Unilateral Hemidiaphragm Weakness Is Associated with Positional Hypoxemia in REM Sleep

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Background: Patients with unilateral diaphragmatic paralysis (UDP) have been reported to have varied respiratory symptoms and often reduced lung function. We sought to describe the polysomnographic respiratory characteristics in patients with UDP without obstructive sleep apnea.

Methods: We prospectively collected 5 cases with clinical investigation regarding symptoms, lung function, and nocturnal polysomnography. The respiratory sleep characteristics were analyzed with standardized scoring of respiratory events in 30-sec epochs and comparison according to sleep-wake stages and body position with respect to oximetry. The cases were compared to 5 controls matched for age, gender, and body mass index.

Results: Three of 5 patients had significant awake lung restriction with a mean (range) forced vital capacity of 1.89 (1.48-2.24) liters, 72% (45% to 102%) predicted. All had REM sleep with few apneas and episodes of prolonged hypopneas characterized by important desaturation noted on

oximetry. These desaturations were greatest during REM sleep when the patients slept supine with a mean (SD) saturation of 90.8% (4.5%) and minimum of 64% or on the side unaffected by UDP with a mean saturation of 87.8% (5.3%) and minimum of 67% ($p < 0.0001$ compared to same positions awake). Other sleep stages had few, if any significant desaturations and these events rarely occurred when the patient slept in the supine position. Saturation was lower in all sleep-wake stages and sleep positions compared to controls ($p < 0.0001$).

Conclusion: Patients with UDP demonstrate position-dependent hypopneas in REM sleep with frequent desaturations.

Keywords: Unilateral diaphragmatic paralysis, diaphragmatic disorders, hypoxemia, polysomnography, sleep, sleep disordered breathing

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The diaphragm is the chief muscle of inspiration and receives innervation from cervical nerves 3, 4, and 5. Diaphragmatic paralysis can be unilateral or more rarely bilateral. In the latter case, patients are usually symptomatic and often experience respiratory failure requiring some level of ventilatory support. Unilateral diaphragm paralysis (UDP) however, is thought to be tolerated much more readily, especially in the absence of underlying lung pathology.¹⁻⁴ It has usually been thought that UDP is of little physiologic consequence.¹ However patients may have shortness of breath especially when recumbent and a restrictive pattern is often seen on pulmonary function testing.^{2,5}

Sleep related hypoxemia has been described in patients with generalized muscle weakness, and in those with bilateral diaphragm dysfunction.⁶⁻¹³ However, little is known about the sleep characteristics of individuals with UDP and no other concomitant respiratory or muscle pathology. In this paper, we report the polysomnographic findings in five patients with UDP who were free of significant intrinsic lung disease, generalized muscle weakness, and underlying obstructive sleep apnea. Our findings suggest that UDP may be a significant and unsuspected cause of sleep disordered breathing in these patients.

BRIEF SUMMARY

Current Knowledge/Study Rationale: Isolated unilateral diaphragmatic paralysis has been thought to be of little physiologic consequence and infrequently symptomatic for orthopnea. The polysomnographic characteristics have been reported for patients who also have other lung or muscle pathology.

Study Impact: Compared to matched controls, oximetry was lower in all sleep-wake stages and body positions. This was most pronounced in REM sleep, especially supine or sleeping on the side unaffected by diaphragmatic paralysis.

METHODS

Study Design

Patient Recruitment and Clinical Evaluation

A sentinel case was originally seen 6 years prior to the writing of this manuscript and is described in detail below. As a result of the findings in the sentinel case we began a study to prospectively evaluate the sleep characteristics of patients with UDP who did not have concomitant intrinsic lung disease

Table 1—Demographic and historical characteristics of the case patients

Patient	Age	Gender	Pack-years	Surgery same side	BMI	Side	Tidal Fluoroscopy
1	74	F	20	No	31.4	right	decreased
2	73	M	0	Yes	28.4	right	no movement
3	39	M	0	Yes	31.0	right	decreased
4	80	F	44	Yes	27.3	left	decreased
5	78	F	0	No	26.6	right	decreased

or obstructive sleep apnea. All patients were collected from the specialty practices of the authors at Mount Sinai Hospital. Data collection was prospective. All our patients were evaluated with a history and physical examination as well as with standard questionnaires for the assessment of shortness of breath^{14,15} and daytime sleepiness.¹⁶ Standard chest radiography demonstrated unilateral hemidiaphragmatic elevation, which was then studied with fluoroscopy.¹⁷ For the purposes of this study, we declared UDP present when in the seated upright position the involved diaphragm demonstrated reduced movement on tidal breathing, vital capacity, and unobstructed sniff maneuvers. Computed tomography of the chest was performed to exclude intrinsic lung disease and malignant causes of UDP. Lung function testing was performed including whole body plethysmography and maximal mouth pressures by standardized methods¹⁸⁻²² with comparison to predicted values. Five patients qualified of 11 who were evaluated in detail. The 6 who did not qualify had either significant chronic obstructive lung disease as defined by an $FEV_1 < 80\%$ of predicted as well as a ratio with FVC < 0.70 ,²³ or significant sleep apnea/hypopnea with ≥ 5 apneas and hypopneas per hour of EEG sleep.²⁴ Five control patients matched by age, gender, and BMI were chosen by review of patents also undergoing polysomnography. This study was approved by the Ethics Committee of the Mount Sinai Hospital Center.

Laboratory Nocturnal Polysomnography Assessment (PSG)

Participants were monitored in a supervised sleep laboratory from 22:00 to 07:00. Monitoring included 3 leads EEG, EOG, bilateral anterior tibialis and chin EMG, ECG, pulse oximetry, nasal and oral airflow with nasal pressure cannulae (a thermistor for back-up if technical difficulties were detected during recording), and inductance plethysmography for measurement of respiratory effort.²⁵ Body position was monitored with continuous video. All signals were acquired on a digital data management system (Sandman, Tyco and Covidien, Ottawa, Canada). A certified polysomnographic technologist (RPGST) with over 10 years of experience manually scored the studies blind to the results of symptom assessments. Sleep stages were first scored in 30-sec epochs according to standard criteria. Next, EEG arousals were scored according to standard current consensus criteria.²⁶ An apnea event was scored when there was a cessation of breathing for ≥ 10 sec. A hypopnea was defined a priori as an event lasting ≥ 10 sec with a decrease $> 50\%$ from baseline in the amplitude compared to the mean of the largest 3 breaths over the previous 4 epochs, or a lesser reduction in airflow signal amplitude accompanied by $\geq 3\%$ desaturation or an EEG arousal.²⁷ Each 30-sec epoch from lights out to lights

on was reviewed for mean oxygen saturation and the stages of wake and sleep.

Statistical Analysis

All sleep/wake epochs were taken as the unit of analysis when contrasting the oximetry values between sleep stages and the sleep positions. Comparisons were performed with *t*-tests for continuous variables and χ^2 tests for proportions. Analysis with log-transformed oximetry values revealed no significant differences from the values presented. Multiple linear regression was performed on untransformed data to estimate the significance of measured variables in predicting oximetry per monitored epoch. Significance was declared when a p-value of 0.05 was reached after correction for multiple comparisons. Post hoc power calculation for the minimum power (81 epochs of REM sleep on the unaffected side) estimates a β error of 0.89 to detect a 1% difference in mean oximetry. The effect size estimates to determine the mean saturation in REM sleep between patients with UDP and controls revealed a Cohen's *d* value of 1.7 (95% confidence intervals of 1.1 to 2.4). For determining the difference in mean saturation in supine sleep the value was 0.63 (0.0 to 1.26).

RESULTS

Case History, Patient 1 (Table 1)

A 74-year-old female ex-smoker of 20 pack-years was referred because she felt she could not sleep on her left side. She had suffered 2 episodes of wheezing after respiratory infections in the past year without persistent cough. She received a provisional diagnosis of asthma but felt no symptomatic improvement with either salbutamol or formoterol and budesonide and stopped these medications because of side effects. She was also diagnosed with hypertension and was treated with hydrochlorothiazide. She had never undergone any surgery. On inquiry she reported stable grade 3 dyspnea while walking her routine 6 km for the past 5 years. She felt no excessive daytime sleepiness with a score of 8/24 on the Epworth sleepiness scale. She told 3 different pulmonary specialists on 3 separate occasions that she preferred to sleep on her right side. Her score on the St. George respiratory questionnaire²¹ were: symptoms 45%; activity 73%; impact 44%; total 53%. All prior chest x-rays demonstrated a right-sided elevated hemidiaphragm (**Figure 1**) without other abnormality on computed tomography of the chest. Fluoroscopy revealed markedly reduced but concordant movement of the right hemidiaphragm on tidal breathing and slow vital capacity maneuvers, and paradoxical movement on rapid sniff inspira-

tion. Pulmonary function testing revealed a restrictive profile with normal mouth pressures (Patient 1 in **Table 2**). Nocturnal oximetry over 8.06 hours revealed desaturations in clusters to as low as 81%, with only 1.2% of the total time at a saturation < 90% and a mean saturation of 94%. Polysomnography demonstrated prolonged hypopneas in REM sleep without apneas or snoring (**Figure 2**). There was no evidence of polycythemia, with a hemoglobin of 157 g/L and a hematocrit of 0.46. The possibility of nocturnal oxygen was discussed but the patient declined the treatment. A second polysomnogram 12 months later showed similar hypopneas and desaturations. A third polysomnogram performed 18 months later again and then a fourth 6 years after the first continued to show a similar pattern.

Case Series with Unilateral Diaphragmatic Paralysis

Four other patients also met the study criteria (**Table 1**). Three of the 5 had undergone prior surgery to the same side of chest or abdomen. All had MRC grade 3 dyspnea and rated their shortness of breath on maximal exertion on a 10-point Borg scale with a mean of 7.2 (range 4 to 10). Three also had at least mild orthopnea. The mean Epworth scale score was 7.0 (range 5 to 8). Only one demonstrated on fluoroscopy no visible movement with tidal breathing of the affected hemidiaphragm. The remaining patients all demonstrated markedly reduced movement of the affected hemidiaphragm with tidal breathing and slow vital capacity maneuvers. Seated sniff testing demonstrated paradoxical motion with upward movement of the affected hemidiaphragm in all participants. Lung function testing revealed a restrictive pattern in the majority with normal or near-normal mouth pressures (**Table 2**).

On nocturnal polysomnography, the mean total sleep time was 4.7 h (3.5-6.4) with a sleep efficiency of 69% (range 57% to 84%). The sleep stages were distributed with a mean stage 1 percent time of 11.9% (10% to 15.8%); stage 2, 42.7% (30.7% to 52.8%); slow wave sleep of 26.9% (12.8% to 43.7%); and REM sleep with a mean of 18.5% (11.7% to 22.6%). The mean apnea-hypopnea index was 2.9/h (range 0.44 to 4.6). Only 2 of the 5 case patients had any apneas during polysomnography with a mean of 1.6 (range 0-5) apneas per night (**Table 3**); 8 of the 141 desaturations were associated with apneas. Overall, a cumulative 18% of REM sleep time was spent with a saturation below 90%, with a mean time below 90% saturation of 7.8 (5.6) minutes. Desaturations were mostly seen due to prolonged hypopneas in REM sleep, which were coincident with phasic REM episodes without snoring or abdominal paradox (**Figure 3**). These occurred more often and more deeply when

compared to awake or other sleep stages (**Table 4**). Depending upon the patient, the hypopneas lasted a mean 30.4 to 36.4 sec. Some of the desaturations classified as wake or stage 1 epochs followed REM sleep associated hypopneas. Sleeping on the unaffected side was also associated with more desaturations and lower mean oximetry (**Table 5**). When comparing cases to control patients, mean oximetry was lower in all sleep stages as well as all positions ($p < 0.001$ of all comparisons of **Tables 4** and **5**). Sleeping supine resulted in fewer desaturations than when on the unaffected side but more than when sleeping on the affected side. When the analysis was stratified by sleep-wake states and position, this positional pattern of hypopneas with desaturations was seen in both REM and NREM sleep, but more pronounced in REM sleep (**Table 6**). The positional effect was such that when sleeping on the unaffected side, two-thirds of REM sleep time was spent at a saturation < 90%. Multivariate analysis by epoch revealed that the most important factors contributing to low oxygen saturation were REM sleep followed by sleeping on the unaffected side and body mass index (**Table 7**). Tidal volume and oxygen saturation were maintained

Figure 1—Chest radiogram of case 1

Table 2—Pulmonary function results of case patients

Patient	FEV1 (%)	FVC (%)	TLC (%)	Pi max (%)	Pe max (%)	Dlco (%)
1	1.10 (61)	1.48 (65)	2.59 (76)	70 (135)	122 (179)	11.3 (70)
2	1.15 (52)	1.6 (57)	3.56 (70)	65 (77)	119 (112)	12.15 (53)
3	1.4 (46)	1.97 (54)	2.78 (53)	55 (50)	137 (85)	13.86 (47)
4	1.58 (94)	2.24 (102)	4.15 (107)	45 (87)	65 (96)	8.47 (55)
5	1.75 (91)	2.16 (83)	3.8 (77)	40 (77)	60 (88)	12.87 (66)

FEV1, forced expiratory volume in the first second; FVC, forced vital capacity; TLC, total lung capacity; Pi max, maximal inspiratory mouth pressure; Pe max, maximal expiratory mouth pressure; Dlco, diffusing capacity of carbon monoxide in the lung.

Figure 2—Polysomnography hypnogram of case 1

Note the prominent desaturations in REM sleep.

Table 3—Respiratory events by type and position for individual participants

Participant	OA supine	OA side	CA supine	CA side	H supine	H side	AHI supine	AHI side	AHI total
UDP 1	0	0	0	0	38	0	12.3	0.0	4.8
UDP 2	0	0	0	0	13	2	13.0	0.8	4.3
UDP 3	0	0	0	0	0	3	0.0	0.7	0.7
UDP 4	0	0	0	0	9	4	2.6	1.4	2.0
UDP 5	0	0	0	0	25	4	10.4	1.0	4.5
Control 1	0	0	0	0	1	4	0.6	1.1	0.9
Control 2	10	0	1	0	4	0	4.5	0.0	2.4
Control 3	0	0	0	0	4	2	5.7	0.5	1.2
Control 4	13	0	2	0	6	0	11.7	0.0	3.6
Control 5	0	0	0	1	0	7	0.0	1.5	1.3

when sleeping with the affected side down in all sleep-wake states. No flattening of the inspiratory limb of the flow signal was detected during the hypopneas detected in REM sleep. Four of the 5 patients (including the sentinel case) had repeat polysomnography > 1 year later, with no change in the pattern of sleep disordered breathing.

DISCUSSION

In our study participants were selected by having unilateral diaphragmatic paralysis (UDP) without significant COPD

or OSA on polysomnography. Most had restrictive physiology on spirometry and plethysmography. We demonstrated that frequent events characterized by sustained oxygen desaturation occurred in REM sleep. These events were rarely associated with apneas but rather were related to prolonged hypopneas clustered during REM sleep. This effect was more pronounced when the patients were sleeping supine, especially when sleeping on the side unaffected by the UDP. The polysomnographic profile of the disordered breathing is of prolonged hypopneas without the characteristic signs of upper airway obstruction.

Figure 3—Example of the polysomnographic record depicting prolonged REM sleep hypopnea.

The frame duration is 5 minutes, with the patient sleeping supine. The flow signal is a thermistor.

Table 4—Oximetry compared by sleep stages

Participants with unilateral diaphragmatic paralysis

Stage	Epochs	Mean (SD) oximetry	Desaturations (total number)	Minimum saturation	% time < 90% saturation
REM	529	91.4 (3.8)*	94	64	17.8%*
Wake	1181	93.6 (1.5)	11	77	0.9%
1	334	93.1 (2.4)	10	67	3.0%
2	1221	93.3 (1.3)	10	88	0.8%
3+4	726	92.8 (1.5)	16	89	2.2%

Control patients

Stage	Epochs	Mean (SD) oximetry	Desaturations (total number)	Minimum saturation	% time < 90% saturation
REM	438	95.9 (1.4)	0	91	0.0%
Wake	593	95.1 (1.7)	3	88	0.5%
1	738	95.3 (1.8)	4	87	0.5%
2	2010	95.1 (1.5)	6	86	0.3%
3+4	308	94.8 (1.0)	0	90	0.0%

*p-value < 0.001 compared to all other sleep/wake stages. % time < 90%, percent of time less than 90% saturation.

Table 5—Oximetry compared by sleep position**Participants with unilateral diaphragmatic paralysis**

Side	Epochs	Mean oximetry	Desaturations	Minimum	% time < 90%
Unaffected	1352	92.7 (2.3)*	90	67	6.7%*
Affected	1208	93.2 (1.4)	2	89	0.2%
Supine	1431	93.2 (2.3)	49	64	3.4%**

Control patients

Side	Epochs	Mean oximetry	Desaturations	Minimum	% time < 90%
Unaffected	908	95.2 (1.9)	3	86	0.3%
Affected	1819	95.8 (1.1)	1	88	0.1%
Supine	1302	94.4 (1.5)	9	87	0.7%

*p-value < 0.001 compared to sleeping on the affected side or supine. **p-value < 0.001 compared to sleeping on the affected side. % time < 90%, percent of time < 90% saturation.

Table 6—Oximetry profiles stratified by sleep stage and side

Stage	Side	Epochs	SpO ₂	SD	p vs. Wake*	CI 95%.	CI 95%	p vs. REM*	Desats	Min	% time < 90%	p vs. Wake*	p vs. REM*
Wake	Unaffected	465	93.3	1.4	1	93.1	93.4		8	77	1.7%	1	
	Affected	307	94.1	1.1	1	94.0	94.2		0	91	0.0%	1	
	Supine	409	93.5	1.6	1	93.3	93.6		3	82	0.7%	1	
NREM	Unaffected	792	92.8	1.6		92.7	93.0	< 0.0001	28	85	3.5%**		< 0.0001
	Affected	604	93.0	1.2		92.9	93.1	< 0.0001	0	90	0.0%		0.0435
	Supine	885	93.5	1.7		93.4	93.6	< 0.0001	8	67	0.9%		< 0.0001
REM	Unaffected	81	87.8**	5.3	< 0.0001	86.7	89.0	1	54	67	66.7%**	< 0.0001	1
	Affected	297	92.7	1.7	< 0.0001	92.5	92.9	1	2	89	0.7%	0.1503	1
	Supine	151	90.8	4.5	< 0.0001	90.1	91.5	1	38	64	25.2%***	0.0001	1

*The mean SpO₂ or % time < 90% is compared in NREM vs. Wake or NREM vs. REM. **p-value < 0.001 compared to sleeping on the affected side or supine.

***p-value < 0.001 compared to sleeping on the affected side. % time < 90%, percent of time less than 90% saturation.

Table 7—Multiple regression of factors to predict pulse oximetry per epoch

Variable	Beta coefficient	Standard error (beta)	t-value	p-value
Age	-0.022	0.0038	-5.66	< 0.0001
Gender (male)	0.63	0.11	5.76	< 0.0001
BMI	0.030	0.025	11.96	< 0.0001
Supine	0.58	0.093	6.29	< 0.0001
Unaffected side	-1.10	0.096	-11.41	< 0.0001
NREM	-0.66	0.070	-9.51	< 0.0001
REM	-2.63	0.10	-25.45	< 0.0001

Regression multiple $r = 0.44$, F-value 133.4. BMI, body mass index; NREM, non-REM sleep; REM, REM sleep.

UDP has been described for decades in case series dating back to 1921.²⁸ Many of the cases of UDP have been described in reports in which patients with generalized muscle weakness or bilateral diaphragmatic paralysis have been included.⁶⁻¹³

UDP may present as an incidental finding on radiography in an asymptomatic patient. However some individuals may have shortness of breath on exertion or orthopnea.² Various etiologies have been described, including surgical trauma, post-infectious paresis, and tumor infiltration. In some cases (labeled idiopathic), no cause is determined.^{1,2,5} Either side may be affected, but case series have suggested that right-sided UDP is more common.¹ When the cause is benign, patients tend to remain with stable radiographic and symptomatic manifestations. Occasionally spontaneous regression may occur.¹⁻⁴

Imaging for confirming the diagnosis of the UDP has traditionally been dynamic fluoroscopy with upright tidal breathing, vital capacity maneuvers, and rapid inspiration with a closed mouth in a “sniff” maneuver.⁵ Ultrasound to evaluate diaphragmatic excursion has also been found to be useful.²⁹

Normal individuals when supine have a similar contribution of the chest wall to the tidal volume of quiet breathing when awake or in NREM sleep.³⁰ The chest wall contribution in REM sleep is reduced to approximately half that seen in NREM sleep,³⁰ with a marked reduction of the intercostal EMG and a compensatory increase in diaphragmatic activation to maintain tidal volume.^{30,31} Phasic episodes of rapid

eye movements correlate with immediate breath-by-breath reductions in ribcage excursion and total ventilation in supine REM sleep.³²

In patients with UDP, the explanation for the REM-associated hypopneas observed by us and others requires taking into account the sleep stage dependent and positional nature of these events. Reduction in vital capacity and oxygen saturation can be seen even in the awake supine position.^{1,10,13} When studied awake with detailed transdiaphragmatic pressure profiles, reduced maximal transdiaphragmatic pressures were found.¹³ Two reports have specifically described the sleep related breathing characteristics of patients with isolated UDP.^{10,13} When asleep, inspiration was assisted by contraction of the intercostals muscles with increased activation of accessory muscles. The latter persisted in both REM and NREM sleep.¹³ This persistent activation of the accessory muscles is likely to compensate for the physiologic reduction of intercostal activation and the poor compensation by the diaphragm in maintaining tidal volume. With the typically increased upper airway resistance and low intercostal muscle tone during REM sleep, this may produce increased coupling of the 2 hemidiaphragms where the healthy side contracts and the weak side moves paradoxically with dissipation of respiratory effort and ineffective ventilation. This may explain the hypopneas with little evidence of typical obstruction and desaturations observed in patients with UDP during supine REM sleep. This may also explain why lying on the paralysed side stabilizes ventilation. We did not observe flattening of the inspiratory limb of the flow signal during the hypopneas in REM sleep, which suggests to us that the hypopneas were the result of dissipation of respiratory effort rather than obstructive upper airway resistance.

We found that our patients with UDP the lateral decubitus position with the unaffected side down was most vulnerable to hypopnea and desaturations. This may potentially be explained by the fact that the unaffected hemidiaphragm must generate the majority of the inspiratory diaphragmatic force for tidal breathing. When the healthy hemidiaphragm is dependent, it is most compressed by the abdominal contents and has a higher pressure to counter while generating the force required for inspiration. The chest wall is also immobilized and compressed by the pressure of the body against the bed. Any lack of coordination of the muscle fibers as a result of REM sleep state may compound this state of mechanical disadvantage. We believe that this combination of distorted forces leads to the sleep state and positional influences on the breathing of patients with UDP.

We here describe REM sleep disordered breathing in this series of patients who all are strictly characterized as having neither obstructive sleep apnea nor obstructive lung disease. We extend these previous findings with detailed analysis of the positional nature of these hypopneas. Our findings are different from those reported by Steir et al.¹³ in that the sleep position most likely to incur desaturations was with the unaffected hemidiaphragm down, followed by to a lesser extent the supine position. The previous polysomnographic series described that the patients preferred to sleep with the unaffected hemidiaphragm down, a preference not shared by our sentinel case. The discrepancy between these studies regarding the influence of sleep position is unclear and may warrant further study.

The repeated polysomnographic monitoring demonstrating similar findings up to 6 years suggests that the sleep disordered breathing seen in UDP may persist as a stable abnormality over a prolonged period. This is consistent with clinical observations published elsewhere when the patients were found to have idiopathic cause without intrinsic parenchymal lung disease.^{1-4,10}

This study had several limitations. We excluded patients with concomitant sleep apnea and significant obstructive lung disease, so the findings here cannot be generalized to patients with combined pathologies which were often present in previous studies. However, one may speculate that any intrinsic lung disease might actually accentuate the effect of UDP in sleep. Our 5 patients may not necessarily be representative of all patients with UDP. Invasive transdiaphragmatic monitoring was not performed to ascertain maximal inspiratory pressures or electromyography. This has been studied elsewhere.¹³ Yet all patients had significant amounts of all sleep stage categories and slept spontaneously in several positions, providing detailed data to study the various combinations of sleep states and positions.

In conclusion, this study describes the type of sleep disordered breathing found in patients with UDP without obstructive sleep apnea or obstructive lung disease. The hypopneas are often in concert with phasic bursts of rapid eye movements, and cause important desaturations especially in REM sleep. These desaturations were most frequent and more severe when sleeping with the unaffected hemidiaphragm down.

ABBREVIATIONS

BMI, body mass index
COPD, chronic obstructive pulmonary disease
FEV₁, forced expiratory volume in 1 second
FVC, forced vital capacity
MRC, Medical Research Council dyspnea score
PSG, polysomnography
SD, standard deviation
SGRQ, St. George respiratory questionnaire
SpO₂, oxygen saturation
UDP, unilateral diaphragmatic paralysis
VAS, visual analogue scale

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