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Sleep in Postpolio Syndrome*

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Post-polio patients may develop additional neuromuscular and respiratory symptoms decades after the acute attack, the post-polio syndrome. We hypothesize some post-polio symptoms may be due to breathing disorders occurring during sleep. We performed polysomnography on 13 post-polio patients: group 1 (five patients) were those already on ventilatory assistance (rocking beds) and group 2 (eight patients), those without any assistance. Patients requiring new treatment were then evaluated on nasal CPAP or nasal mask ventilation. Group 1 patients, on rocking beds, demonstrated consistently poor sleep quality with decreased total sleep time, sleep efficiency, percentage stage 2, slow wave sleep, rapid eye movement sleep and an increase in the number of arousals and percentage stage 1 sleep. Respiratory abnormalities were also present and in all cases caused significant O_2 desaturation. These patients did not respond to CPAP with the rocking bed. Repeat night-time polysomnography on nasal mask ventilation demonstrated an improvement in sleep structure and gas exchange. Three group 2 patients, (group 2a) had sleep within normal limits. The five remaining (group 2b) had poor sleep quality that was similar to but not as disrupted as group 1 patients. All but one patient demonstrated obstructive or mixed apnea and were treated effectively with nasal CPAP. One patient required nasal mask ventilation (due to mixed apnea and marked hypoventilation) to which there was a dramatic response. These patients demonstrated improved sleep quality and an improvement in daytime symptomatology. Sleep studies should be performed on post-polio patients with excessive daytime sleepiness and respiratory complaints. Those with obstructive and mixed apnea can often be treated with nasal CPAP. Those with hypoventilation syndrome and sleep apnea attributable to sleepiness and respiratory complaints. Those with obstructive and mixed apnea can often be treated with nasal CPAP. Those with hypoventilation syndrome and sleep apnea attributable to respiratory muscle weakness can be treated with nasal mask ventilation. Individuals already on respiratory assistance such as rocking beds who have features of respiratory failure can also be treated effectively with long-term nasal mechanical ventilation. ***(Chest 1990; 98:133-40)***

Patients who had poliomyelitis were frequently clinically stable three to four decades after the acute attack. Deterioration of overall function then occurred in approximately 25 percent of the patients.[1] Two mechanisms have been hypothesized to cause the worsening when it involved respiration-superimposition on an already compromised respiratory system by the effects of aging, or the postpolio syndrome.[2] The features of this syndrome are fatigue, new weakness in muscles (both those originally affected and those unaffected), pain in muscles or joints, dysphagia, and hypoventilation.[3] Since apnea during sleep has been described in postpolio patients,[4] we wondered whether abnormal sleep related to impaired ventilation may cause some of the symptoms of the syndrome. Since sleep is the period of worst gas exchange when compromised lung function is present, we hypothesized that sleep studies would be helpful in detecting patients with remote poliomyelitis whose respiratory insufficiency was undetected or undertreated. Accordingly, we studied sleep in two groups of stable postpolio patients: those already receiving ventilatory assistance and those who were not. Patients requiring additional ventilatory assistance were then evaluated on various ventilatory devices.

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Patients and Methods

Thirteen patients were recruited for the study from the local postpolio support group from April 1988 to August 1988. These individuals were thought to be clinically stable but had subjective complaints of progressive fatigue, weakness in muscles, pain in muscles or joints, sleep problems, breathing difficulties and swallowing problems consistent with the diagnosis of the postpolio syndrome.

These 13 patients (Fig 1) were divided in two groups: group 1 consisted of five patients (two women, three men with a mean age of 60.2 years ± 2.1 SD, range 58 to 63) who were on rocking beds only at night for respiratory assistance (Fig 2); group 2 consisted of eight patients (seven women and one man with a mean age of 55.9 years ± 13.3 SD, range 30 to 70 years) who were not receiving any respiratory assistance. This latter group was broken down *post hoc* into: 2a, those who showed no sleep abnormalities and required no treatment, and 2b, those who had sleep abnormalities and required treatment. The degree of physical disability is shown in Figure 1.

After history and physical examination, the patients had pulmonary function tests, arterial blood gas determinations, and a complete blood count. Polysomnography was performed on all patients.

Each patient spent two or three consecutive nights in the sleep laboratory. The first was an adaptation night and the second was the study night. The third night was used to initiate treatment if required. Treated patients returned in four to six months for a follow-up study with polysomnography for a further two nights, the first being the adapatation night and the second being the study night on treatment. The patients were fully instrumented on all nights (see below) with measurements being recorded. All patients were studied while breathing room air. The patients went to bed at 10:30 to 11:00 PM, and the study was terminated between 7:00 and 7:30 AM. Routine schedule and regular medications, if taken, were maintained during the study. None was receiving medication known to affect respiration.

We recorded the EEG, EOG, and EMG from surface electrodes. Arterial oxygen saturation was continuously recorded with a pulse ear oximeter set on its fastest response. Respiratory movement was monitored by inductance plethysmography with transducers placed around the chest and abdomen. The ECG and heart rate were continuously recorded from standard limb leads. Airflow was detected by monitoring expired CO_2 at the nose and mouth through nasal/oral CO_2 cannula attached to a CO_2 analyzer. All variables were continuously recorded on a polygraph at a paper speed of 10 mm/s. A microcomputer continuously monitored airflow and respiratory movement and stored SaO_2 and heart rate twice for each complete respiratory cycle on a mass storage medium. The computer generated a binary stamp on the polygraph so that the polygraph data and computer data could be synchronized during later analysis.[5]

A rocking bed was brought into the laboratory for the group 1 patients. The bed was set up and "tuned" to conform with the patient's own bed. The contacts on the electrode input box were reinforced to minimize motion artifact on the neurophysiologic recordings.

Respiratory and sleep stage variables were analyzed independently. The polysomnogram was scored manually for sleep stage and arousals according to established criteria using the EEG, EOG, and EMG records.[6] The polysomnograms were also manually scored for respiratory events. A computer program performed an integrated analysis of respiration and sleep stage.[5]

The following definitions were used to classify breathing patterns and arousals: apnea was defined as the absence of airflow for more than 10 seconds. In central apnea, respiratory effort was absent, whereas in obstructive apnea, respiratory efforts continued. Hypopnea was defined as a reduction the amplitude of respiratory movement for more than 10 seconds to less than 50 percent of the maximum thoracoabdominal amplitude during the breathing cycle.[7] Paradoxic respirations were defined as out of phase chest and abdominal wall movement as indicated by inductance plethysmography. An arousal was defined as an awakening from sleep for >5 seconds as shown by alpha activity on the EEG, EMG activation, and eye movements which occurred simultaneously. The apnea hypopnea index is the number of apneas and hypopneas per hour of total sleep time.

In the group 1 patients, since rocking beds maintained "respiratory effort," their apneic or hypopneic episodes were documented as loss of airflow (as indicated by absence of CO_2 measured at the nose and mouth). These episodes most closely resembled obstructive episodes (Fig 3). Even though the rocking beds generate constant respiratory efforts, hypopnea is seen as a reduced displacement of abdomen and ribcage similar to hypopnea in a routine study as described previously. All group 1 patients were evaluated first on CPAP then on nasal ventilation.

The group 2 patients thought to require ventilatory assistance were first tried on nasal CPAP and on nasal ventilation if CPAP was insufficient.

The protocol was approved by the Ethics Committee at our institution and a written informed consent was obtained from each patient.

Details of CPAP and Mechanical Ventilation Application

The patients who demonstrated obstructive sleep apnea, mixed apnea, or hypoventilation syndrome were first tried on nasal CPAP with an appropriately fitting nasal mask. The CPAP was started at 5 cmH_2O at night while polysomnography was being done. The CPAP was titrated in increments of 2.5 cmH_2O pressure in order to determine a proper level and the efficacy of the treatment. Those who were treated were followed up in four to six months with night polysomnography to ensure continuing effectiveness of treatment and a clinical evaluation of patient's general condition.

Those who were not treated effectively with CPAP then received nasal mask intermittent positive pressure ventilation with a portable home volume ventilator. This ventilator was set up in a time cycled pressure limited mode using the Lifecare pressure limiting valve. The patients were fitted with two types of nasal masks which were used on a rotational basis to avoid consistent pressure points. These systems were tested while polysomnography was being done to ensure efficacy throughout NREM and REM sleep. These individuals were ventilated with room air. An artificial nose, placed between the exhalation valve and the patient, was used in line if necessary for humidification. The air was driven through the pressure limit valve attached to corrugated tubing, one way valve, exhalation valve, artificial nose, inspiratory assist line, and patient nasal mask. The one way valve essentially provided for a noncontaminated

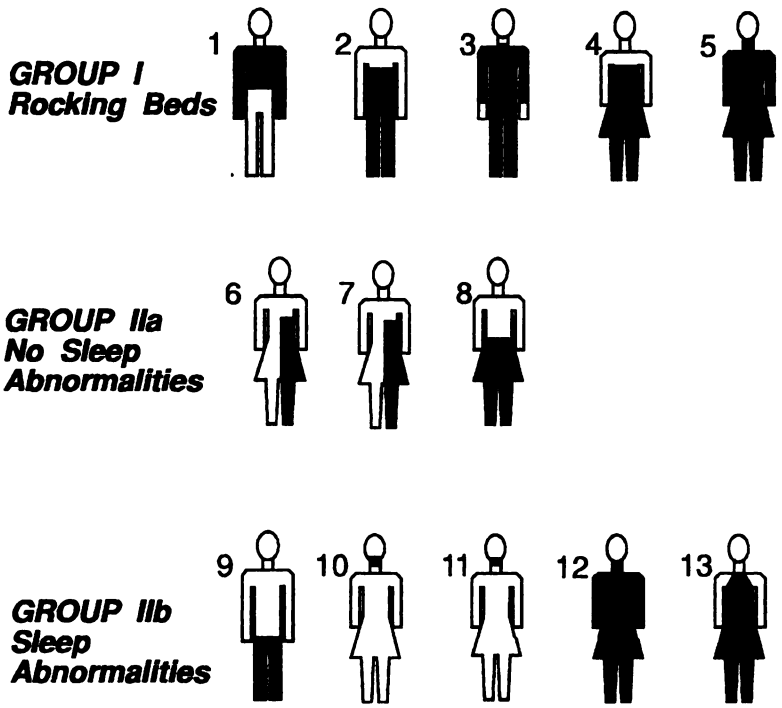

FIGURE 1. The 13 postpolio patients. Shaded areas represent patient's regions of paralysis.

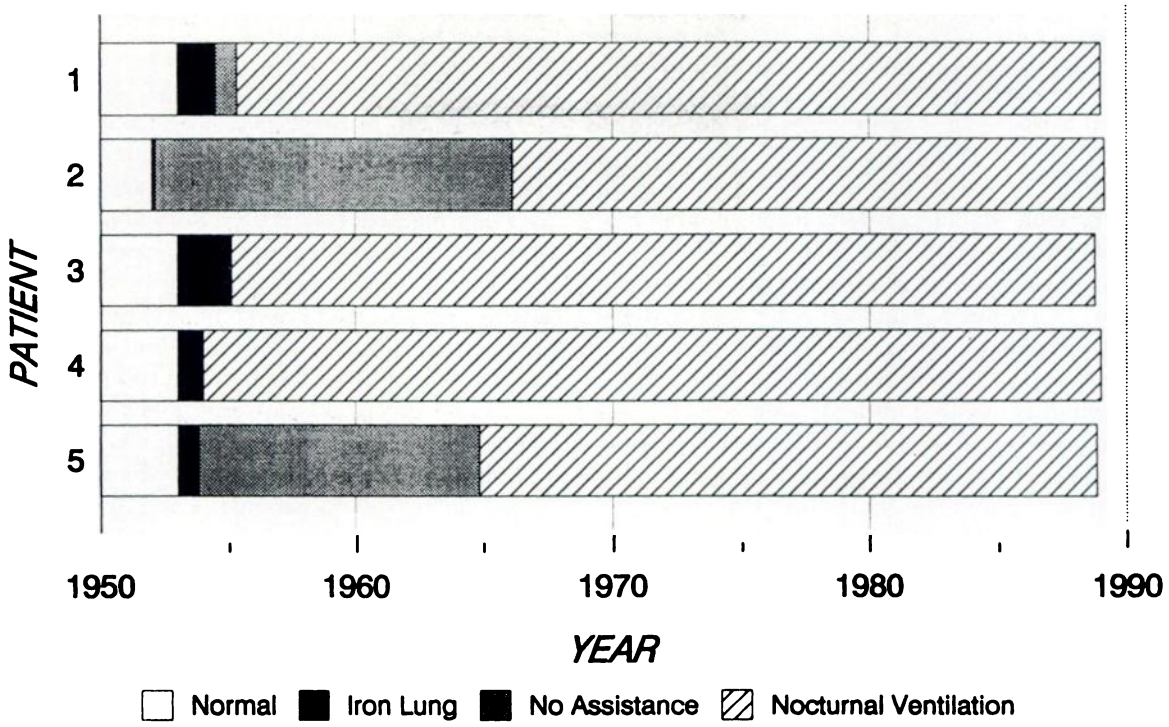

FIGURE 2. Clinical course of group I patients.

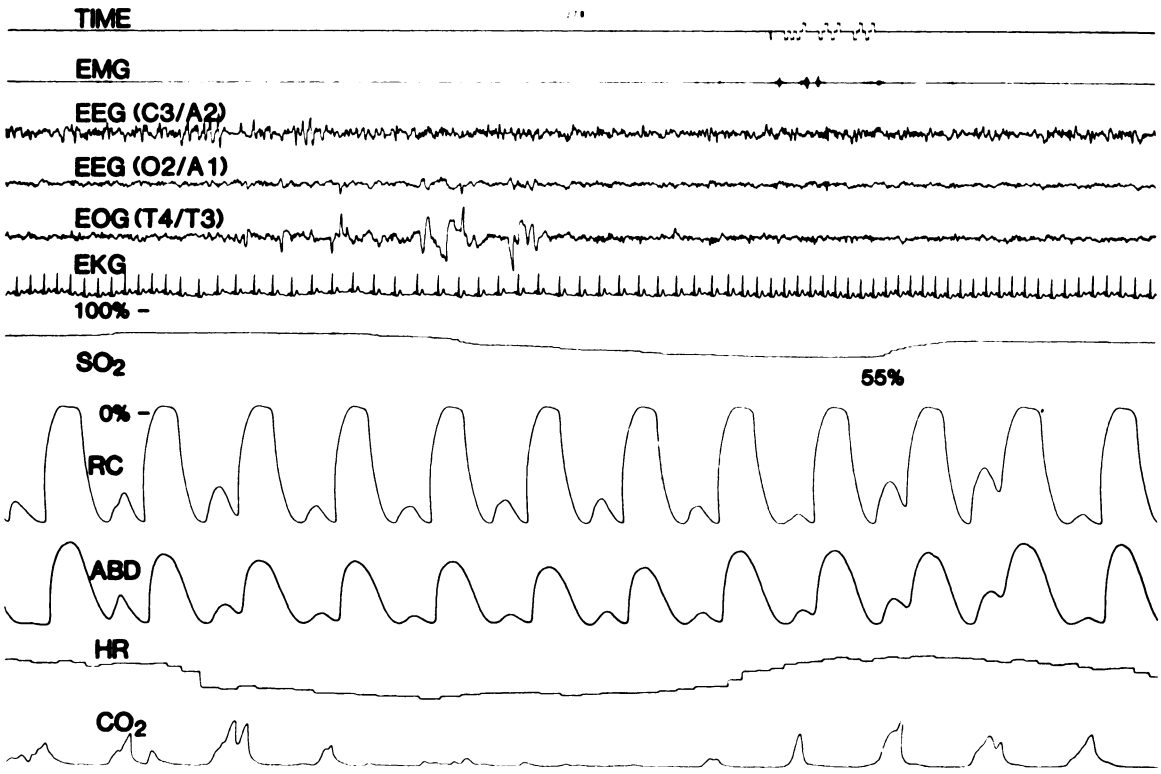

FIGURE 3. Polysomnogram demonstrating EMG, EEG (C3/A2), EEG (O2/A1), EOG (T4/T3), ECG, SaO_2, heart rate (HR), airflow (CO_2) with patient in REM sleep on a rocking bed. Regular large breaths depicted are generated by the rocking bed with smaller breaths in between patient effort. Despite this, there is no substantial airflow detected and a significant drop in oxygen saturation. The tiny CO_2 deflections and the lack of ribcage abdominal paradox suggests that the upper airway is not totally occluded (obstructive hypopnea).

circuit so that circuit cleaning at home would be simplified. These patients were then followed at home on a daily basis for two weeks and then a weekly basis for four weeks, then a monthly basis until approximately six months at which time follow-up polysomnography was done and patients were re-evaluated for improvement or changes in their general condition.

Results

The 13 patients (Table 1) with a mean number of years since polio onset of 39.6 years ± 5.8 SD were studied. Group 1, (36.6 ± 0.4 SD years since onset of polio), had all required ventilatory support at the initial onset of polio in the form of a tank respirator for an average of 9.3 months (range 1.5 to 18 months) prior to attempted weaning. Patients 3 and 4 could not be weaned and required the rocking bed only for nocturnal support but otherwise breathed spontaneously while sitting up. The remaining patients were weaned completely for an average of 8.6 years (range ten to 168 months) after which they were started on rocking beds at night. At the time of the study, all the patients were using nocturnal respiratory assistance for a mean of 10.7 hours per day (range ten to 14 hours) (Fig 2). All of the patients had subjective complaints of respiratory or sleep problems.

There were 39.4 ± 7.3 years since the onset of polio in the eight group 2 patients. Five of the eight did not require any respiratory assistance at the onset of the disease and three of the patients required ventilator support for less than one month in a tank respirator. Subsequently, none of these individuals had used any form of respiratory assistance. These patients had symptoms of fatigue and excessive daytime sleepiness and nonrestorative sleep.

Group I Patients

Patients on rocking beds had consistently poor quality sleep with a lower than normal TST and an increased number of arousals and stage 1 sleep (Table 2). Differing degrees of respiratory abnormality were apparent during sleep. In some cases, there was a desynchronization of respirations with the patient attempting a breath between breaths generated by the bed to intermittent hypoventilation (Fig 3) which occurred throughout the night. In two patients (1 and 2), there were paradoxic respirations during NREM sleep as well as when the patients were awake. Significant oxygen desaturations occurred in all patients (Table 3). Two patients (1 and 3) had high AHI all night, while one (No. 5) had a high AHI only during REM. The remaining two patients had short periods of NREM sleep (stage 1 and 2 only). One patient (No. 2) slept only 88 minutes and demonstrated continuous hypoventilation without discrete apneas or hypopneas and had a mean SaO_2 of 88 percent. The other patient (No. 4) only slept 44 minutes with a similar breathing pattern and had a mean SaO_2 of 87 percent.

The patients on rocking beds were evaluated first on nasal CPAP with the rocking bed and then on mechanical ventilation via nasal mask. Nasal CPAP was not efficacious in this group. Patients became short of breath and were uncomfortable on CPAP.

These patients were then evaluated on the mechan-

Table 1—*Patient Data*

			ABGs			Pulmonary Functions*				
Patient	Sex	Age	PaO_2	$PaCO_2$	pH	VC	FEV_1	FRC	TLC	RV
Group 1 (rocking bed patients)										
1	M	59.1	71	42	7.45	33	40	63	53	87
2	M	63.0	76	50	7.38	27.5	33.4	62	49	89
3	M	57.8	74	53	7.39	15.1	11	. . .	. . .	. . .
4	F	58.7	80	45	7.39	27.8	23.9	55.3	46.2	78
5	F	61.5	73	46	7.41	24.6	23.4	. . .	. . .	. . .
MEAN		60.2	74.8	47.2	7.404	25.4	26.0	60.1	49.4	84.7
±SD		2.1	3.4	4.3	0.028	6.5	11.7	4.2	3.4	5.9
Group 2a (no sleep abnormality)										
6	F	30.5	71	42	7.45	44.2	54.5	104.8	102	68.8
7	F	70.8	88	42	7.41	70.1	82.7	82.9	82.9	102.5
8	F	46.4	71	45	7.41	33.3	26.7	. . .	. . .	. . .
MEAN		49.4	76.7	43.0	7.423	49.2	54.6	93.9	92.5	85.7
±SD		20.3	9.8	1.7	0.023	18.9	28.0	15.5	13.5	23.8
Group 2b (sleep abnormalities)										
9	M	65.5	81	39	7.39	76.5	64.4	76.3	83.6	96.4
10	F	60.6	93	34	7.47	99.2	81.8	92.9	94.8	87.8
11	F	57.8	82	34	7.51	81.2	110	66	75.5	66
12	F	48.1	85	41	7.39	38.1	34.9	65.4	57.9	97.3
13	F	66.1	67	43	7.46	37	40	81	62	101
MEAN		59.8	81.6	38.2	7.444	66.4	64.2	76.3	74.8	89.7
±SD		7.3	9.4	4.1	0.053	27.7	27.5	11.4	15.2	14.1

*PaO_2 and $PaCO_2$ are in mmHg; VC, vital capacity (L); FEV_1, forced expired volume in 1 sec (L); FRC, functional residual capacity (L); TLC, total lung capacity (L); RV, residual volume (L); all PFT values are percent of predicted values.

Table 2—*Sleep Quality

	TST		Sleep Efficiency		Arousals Per Hour		Stage 1, Percent		Stage 2, Percent		SWS, Percent		REM, Percent	
	Control	RX	Control	RX	Control	RX	Control	RX	Control	RX	Control	RX	Control	RX
Group 1 (rocking bed) n = 5														
Mean	225.3	318.9	52.2	70.2	30.5	14.6	35.7	10.5	37.8	50.0	16.5	15.1	8.1	20.2
SD	152.1	70.1	33.3	17.1	21.6	7.0	36.2	4.5	23.4	23.0	12.6	15.2	8.4	13.9
Group 2a (no sleep abnormality) n = 3														
Mean	406.5		93.1		6.5		8.7		57.4		13.6		19.2	
SD	13.5		2.8		1.7		4.7		14.3		11.4		5.6	
Group 2b (sleep abnormalities) n = 5														
Mean	295.5	340.4	67.5	77.0	19.9	11.2	20.1	15.6	40.7	41.4	23.6	24.7	13.7	15.7
SD	64.8	48.2	15.5	7.1	15.6	3.6	13.2	12.1	13.3	6.6	15.1	9.7	6.5	1.4

*TST, total sleep time (minutes); sleep efficiency, percent of time in bed actually asleep; stage times are as a percent of TST; control, pretreatment; RX, on ventilatory assist device (CPAP or nasal ventilation).

ical ventilator, without rocking bed, and again there was a wide variability in response. All but one patient showed a dramatic improvement in their sleep quality and gas exchange. In one patient (No. 1), even though there was improvement in gas exchange, sleep quality did not improve.

Group 2 Patients

There was a marked variability in the quality of

Table 3—*Apnea/Hypopnea Index

	All Night					For NREM					For REM				
		Duration					Duration					Duration			
	AHI	Mean	SD	SaO_2	SD	AHI	Mean	SD	SaO_2	SD	AHI	Mean	SD	SaO_2	SD
Group 1															
1 RB	38.7	14.4	8.2	80.4	7.8	20.9	11.5	4.6	85.8	4.4	114.5	16.6	9.6	76.3	7.4
MV	1.6	16.3	8.8	88	1.5	1.9	16.3	8.8	88	1.5	0				
2 RB†															
MV															
3 RB	56.4	18.5	5.7	88.4	23.1	56.7	18.9	5.6	88.4	22.2	46.1	27.7	9.1	84.9	42.1
MV	1.4	18.9	12.9	86.4	2.7	2.0	15.0	6.0	86.1	2.7	0.5	50.0	0	89.0	0
4 RB‡															
MV	0					0					0				
5 RB	8.8	25	11.5	69.4	11.1	0					60.0	25	11.5	69.4	11.1
MV	0.8	17	5.7	91.4	0.9	1.0	17	5.7	91.4	0.9	0				
Group 2a															
6 SPON	0.1	10	0	94	0	0					0.7	10	0	94	0
7 SPON	0.14	15	0	92	0	0					1.1	15	0	92	0
8 SPON	11.1	25.4	7.4	87	1.6	10.0	23.5	9.3	86.8	2.3	15.4	23.5	9.3	86.8	2.3
Group 2b															
9 SPON	8.0	14.6	9.5	89.1	2.8	10.2	14.6	9.5	89.1	2.8	0				
CPAP	0.4	10	—	94	1.4	0					2.8	10	—	94	1.4
10 SPON	3.9	18	7.3	89.2	1.7	0.9	20.5	4.1	90.2	1.9	28.2	17.5	8	88.9	1.6
CPAP	0					0					0				
11 SPON	4.5	19.9	7.1	84.7	16.1	1.9	18.1	7.4	88.4	0.8	15.9	20.8	6.9	82.8	19.6
CPAP	0					0					0				
12 SPON	10.8	24.5	10.3	92.9	1.2	0					45.8	24.5	10.3	92.9	1.2
CPAP	6.9	21.8	12.1	95.1	1.4	0.7	11.7	2.9	95.7	1.2	42	22.6	12.2	95.1	1.4
13 SPON	65.9	15.1	6.7	88.5	1.2	65.4	14.6	5.4	87.4	1.3	84.1	14.6	5.4	87.4	1.3
MV	1.3	17.2	4.4	92	1.7	1.6	17.2	4.4	92	1.7	0				

*RB, rocking bed; MV, mechanical ventilator; CPAP, nasal CPAP; SPON, spontaneous unassisted; Duration, mean apnea/hypopnea duration; SaO_2, mean nadir SaO_2 for all the episodes.

†Patient 2 RB slept 88 minutes with continuous hypoventilation (without discrete apneas or hypopneas), with the average SaO_2 during sleep of 88%; on MV, slept for 282 minutes with intermittent hypoventilation (without discrete apneas or hypopneas) due to mouth leakage and had an average SaO_2 of 86.9 percent SD 2.0.

‡Patient 4 RB slept 44 minutes with continuous hypoventilation (without discrete apneas or hypopneas) with mean SaO_2 of 87 percent; respiration was normal on MV.

sleep in these patients varying from extremely poor to within normal limits. Three of the eight patients (group 2a) had results within normal limits. The remaining patients (group 2b) had varying degrees of sleep apnea and hypopnea with poor sleep quality. One of these patients with an apnea hypopnea index of 11.1, had little resultant sleep disruption (Table 3).

Four of the five group 2b patients had a snoring history. One patient did not know whether she snored. One of the patients (No. 9) had an elevated hemoglobin value with symptoms of fatigue, excessive daytime sleepiness, and ankle edema of unknown etiology. He was found to have mild obstructive sleep apnea.

Two patients (No. 10 and 11), with a history of bulbar polio, were found to have mild obstructive sleep apnea which was most severe in REM sleep. In both these patients, the vocal cords were found to be paralyzed and accounted for the obstructions. There was an audible high pitched stridorous noise present in one of the patients when she experienced obstruction while she slept. It is likely that there was significant hypoventilation secondary to the vocal cord paralysis with increased resistance in the larynx in these patients. Both patients had excessive daytime sleepiness. These patients were started on nasal CPAP with improvement of their symptoms. Both patients had dysphagia and complained of stridor with exercise.

Of the remaining two patients, one (No. 12) had symptoms of morning headaches, nausea, vomiting, excessive daytime sleepiness, ankle edema, and fatigue. Studies demonstrated mixed apnea that was severe in REM sleep. This patient was started on nasal CPAP with a slight reduction in the number of episodes. The headaches were ameliorated, but she refused long-term treatment.

The last patient (No. 13) had severe mixed apnea. She could not be maintained on CPAP because of continuing hypoventilation, and therefore was started on nasal mask ventilation utilizing the same techniques as the patients in group 1 (Table 4).

Long-Term Follow-up

Six of the patients have now been receiving mechanical ventilation for a mean of 9.3 months. Because the patients were no longer reliant on rocking beds, in several cases, they were able to travel extensively for the first time in over three decades. Two patients previously unable to function at work were able to continue employment.

Complications: While receiving mechanical ventilation, mask leaks or leaks through the mouth were present in all the patients. The mechanical ventilator was set to deliver a much greater volume (Table 4) than the patient actually received. The goal was to increase the machine-delivered volume until that actually delivered to the patient was about 10 ml/kg. One of the patients required a chin strap. All the patients were given two masks to take home. One of the patients who had had a carcinoma of the skin removed in the area of his nose had irritation when he first started on nasal ventilation and required the evaluation of several different nasal masks and oral/nasal masks before the system was adequate. Two patients had traumatic accidents with fractures and required nasal mask ventilation continuously for several days. These patients had a breakdown of the soft tissue on the bridge of the nose by the nasal mask as it was not rotated with any regularity by nursing staff in acute care facilities. These areas healed with time and the introduction of alternate mouth and nasal masks which changed pressure points.

Patients requiring increased humidity because of nasal dryness had artificial nose humidifiers inserted into the patient circuit with the desired effects.

Discussion

Sleep quality may be very poor in postpolio patients and the poor sleep quality may be related to abnormal breathing during sleep. Because of the effect of polio on respiratory control and respiratory function, the variability in these results was not unexpected. Somewhat unexpected was the fact that several of the patients who were receiving mechanical ventilatory assistance (rocking beds) were clearly not being optimally treated. In two patients on rocking beds, there was paradoxic rib cage abdominal breathing. In two

Table 4—*Ventilator Settings**

Patient No.	Delivered V_T, ml	RR	I/E Ratio	Flow Rate, LPM	Pressure Limit Valve, PSI	Patient's Mean Airway Pressure, cmH_2O	Low Pressure Limit, cmH_2O	Patient's Actual V_T, ml
1	900	15	1:2.3	68	30	10-12	7	590
2	930	16	1:1.7	62	35	18-20	12	600
3	980	15	1:4.0	74	35	16-18	10	680
4	930	15	1:4.0	87	35	20-22	15	650
5	1050	14	1:1.9	64	35	22-24	15	550
13	1000	14	1:3.3	86	40	35-37	25	600

*Patients were all on assist/control mode. V_T, tidal volume delivered by ventilator (ml); RR, respiratory rate; I/E ratio, inspiratory/expiratory ratio; flow rate, liters per minute; pressure limit value, pounds/square inch.

patients, there was the desynchronization of respirations with the patient attempting a breath between breaths generated by the bed, at times leading to upper airway obstruction likely secondary to desynchronization between the patient's upper airway and the rocking bed. The fact that they had been apparently stable for so long led to a false impression of the stability of their ventilatory status. Patients with remote poliomyelitis who are not currently on mechanical ventilation should be assessed for a sleep disorder, particularly hypoventilation and/or obstructive apnea if clinically they have developed some of the daytime features suggestive of sleep respiratory abnormalities. These features include excessive daytime sleepiness, disrupted sleep, snoring, and the unexplained development of peripheral edema and/or polycythemia.

In all cases, the patients far preferred nasal ventilation to rocking beds in part because of the psychosocial aspects and the mobility afforded by the portable ventilator.

Our experience in being able to nasally ventilate postpolio patients is similar to findings recently reported by other centers.[8] In most patients using nasal mask ventilation, there are several practical aspects to consider for successful long-term ventilation. First, there may be leaks in the system, particularly through the mouth during the inspiratory phase initiated by the mechanical ventilator. Leak compensation is best achieved by increasing tidal volumes and pressure limiting these volumes.

For several leaks caused by the jaw dropping, particularly in REM sleep, a chin strap, a mouthseal or even a single strip of tape can be used to seal the lips. It is possible that in some patients, the leak cannot be compensated for, but this is surprisingly uncommon.[8] Such patients may eventually require other airway options.

It is also important to have a minimum of two different nasal masks and alternate their use, as this will avoid consistent pressure points that can develop, particularly if the patient has an acute medical problem and requires extended hours of use.

It is also noteworthy that one patient outside the study who was ventilating with a mouthpiece has been stable for 15 years, and switching him to nasal ventilation did not result in any changes as nocturnal ventilation was acceptable. Fitting mouthpieces for nocturnal ventilation is much more costly and generally not as effective because of the movement of the lower mandible.

Postpolio patients receiving nocturnal ventilation may develop respiratory muscle fatigue in the daytime. Treatment should then also include respiratory assistance during the daytime in order to minimize respiratory muscle fatigue. For mobility, this requires an external battery to operate the ventilator and can be adapted to a wheelchair or cart mount. For daytime use, often a mouth hook or mouthpiece are more practical.

In some of these patients, the symptoms frequently attributable to the "postpolio syndrome" were linked to the sleep breathing abnormalities because the symptoms regressed when nocturnal ventilation was improved. It is thus reasonable to perform a sleep study on patients with poliomyelitis whose symptomatology is unexplained and whose daytime features suggest the development of abnormalities in respiration during sleep and subsequent poor sleep quality.

There has been a tendency in some centers to perform tracheostomies when the polio patients on their current mode of ventilation start to develop features of respiratory failure. Our data suggest that the nasal airway is an adequate airway in many of these patients and use of nasal ventilation may postpone or obviate the need for tracheostomy in many postpolio patients. Nasal ventilation is becoming an option in a wide variety of patients with disorders requiring mechanical ventilation,[8-13] and this trend is likely to continue with improvements in nasal mask design. One cannot overemphasize the change in quality of lifestyle that has resulted in the patients no longer being reliant on their rocking beds.

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