26.9]	21.4 [17.0; 27.0]	21.8 [16.6; 23.4]	0.604
Respiratory micro-arousal index	9.3 [3.4; 18.6]	1.8 [0.7; 3.7]	12.8 [7.4; 23.7] ($)	12.7 [4.3; 26.1] ($)	**<0.001**
AHI	14.5 [4.9; 27.4]	1.8 [0.6; 3.4]	20.0 [12.7; 33.2] ($)	19.4 [10.9; 34.9] ($)	**<0.001**
RDI	18.0 [6.7; 32.6]	2.9 [1.1; 5.7]	23.3 [15.4; 37.6] ($)	19.4 [10.9; 35.7] ($)	**<0.001**
Percentage of central respiratory events	11.1 [1.9; 34.8]	46.2 [8.7; 90.0]	7.6 [1.1; 22.8] ($)	4.4 [0.9; 25.7] ($)	**<0.001**
ODI	13.0 [4.8; 26.3]	2.4 [1.1; 4.7]	18.2 [10.2; 30.5] ($)	29.3 [11.5; 45.5] ($)	**<0.001**
Cumulative time spent below 90% of SaO_2 (%)	0.0 [0.0; 3.1]	0.0 [0.0; 0.0]	0.4 [0.0; 4.3] ($)	2.5 [0.0; 25.2] ($)	**<0.001**
Mean SpO_2 (%)	93.0 [92.0; 95.0]	95.0 [93.0; 96.0]	93.0 [92.0; 94.0] ($)	92.0 [90.0; 94.0] ($)	**<0.001**
Nadir SpO_2 (%)	86.0 [82.0; 89.0]	91.0 [89.0; 93.0]	84.0 [81.0; 87.0] ($)	82.0 [76.0; 86.0] ($)	**<0.001**

Abbreviations: AHI: apnoea-hypopnoea index; BMI: body mass index; COPD: chronic obstructive pulmonary disease; EDS: excessive daytime sleepiness; ESS: Epworth Sleepiness Scale; MRC: Medical Research Council; OSA: obstructive sleep apnoea; PLM: periodic limb movement; RDI: respiratory disturbance index; REM: Rapid Eye Movement sleep; RLS: restless legs syndrome; SpO_2: peripheral oxygen saturation; TST: total sleep time; WASO: wake after sleep onset.

Data are presented as median [Q1; Q3] for quantitative variables or number (percentages) for qualitative variables.

A Chi2 test was used for comparisons of qualitative variables between groups. A Non-parametric Wilcoxon test was used to compare the quantitative variables. For the multiple comparisons, the Bonferroni correction was used. The threshold is 0.0167 (=0.05/3). $ Significantly different from No SA group. * Significantly different from untreated SA group.

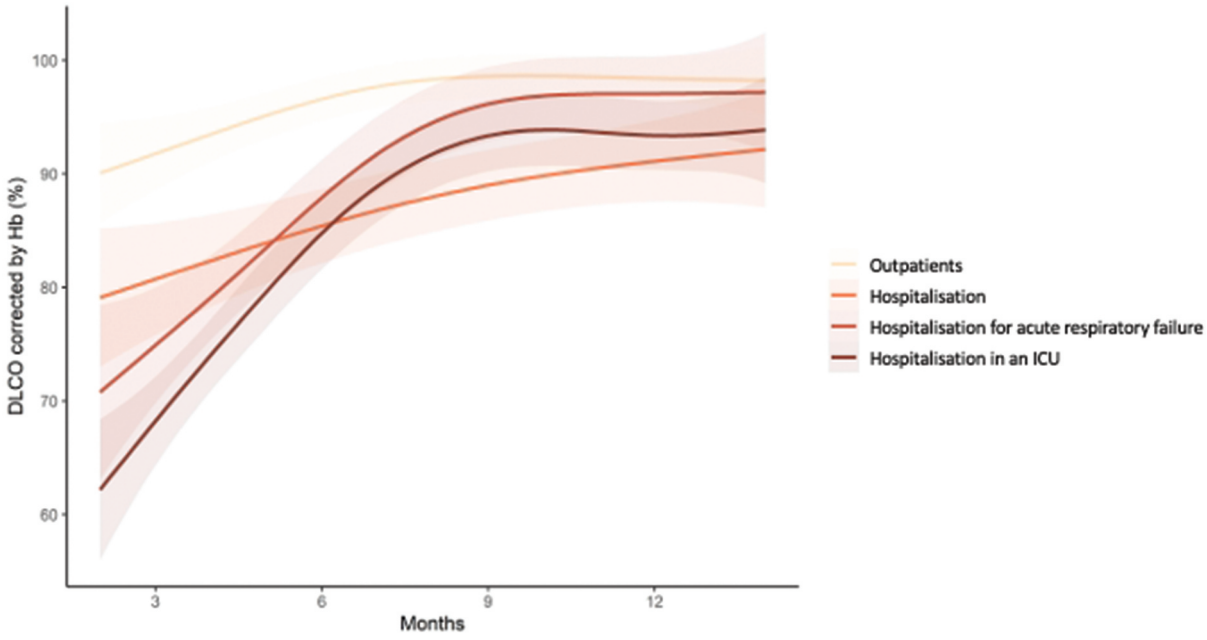

Figure 2. Trajectories of recovery of diffusing capacity of the lung for carbon monoxide corrected by haemoglobin according to initial COVID-19 severity over the year following COVID-19 acute care. Note: Given our conditional monitoring methodology, we employed domain-specific imputation using the last observation carried forward technique. Values recorded as normal at 3 or 6 months were maintained for subsequent timepoints. Abbreviations: DLCO: diffusing capacity of the lung for carbon monoxide; Hb: haemoglobin; ICU: intensive care unit.

At 6 and 12 months, there was no difference of DLCO-Hb between the three groups (Figure 3). The proportion of patients with DLCO-Hb ≥80% was significantly lower in the Untreated OSA group (No OSA (n [%]) = 34 [87.2], Untreated OSA = 71 [62.3], Treated OSA = 18 [78.3], $p = 0.009$) at 6 months, but not at 12 months ($p = 0.07$) indicating comparable recovery between the three groups. The other parameters, impaired at baseline, followed the same recovery trajectories (Table 2).

In multivariable analysis (Table 4 – see Supplementary Table S2 for results of univariable analysis), COVID-19's initial severity had no significant effect on DLCO-Hb ($p = 0.27$). There was a significant association between OSA severity and its treatment and the evolution of DLCO-Hb ($p = 0.03$).

Discussion

In this longitudinal, prospective multicentric cohort study, we assessed the trajectories of pulmonary function recovery over the year post-COVID-19 diagnosis. We applied a prolonged pragmatic, conditional monitoring based on the results of pulmonary function testing, as previously performed.[2] To our knowledge, this is the first study to prospectively assess the impact of OSA and its treatment on recovery trajectories of lung function following mild to severe forms of COVID-19. We showed that OSA delayed lung diffusion capacity recovery trajectories, whatever the initial severity of the COVID-19 infection. OSA treatment by CPAP effectively accelerated the lung function recovery process.

In our cohort, 67.9% of included patients were hospitalised, whereas 32.19% required outpatient care. The proportion of patients requiring hospitalisation was higher in our study than in larger prospective observational cohort studies conducted during the same period, where only <20% of symptomatic patients developed moderate-to-severe forms of COVID-19 infections characterised by hypoxaemic pneumonia requiring hospitalisation.[3] The university hospital-based recruitment of the majority of the patients may explain this ratio. However, the proportion of hospitalised patients requiring additional ventilatory support and intensive care unit (ICU) admission for acute respiratory distress syndrome (33.5%) was comparable.[3] In our study, OSA was documented in 76.4% of the patients, and moderate-severe OSA (AHI ≥ 15/h) was diagnosed in 54.7% of the included patients. The prevalence of moderate-severe OSA in our study is higher compared to available data (for review, see Miller MA and Cappuccio FP [22]). Our study, based on an objective diagnosis of OSA, with

Table 3. Pulmonary function and blood gases evolution during 1-year follow-up.

		Visit at 3 months					Visit at 6 months			Visit at 12 months		
	Available (%)	**All patients N = 245**	**No OSA N = 62 (23.31%)**	**Untreated OSA N = 149 (60.82%)**	**Treated OSA N = 30 (13.88%)**	**Overall *p* value**	Available (%)	**All N = 240**	**Overall *p* value**	**Available (%)**	**All N = 175**	**Overall *p* value**
VC (% pred.)	240 (98.0)	101.6 [89.4; 114.6]	109.4 [94.6; 118.4]	98.8 [88.4; 113.5]	99.4 [84.1; 110.7]	0.043	235 (97.9)	104.2 [93.8; 118.0]	0.197	164 (93.7)	104.8 [93.9; 117.8]	0.065
FVC (% pred.)	240 (98.0)	101.6 [88.3; 112.6]	106.7 [96.6; 115.3]	99.8 [86.8; 111.5]	98.8 [80.3; 108.8] $	0.026	235 (97.9)	102.9 [92.6; 117.3]	0.116	163 (93.1)	103.5 [92.1; 116.9]	**0.009**
FVC ≥ 80% pred (N, %)	240 (98.0)	205 (85.4)	55 (91.7)	125 (85)	25 (75.8)	0.112	237 (98.8)	211 (89.0)	0.543	163 (93.1)	152 (88.9)	0.099
FEV1% pred.)	240 (98.0)	99.6 [89.7; 110.7]	100.1 [92.4; 108.7]	99.9 [89.1; 111.1]	95.5 [82.3; 111.4]	0.470	235 (97.9)	104.2 [92.1; 112.7]	0.871	163 (93.1)	103.0 [92.7; 114.5]	0.169
FEV1/FVC (%)	240 (98.0)	81.3 [76.8; 85.5]	82.5 [75.5; 85.1]	81.0 [76.8; 85.3]	82.7 [77.0; 86.9]	0.695	235 (97.9)	81.1 [76.0; 84.8]	0.870	164 (93.7)	80.5 [74.6; 84.3]	0.428
TLC (% pred.)	235 (95.9)	99.6 [84.7; 112.2]	110.9 [94.2; 119.2]	94.5 [83.8; 106.3] $	96.6 [83.5; 115.4]	**<.001**	224 (93.3)	102.0 [87.6; 110.7]	**0.005**	155 (88.6)	98.6 [86.3; 110.7]	0.032
FRC (L)	234 (95.5)	3.2 [2.6; 3.8]	3.2 [2.7; 3.7]	3.1 [2.5; 3.9]	3.2 [2.6; 4.1]	0.835	223 (92.9)	3.2 [2.6; 3.9]	0.878	157 (89.7)	3.0 [2.4; 3.7]	0.739
RV (% pred.)	234 (95.5)	96.1 [78.6; 123.6]	120.1 [88.0; 157.0]	91.1 [74.8;112.6] $	97.2 [77.6; 125.2] $	**<.001**	224 (93.3)	99.7 [78.4; 121.3]	**0.002**	155 (88.6)	100.0 [77.0; 116.0]	0.480
D_{LCO} (% pred.)	243 (99.2)	83.0 [65.4; 94.3]	86.5 [76.8; 92.1]	79.5 [53.8; 94.4]	87.0 [68.4; 100.1]	0.077	233 (97.1)	85.7 [69.9; 99.8]	0.162	162 (92.6)	85.1 [74.5; 97.2]	0.465
D_{LCO}-Hb (% pred.)	229 (93.5)	82.3 [63.5; 93.5]	85.4 [71.5; 90.8]	79.2 [56.6; 96.0]	86.3 [65.3; 102.8]	0.247	176 (73.3)	88.6 [76.4; 100.2]	0.054	104 (59.4)	92.2 [84.1; 102.8]	0.896
D_{LCO}-Hb ≥ 80% pred.	229 (93.5)	122 (53.3)	37 (62.7)	68 (48.6)	17 (56.7)	0.174	176 (73.3)	123 (69.9)	**0.009**	104 (59.4)	89 (85.6)	0.711
D_{LCO}-Hb ≥ 70% pred.	229 (93.5)	151 (65.9)	45 (76.3)	86 (61.4)	20 (66.7)	0.130	176 (73.3)	141 (80.1)	**0.006**	104 (59.4)	95 (91.3)	0.543
D_{LCO}-Hb ≥ 50% pred.	229 (93.5)	191 (83.4)	55 (93.2)	108 (77.1) $	28 (93.3)	**0.006**	176 (73.3)	152 (86.4)	**0.012**	104 (59.4)	101 (97.1)	0.366
KCO (% pred.)	231 (94.3)	91.2 [80.2; 102.5]	88.5 [80.9; 94.4]	91.3 [72.6; 102.7]	103.1 [96.9; 111.7] $*	**<.001**	123 (51.3)	93.7 [77.3; 107.5]	0.147	117 (66.9)	97.1 [89.9; 111.7]	0.064
Blood gases												
PaO_2 (kPa)	196 (80)	11.6 [10.6; 12.4]	12.1 [11.4; 12.9]	11.5 [10.6;12.4] $	11.1 [10.3;12.1] $	**0.002**	60 (25.0)	11.3 [10.5; 12.1]	**0.015**	22 (12.6)	11.0 [10.7; 11.8]	0.188
$PaCO_2$ (kPa)	198 (80.8)	4.9 [4.6; 5.3]	4.8 [4.6; 5.3]	4.9 [4.6; 5.3]	5.1 [4.8; 5.3]	0.304	60 (25.0)	5.0 [4.5; 5.3]	0.758	22 (12.6)	4.8 [4.5; 5.5]	0.063

(*Continued*)

Table 3. (Continued).

		Visit at 3 months					Visit at 6 months			Visit at 12 months		
pH	198 (80.8)	7.4 [7.4; 7.4]	7.4 [7.4; 7.4]	7.4 [7.4;7.4]	7.4 [7.4;7.4]	0.105	60 (25.0)	7.4 [7.4; 7.4]	0.937	22 (12.6)	7.4 [7.4; 7.4]	0.030
$HCO3^-$ (mmol/L)	196 (80)	23.5 [22.0; 24.7]	23.5 [22.2; 24.6]	23.2 [21.4; 24.8]	23.7 [22.9; 24.7]	0.607	60 (25.0)	23.6 [22.3; 25.1]	0.975	22 (12.6)	22.4 [21.4; 25.2]	0.128
O_2 Saturation (%)	199 (81.3)	97.0 [96.0; 97.0]	97.0 [96.6; 98.0]	97.0 [96.0; 97.0] $	97.0 [96.0; 97.0] $	**0.013**	59 (24.6)	97.0 [96.0; 97.0]	**0.008**	22 (12.6)	96.0 [96.0; 97.0]	0.055
Lactate (mmol/L)	183 (74.7)	1.3 [1.0; 1.6]	1.1 [1.0; 1.5]	1.3 [1.1; 1.6]	1.3 [1.0; 1.7]	0.136	57 (23.8)	1.5 [1.2; 1.8]	0.851	21 (12.0)	1.4 [1.2; 1.8]	0.107

Abbreviations: D_{LCO}: diffusing capacity of the lung for carbon monoxide; FVC: forced vital capacity; FEV1: forced expiratory volume in 1 s; FRC: functional residual capacity; Hb: haemoglobin; $HCO3^-$: serum bicarbonates; KCO: carbon monoxide transfer coefficient; O_2: dioxygen; OSA: obstructive sleep apnoea; $PaCO_2$: partial pressure in carbon dioxygen; PaO_2: partial pressure of dioxygen; RV: residual volume; VC: vital capacity.

Unless otherwise stated, data are presented as n (%) or median (interquartile range). Percentages are calculated by category after excluding patients with missing values for that variable.

Data are presented as median [Q1; Q3] for quantitative variables or number (percentages) for qualitative variables.

A Chi2 test was used for comparisons of qualitative variables between groups. A Non-parametric Wilcoxon test was used to compare the quantitative variables. For the multiple comparisons, the Bonferroni correction was used. The threshold is 0.0167 (=0.05/3). $ Significantly different from No SA group. * Significantly different from Untreated SA group.

Table 4. Univariable and multivariable models of DLCO corrected by Hb (%) evolution with quadratic time effect.

Variables	Items	Univariable			Multivariable		
		Coefficient [CI 95%]	*p* value	Overall *p* value	Coefficient [CI 95%]	*p* value	Overall *p* value
Time	–	–	–	–	0.76 [0.20; 1.33]	**0.0079**	
Time*Covid-19 severity		-	–	–	−0.04 [−0.07; −0.01]	**0.0157**	
D_{LCO}-Hb – Baseline		0.96 [0.92; 1.00]	**<.0001**		0.96 [0.91; 1.00]	**<.0001**	
Covid-19 severity				**<.0001**			0.2700
	Covid-19 requiring hospitalisation *vs*. Covid-19 outpatient	−15.83 [−27.79; −3.87]	**0.0095**		−1.70 [−5.73; 2.34]	0.4096	
	Covid-19 with respiratory distress *vs*. Covid-19 outpatient	−22.64 [−30.91; −14.37]	**<.0001**		−1.06 [−4.09; 1.97]	0.4934	
	Covid-19 requesting ICU *vs*. Covid-19 outpatient	−21.88 [−29.63; −14.12]	**<.0001**		1.37 [−1.49; 4.24]	0.3477	
OSA groups				**0.0004**			**0.0305**
	Untreated OSA *vs*. No OSA	−13.85 [−21.66; −6.05]	**0.0005**		−3.75 [−6.54; −0.96]	**0.0086**	
	Treated severe OSA *vs*. No OSA	−0.44 [−11.73; 10.85]	0.9397		−3.83 [−8.03; 0.37]	**0.0736**	
Diabetes	Yes *vs*. No	−11.21 [−20.02; −2.41]	**0.0126**		−2.09 [−5.10; 0.92]	0.1744	
Cardiovascular diseases	Yes *vs*. No	−6.17 [−12.83; 0.49]	**0.0694**		−0.02 [−2.40; 2.35]	0.9863	
Age		−0.17 [−0.42; 0.08]	**0.1825**		0.10 [0.00; 0.19]	**0.0438**	
Alcohol consumption		8.77 [−4.81; 22.35]	**0.2053**		1.87 [−2.29; 6.03]	0.3771	
Physical exercise	Yes *vs*. No	18.52 [11.74; 25.31]	**<.0001**		0.00 [−2.34; 2.34]	0.9978	
BMI		−0.62 [−1.17; −0.06]	**0.0302**		−0.02 [−0.22; 0.17]	0.8182	
Gender	Male *vs*. Female	0.55 [−6.36; 7.46]	0.8762		-	–	–
Smoking status				0.6231	–	–	–
	Current smoking *vs*. No smoking	8.3 [−13.37; 29.98]	0.4525		-	–	–
	Former smoking *vs*. No smoking	0.55 [−6.36; 7.46]	0.8762		-	–	–
Cerebrovascular diseases	Yes *vs*. No	−6.63 [−22.55; 9.30]	0.4145		-	–	–
Respiratory diseases	Yes *vs*. No	−1.56 [−8.26; 5.14]	0.6472		-	–	–

Note: The threshold of the univariable models was fixed at 20% for the multivariable analysis.
Abbreviations: BMI: body mass index; D_{LCO}: diffusing capacity of the lung for carbon monoxide; D_{LCO}-Hb: diffusing capacity of the lung for carbon monoxide (DLCO) corrected by haemoglobin; OSA: obstructive sleep apnoea.

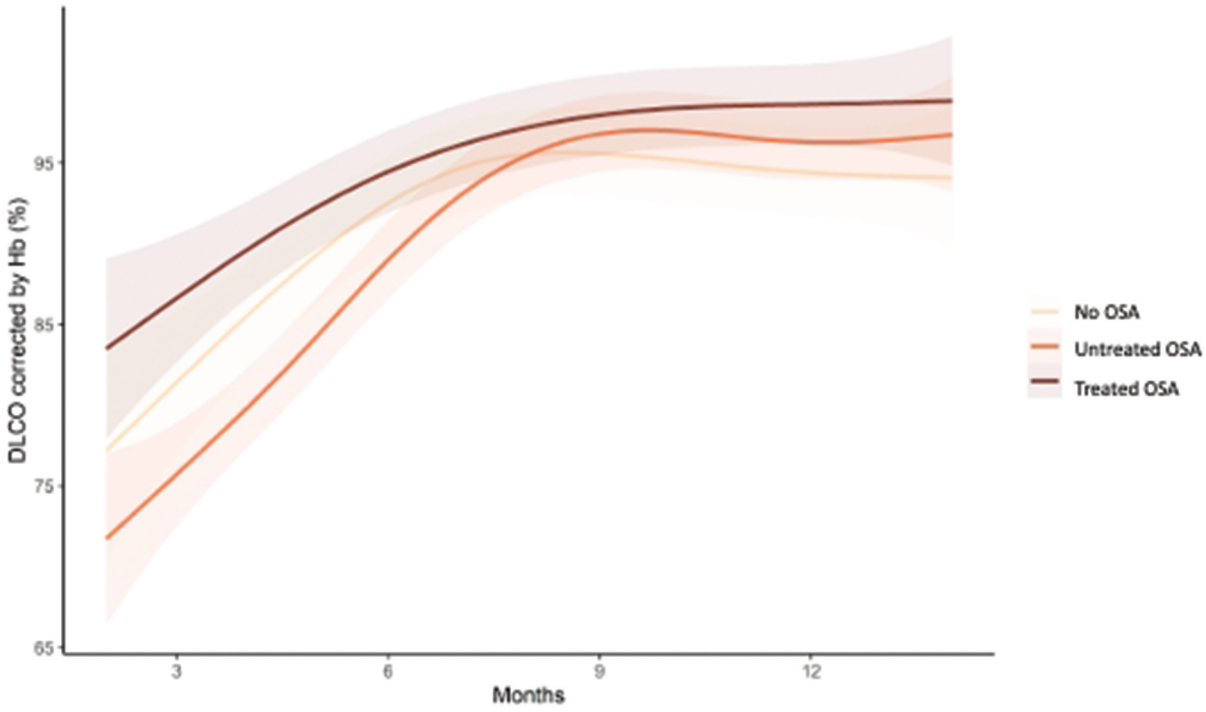

Figure 3. Trajectories of recovery of diffusing capacity of the lung for carbon monoxide corrected by haemoglobin according to obstructive sleep apnoea severity and treatment over the year following COVID-19 acute care. Note: Given our conditional monitoring methodology, we employed domain-specific imputation using the last observation carried forward technique. Values recorded as normal at 3 or 6 months were maintained for subsequent timepoints. Abbreviations: DLCO: diffusing capacity of the lung for carbon monoxide; Hb: haemoglobin; OSA: obstructive sleep apnoea.

a majority by polysomnography, may explain these discrepancies, as most of the available evidence comes from retrospective cohorts or small sample prospective studies, with different baseline severity of COVID-19.[22] In line with our results, Perger E. et al. reported that during the acute phase of COVID-19, two-thirds of patients exhibited OSA, of which severity was related to a COVID-19 syndrome with poor outcomes.[23] Beyond its impact on acute severity,[10,11] OSA is increasingly acknowledged as being a risk factor for the development of post-acute sequelae of SARS-CoV-2 infection.[9,13,14,18] We showed in our multivariable models that OSA and its treatment by CPAP affected lung recovery trajectories independently of initial COVID-19 severity. We observed a differential recovery of lung function, with improvement obtained during the first 6 months. If only 53.3% of patients presented a DLCO-Hb ≥ 80% of predicted values at 3 months, up to 69.9% and 85.6% of patients reached a normal diffusing capacity of the lung at 6 and 12 months, respectively. This observation is consistent with studies conducted in population-based cohorts and patients with more severe forms of COVID-19.[2,14,24,25] More recently, Han X. et al. showed that the observed improvement in lung diffusing capacity was maintained at three years, with a gradual and significant improvement from 6 months to 3 years regardless of acute COVID-19 severity.[26] In the smaller observational prospective study from Labarca G. et al., DLCO remains abnormal in OSA patients one year following COVID-19 diagnosis (25% of OSA patients with abnormal persistent DLCO values vs. 3.6% non-OSA, $p = 0.02$).[14] Interestingly, they also showed a significant difference in circulating levels of the pro-inflammatory cytokine IL-6 between untreated OSA and non-OSA patients 4 months post-COVID-19 diagnosis, with an Odd Ration (OR) of increased IL-6 of 2.54 (95%-CI, 1.21–5.32, $p = 0.013$). Systemic inflammation is a pathophysiologic feature shared by both COVID-19 and OSA, reinforcing their synergistic detrimental association. OSA, through intermittent hypoxia and fragmented sleep, triggers low-grade inflammation,[27] including vascular and lung inflammation,[28] and it has been suggested that OSA might exacerbate systemic inflammation incriminated in the severity of COVID-19 outcomes that span physical health, mental health, and cognitive impairment.[23,29] Additionally, we observed significant differences in forced vital capacity (FVC), total lung capacity (TLC), and residual volume across OSA subgroups at baseline. OSA can affect pulmonary function test results by causing mild reductions in FVC, forced expiratory volume in one second (FEV_1), TLC, and

functional residual capacity (FRC), as well as increased airway resistance, particularly in obese individuals and those with more severe OSA.[30,31] In patients without underlying lung disease, OSA does not typically cause clinically significant obstructive or restrictive ventilatory defects on spirometry during wakefulness. In our study, the frequency of pre-existing lung diseases in patient histories has been reported (asthma and COPD, with no statistical difference between OSA groups. No patient with a history of interstitial lung disease was included).

One of the original aspects of our study is to demonstrate the significant impact of OSA treatment on lung recovery trajectories, despite a low CPAP adherence (treatment use >4 h/night) prevalence, reaching only 63.5% in our cohort, compared to the 76% at 1 year observed in the French Nationwide Claims Data Lake for Sleep Apnea study.[32] Attias D. et al.,[33] in a cohort of 7485 patients with OSA, assessed the impact of the coronavirus disease (COVID-19) national lockdown in France on objective adherence to CPAP by telemonitoring, in comparison with the pre-COVID-19 period. They observed a 3.9% ($p < 0.001$) increase in adherence during lockdown and hypothesised that fear of being hospitalised, decreased occupational stress, and increased opportunity for sleep may have directly influenced CPAP adherence. While our study demonstrates a significant impact of OSA treatment on lung recovery trajectories despite suboptimal adherence, we hypothesise that optimal adherence (>70%) could further amplify the observed positive effects. Adherence to CPAP therapy facilitates lung recovery following infection through several specific mechanisms: reduction of inflammation, promotion of alveolar repair, and prevention of secondary complications.[34–37]

Our study has several strengths, including its multicenter design, with repeated comprehensive but pragmatic assessment of lung function. This pragmatic approach, used also in several studies,[2,14] is a realistic picture of clinical follow-up of patients recovering from COVID-19, enabling its transposition in most centres. As we broadly enrolled COVID-19 patients with different initial levels of severity, our study provided a realistic representation of initial disease severity subgroups.

Our study has also several limitations. The first assessment of pulmonary function testing was performed 3 months following post-COVID-19 diagnosis. We recognise the lack of pre-infection or acute pulmonary function test measurements, which would have provided greater insight into the progression of pulmonary function. We recognise that consistency in the type of examination would have been preferable. However, due to the prevailing health conditions during the COVID-19 pandemic, it was not possible to implement strict standardisation for pulmonary function test procedures and equipment.

We did not control for other important, sleep-related parameters, such as sleep duration, that may have been greatly affected during the pandemic,[38,39] and may contribute to exacerbate the cytokine storm in COVID-19.[11,40] Regarding follow-up evaluations, and due to the pragmatic study design, we could not capture potential worsening in any of the participants discarded for follow-up at 6 months and 1 year, although this seems unlikely. Moreover, as our recruitment spanned several COVID-19 waves, acute care may have evolved and impacted the outcome. Lastly, even if CPAP showed a positive effect, adherence in our study didn't reflect typical use in the general population. Additionally, our study categorises the 'Treated OSA' group based solely on CPAP adherence at the time of COVID-19 diagnosis (treatment use ≥4 hours/night), which may not fully capture optimal treatment outcomes due to the lack of systematic assessment of residual AHI. Nonetheless, the effects observed in this cohort are considered to offer meaningful clinical benefit, even if treatment is not entirely optimal.

In conclusion, OSA treatment by CPAP improves lung diffusing capacity recovery trajectory following COVID-19. This impairment in the initial recovery trajectory following acute severe lung infection related to untreated OSA emphasises the need for systematic screening of OSA and the development of personalised care and follow-up in this high-risk population.

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Author contributions

Conception and design: R. Tamisier, JL. Pépin; Substantial contribution to the acquisition of the data: S. Baillieul, L. Sesé, L. Boyer, B. Chenuel, P. Romand, C. Planes, G. Derumeaux, S. Bayat, M. Destors, F. Arbib, M. Poussel, E. Allado, T. Gille, JL. Pépin, R. Tamisier; Statistical analysis: V. Ngo Thi Hong; Initial drafting of the manuscript: S. Baillieul, JL. Pépin, R. Tamisier. All authors contributed to data interpretation, critical review and revision of the manuscript, and final approval of the version to be published.

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ORCID

Sébastien Baillieul http://orcid.org/0000-0002-2348-6918
Renaud Tamisier http://orcid.org/0000-0003-1128-6529

Data availability statement

The data underlying this article will be shared on reasonable request to the corresponding author.

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