

Adaptation to intermittent positive pressure ventilation applied through the nose during day and night

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Adaptation to intermittent positive pressure ventilation applied through the nose during day and night. D.O. Rodenstein, D.C. Stanescu, P. Delguste, G. Liistro, G. Aubert-Tulkens.

ABSTRACT: A 49 yr old poliomyelitic patient had been under cuirass-type nocturnal negative pressure ventilation for more than 20 yrs. He had a severe restrictive ventilatory impairment, and normal awake blood gases at rest and during light exercise. He was offered a trial of intermittent positive pressure ventilation applied through the nose (nIPPV). Two daytime studies and one night study were carried out under nIPPV, and one night study was performed under negative pressure ventilation. Tidal volume, respiratory frequency (Respirace), blood gases and electromyogram (EMG) of the diaphragm (DEMG, oesophageal electrode) and/or sternocleidomastoid (ScEMG, surface electrodes) were measured. During daytime studies under nIPPV, the DEMG (and/or the ScEMG) did not decrease by more than 25% ($p < 0.005$). However, when the patient was encouraged to relax, the DEMG decreased by 62% ($p < 0.001$). Tidal volume and ventilation significantly increased during daytime nIPPV ($p < 0.025$), whereas blood gases were kept at physiological levels. At night, the ScEMG was present and prominent until sleep onset. Thereafter it disappeared and remained silent, including periods of wakefulness during sleep time, until final awakening in the morning. This was true for both negative pressure ventilation and nIPPV. Snoring was present throughout sleep under negative pressure ventilation but not under nIPPV. We conclude that the behavioural response of the subject may determine the electrical activity of respiratory muscles during assisted ventilation.

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Nocturnal assisted ventilation has been used for years in patients with respiratory failure due to neuromusculo skeletal diseases [1-3]. Negative (tank respirator, cuirass) or positive pressure devices have been used. The latter have the major drawback of requiring a permanent tracheostomy, whereas the former are cumbersome and may induce obstructive sleep apnoea [4]. Recently, several groups have demonstrated the feasibility of nocturnal intermittent positive pressure ventilation applied non-invasively through a nose mask (nIPPV), both on a short- and long-term basis [4-7]. None of these reports have focused on the activity of the respiratory muscles during assisted ventilation. We had the opportunity to study a patient with sequelae of poliomyelitis during both wakefulness and sleep, and recorded the electromyogram of respiratory muscles during spontaneous breathing and during both negative pressure assisted ventilation (NPV) and nIPPV.

Case report

A 49 yr old man had had poliomyelitis in 1952, with long-term sequelae involving the respiratory muscles. He had had repetitive episodes of respiratory infection, and had been on nocturnal assisted ventilation with a cuirass type respirator for more than 20 yrs. He was reluctant to sleep without his cuirass for fear of complications.

Since it was becoming difficult to find spare parts for his old negative pressure pump, the patient was willing to change the ventilatory mode, provided this did not necessitate a tracheostomy. He was admitted to our hospital for training and trial of nIPPV.

On clinical examination he was thin (height 176 cm, weight 48.5 kg), not cyanosed, with a convex scoliosis to the right. The cervical muscles were prominent and active during inspiration (scaleni and

sternocleidomastoids). Heart rate was 76 beats·min⁻¹ and blood pressure 140/70 mmHg (18.7/9.3 kPa). Heart sounds were normal. On radiographic screening the left hemidiaphragm showed a maximal excursion (sniff test) of 1.5 cm, whereas the right was immobile. Blood gases at rest and during a 20 Watt exercise test continued for 4 mins were, respectively: arterial oxygen tension (Pao₂) 80 and 76 mmHg (10.7 and 10.1 kPa); arterial carbon dioxide tension (Paco₂) 41 and 45 mmHg (5.5 and 6.0 kPa); arterial oxygen saturation (Sao₂) 96 and 95%; pH 7.40 and 7.35. Pulmonary function tests showed a vital capacity of 1.15 l (21% of predicted) [8]; forced expiratory volume in one second (FEV₁) 1.01 l; total lung capacity 4.23 l (58% of predicted). Maximal mouth pressures were 75 cmH₂O (7.36 kPa) for both inspiration and expiration.

Methods

Day studies were performed with the patient supine and awake. The patient was ventilated with a PLV-100® portable positive pressure ventilator (Lifecare Inc., Lafayette, CO) connected to a nose mask (Respironics Inc., Monroeville, PA). Tidal volume (V_T), respiratory frequency (f) and inspiratory flow were adjusted on a trial and error basis until the patient felt comfortable. A Respitrace® (Ambulatory Monitoring, Ardsley, NY) pneumograph calibrated using the iso-volume manoeuvre was used to measure tidal volume (V_T), respiratory frequency (f) and minute ventilation (V_E) [9]. Blood gases were analysed with Corning 175 or 178 blood gas analysers (Corning Medical, Medfield, MA). Diaphragmatic electromyogram (DEMG) was obtained from a bipolar oesophageal electrode positioned in the lower third of the oesophagus, where the electrocardiogram (ECG) signal was minimal and the EMG signal maximal. The proximal end of the electrode was taped to the nose after its position was adjusted. The DEMG signal was filtered between 200 and 2000 Hz [10, 11], rectified and integrated via a leaky integrator with a time constant of 0.15 s. Sternocleidomastoid raw EMG (ScEMG) was obtained from surface electrodes positioned 2 cm apart over the main body of the left muscle. Only on the second daytime study was this signal filtered, rectified and integrated. The more superficial platysma muscle may have contributed to the recordings. If so, its activity was similar to that of the sternocleidomastoid: phasic EMG bursts were clearly inspiratory, there was no EMG activity during expiration and no EMG activity was recorded during glossopharyngeal breathing (see below). Pressure in the nose mask was measured with a Validyne transducer.

The patient was studied twice under nIPPV: on the first afternoon of his admission, and on the last day in hospital, after four days and two nights of nIPPV. During the second study DEMG and blood gases were not measured, and the ScEMG was filtered, rectified and integrated. For each condition (see below) 20 to 30 breaths were analysed for V_T, f, V_E and peak integrated EMG (in mm of paper). Comparisons were made with the unpaired t-test, and p<0.05 was considered as significant.

Nocturnal studies were performed by full night polysomnography, using standard methods [12]. In addition to electroencephalogram (EEG), chin EMG, electro-oculogram (EOG), electrocardiogram (ECG), respiratory movements (Nihon Kohden thoracic strain gauge), nasal and oral flow (thermocouples placed in front of mouth and nostrils or via the nasal mask pressure records), respiratory sounds (microphone glued to the skin over the larynx), and Sao₂ (Nellcor N-100 pulse oximeter, Nellcor Inc., Hayward, CA), the raw surface ScEMG was also recorded. The patient was studied twice: the first night in hospital, during NPV with his own cuirass type ventilator; the fourth night during nIPPV with the ventilator setting determined the first day.

Table 1. – Evolution of some physiological variables with intermittent positive pressure ventilation (nIPPV) applied through the nose during the daytime

	Spontaneous breathing	nIPPV	nIPPV2	nIPPV3
V _T ml	173	296*	205	272*
f breaths·min ⁻¹	20	16	17	17
V _E l·min ⁻¹	3.46	4.72*	3.42	4.54*
Peak DEMG mm of paper	18.4	28.9*	8.1*	6.9*
Pao ₂ mmHg	86	82	-	92
kPa	11.5	10.9	-	12.3
Paco ₂ mmHg	38	44	-	39
kPa	5.1	5.9	-	5.2
Sao ₂ %	96	96	-	97

nIPPV: ventilator tidal volume 600 ml, f 16 strokes per min; nIPPV2: same, patient asked to relax; nIPPV3: ventilator tidal volume 700 ml, f 17 strokes per min; V_T: tidal volume; V_E: minute ventilation; Peak DEMG: integrated diaphragmatic EMG; Pao₂: arterial oxygen tension; Paco₂: arterial carbon dioxide tension; Sao₂: arterial oxygen saturation. Significance of the differences with respect to spontaneous breathing: * p<0.001; * p<0.025.

Results

Day studies

First day. After 5 min spontaneous unassisted breathing (during which control measurements were recorded), the ventilator was turned on to apply nIPPV. The V_T, V_E and peak DEMG significantly increased, whereas the Paco₂ only slightly increased (table 1 and fig. 1). After 15 min, the patient was asked to relax. The V_T and V_E decreased to levels no different from those seen during control measurements, whereas the DEMG significantly decreased (table 1 and fig. 2). However, periodic sighs (with increases in peak DEMG and V_T) were apparent (fig. 2). Ventilation was then changed by increasing the tidal volume delivered by the ventilator, whilst the patient was encouraged to remain relaxed. This resulted in significant increases in V_T and V_E, with a slight decrease in Paco₂, and peak DEMG values significantly lower than during spontaneous breathing (table 1 and fig. 2). The DEMG remained at these low values for the rest of the session (90 min). Although

the ScEMG was not integrated, visual inspection of traces showed nearly equal magnitudes and a similar evolution of raw DEMG and raw ScEMG (figs. 1 and 2).

spindles). Breathing was highly regular, at 16 breaths·min⁻¹. Snoring was present throughout sleep, but no apnoeas were noted. At the beginning of the night,

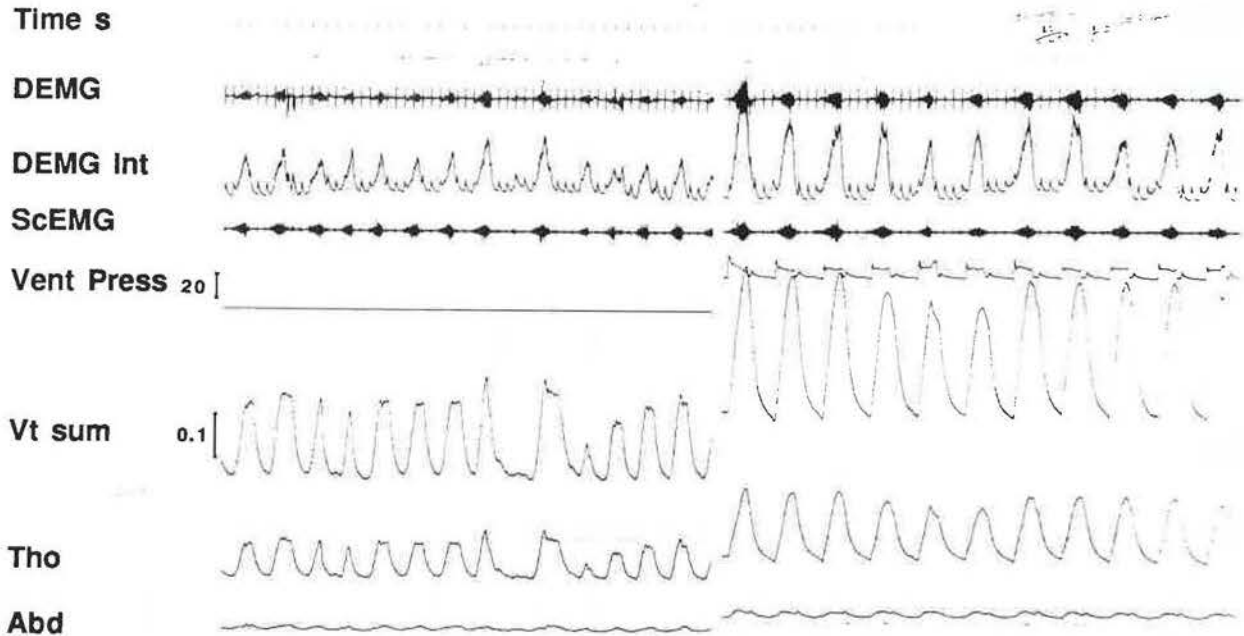


Fig. 1. – Evolution of some physiological variables during spontaneous breathing (left panel) and during intermittent positive pressure ventilation applied through the nose (right panel). DEMG: diaphragmatic electromyogram; DEMG int: DEMG integrated; ScEMG: sternocleidomastoid EMG; Vent Press: pressure in the nose mask, in cmH₂O (1 cmH₂O=0.0981 kPa); Vt sum: tidal volume sum (in l) obtained from the sum signal of a Resptrace; Tho and Abd: thoracic and abdominal signals of the Resptrace. The Vent Press trace was displaced upwards between the left and the right recordings. Note that during nIPPV Vt increases, and so does DEMG. Note also the good correspondence between raw DEMG and raw ScEMG signals.

Fifth day. The patient was studied again on the fifth day. Control (spontaneous breathing) average Vt was 177 ml, f 27 breaths·min⁻¹, \dot{V}_E 4.80 l·min⁻¹ and peak integrated ScEMG 22.8 mm. During the first minutes of nIPPV (with the ventilator setting of the first day, see nIPPV3 on table 1) the Vt increased to 296 ml ($p<0.001$ with respect to control); the f decreased to 16.7 breaths·min⁻¹ and \dot{V}_E was 4.94 l·min⁻¹ (ns). The ScEMG was 21.3 mm, a non-significant change. After two hours of nIPPV the ScEMG was 17.1 mm ($p<0.005$ with respect to control, a 25% decrease). At the end of nIPPV, the patient resumed breathing using glossopharyngeal respiration.

Night studies

Cuirass assisted ventilation. Sleep was scarce, interrupted by multiple arousals. Total sleep time (TST) was 189 min, with more than 50% stage 2 sleep. Deep sleep (stages 3 and 4) accounted for 34% of TST, and rapid eye movement (REM) sleep was absent (although REMs were found at the end of the night, but scored as stage II since EEG was typical of this stage with

when the patient was still awake, the ScEMG was conspicuous, in the form of phasic inspiratory bursts (fig. 3). By contrast, from sleep onset the ScEMG became silent and remained so until the end of the night, including the periods of awakening during sleep time (fig. 3). After final awakening in the morning, the ScEMG phasic inspiratory bursts reappeared. The SaO_2 was stable with a mean value of 98%, never falling by more than 4%.

Nasal intermittent positive pressure ventilation. On this night also, sleep was scarce and interrupted by multiple arousals. The TST was 100 min, with 58% stage 2 sleep. Deep sleep was only 8%, but REM sleep was present (9%). The SaO_2 was stable with a mean value of 98% and no falls greater than 4%. Breathing was regular, both when awake and asleep (fig. 4). The ScEMG was prominent during wakefulness, whereas it disappeared from sleep onset (fig. 4) and remained absent for the rest of the night, including during periods of awakening during sleep time. After final awakening, in the morning, the ScEMG phasic inspiratory bursts reappeared. No snoring or apnoeas were noted.

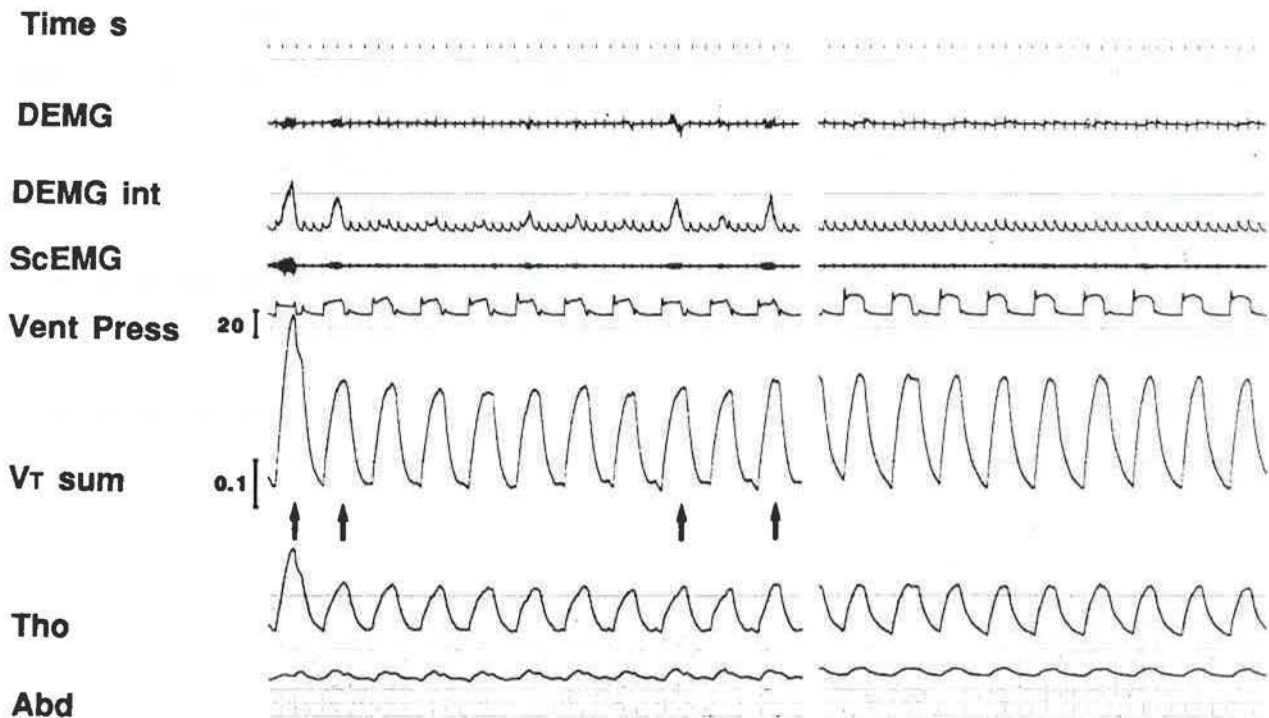


Fig. 2. — When the patient was asked to relax (left panel), the EMG signals (and the V_T) decreased. However, periodic "sighs" (*i.e.* increases in EMG and V_T) were noted (arrows). When the ventilation delivered by the respirator was increased and the patient was encouraged to remain relaxed (right panel), the EMG signals practically disappeared. Note, once again, the good correspondence between the raw DEMG and ScEMG signals. Abbreviations and symbols as in figure 1.

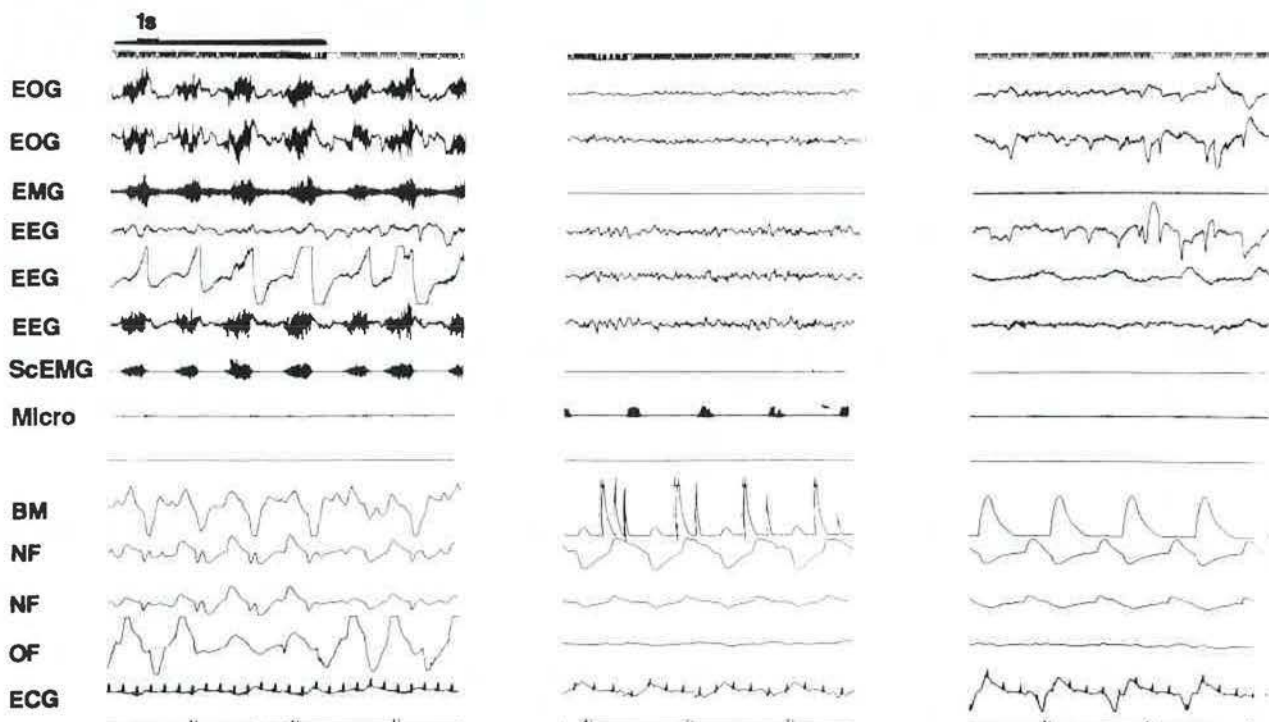


Fig. 3.—Polysomnographic record whilst the patient is being ventilated with a cuirass type negative pressure ventilator. EOG: electrooculogram; EMG: submental electromyogram; EEG: electroencephalogram; ScEMG: sternocleidomastoid EMG; Micro: breathing sounds recorded by a microphone glued to the skin over the larynx; BM: breathing movements; NF: nasal flow; OF: oral flow; ECG: electrocardiogram. When the patient is awake, at the start of the night (left panel), the ScEMG activity shows prominent phasic inspiratory bursts. Note the regularity of the breathing movements, which are entrained by the cuirass type ventilator. When the patient is asleep (middle panel), stage 2, the ScEMG activity disappears. Note the presence of snoring at each inspiration on the Micro tracing. When the patient awakens in the middle of the night (right panel), snoring disappears; note that the ScEMG remains silent.

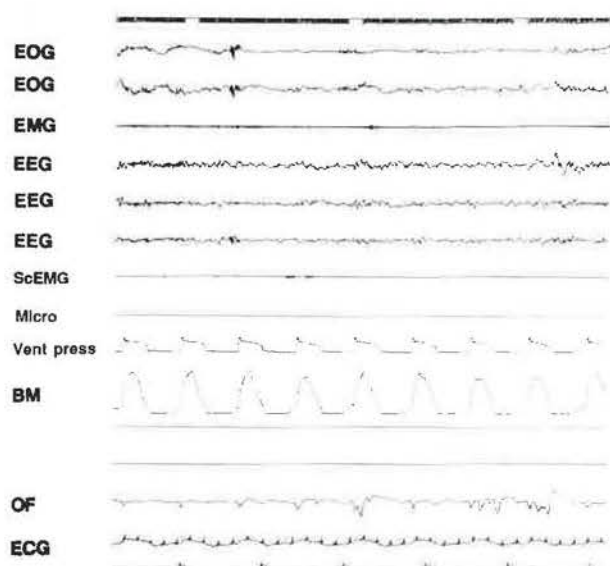


Fig. 4. — Sleep recording (stage 2) whilst the patient is receiving intermittent positive pressure ventilation through the nose (nIPPV). Abbreviations as in figure 5, except Vent Press: pressure in the nose mask. Note the absence of snoring and of ScEMG activity.

Discussion

In this patient, positive pressure ventilation administered through a nose mask has proven feasible and efficient. He could tolerate several hours daytime ventilation from the start of the trials, without local discomfort. Blood gases were kept at physiological levels with peak inspiratory pressures of 15–20 cmH₂O, avoiding mouth leaks. Our results are thus similar to those of several recent papers reporting on this new non-invasive technique of assisted ventilation [4–7]. *ELLIS et al.* compared nIPPV to cuirass- or tank-delivered NPV. They found under NPV but not under nIPPV that during REM sleep all their patients developed upper airway obstructive apnoea [4]. Our patient did not enter REM sleep during the night under NPV, and we did not observe apnoeas. However, snoring, thought to represent partial upper airway obstruction, was conspicuous. Since there was no "active" respiration (inasmuch as the ScEMG is representative of the respiratory muscles), snoring could be attributed to sleep induced neural inhibition of upper airway muscles. Lack of snoring during nIPPV was most probably due to the positive pressure breathing, known to abolish snoring in both snorers and sleep apnoea patients [13, 14].

The main interest of this report rests on the response of the respiratory muscles to assisted ventilation during day and night. Studies of respiratory muscular activity usually centre on the diaphragm, the main respiratory muscle. We recorded the diaphragmatic EMG only during the first trial, whereas for the rest of the trials we studied the sternocleidomastoid EMG. This

seems to be justified both on clinical and electromyographical grounds. Clinically, although this patient did not have diaphragmatic paralysis, (there was neither paradoxical breathing nor positive sniff test) he had severe diaphragmatic weakness so that inspiratory contractions of accessory muscles were prominent and continuous throughout the 5 days he stayed in hospital. Furthermore electromyographically, the ScEMG and the DEMG raw signals were similar during the first day study (figs 1 and 2), and we considered it justified to rely on the ScEMG as an index of global inspiratory muscular activity, avoiding the further use of an oesophageal electrode.

At the start of the first daytime trial under nIPPV, the patient continued to use his inspiratory muscles, and the EMG signals increased significantly compared to unassisted breathing (perhaps explaining, in addition to the small deadspace of the nose mask, the rise in P_{aCO_2}). The patient rapidly adopted the frequency of the respirator (1:1 phase lock). Only when the ventilator setting was readjusted and the patient was asked to relax did the DEMG and ScEMG decrease (fig. 2). On the fifth day, after 5 days habituation to nIPPV, his ScEMG amplitude decreased by at most 25% throughout the 150 min of the trial. Since the setting of the ventilator was the same as during the first day, we attribute the difference between the first (sharp decreases in EMG) and fifth (persistence of EMG) days to the lack of relaxation on the latter occasion.

Polysomnographic studies showed scarce and unstable sleep. This could be the result of the "first night effect" in an anxious patient, without prior experience with sleep recordings. Before sleep onset the ScEMG signals showed prominent contractions with a 1:1 phase locking (with both NPV and nIPPV). By contrast, sleep resulted in a silent EMG, with both cuirass and nIPPV. Once the ScEMG became silent during the first sleep period, it remained silent for the rest of the night, even during awakenings. Hence, during wakefulness, the respiratory muscles of this patient could either remain active, as in the second day study and on both night studies before sleep onset (fig. 3), or stop contracting, as in the first day study, patient relaxed and on both night studies after sleep onset (figs 2–4). We had previously found, in both healthy subjects and patients with chronic airflow obstruction, without previous experience with assisted ventilation, persistence of diaphragmatic EMG during NPV [11, 15]. We had, therefore, suggested that habituation to the procedure was a necessary condition for muscle rest to be obtained during assisted ventilation [11, 15]. In this patient, despite a very long practice with assisted ventilation, decrease in peak EMG activity of respiratory muscles was not achieved during daytime assisted ventilation unless he voluntarily relaxed; at night ScEMG even disappeared during wakefulness. This suggests that the concept of "habituation" as previously presented by us may be an over-simplification; apparently it is the behavioural response which will determine whether the subject will or will not use his own muscles during assisted ventilation.

The effectiveness of assisted ventilation in preventing cardiorespiratory failure in severe restrictive ventilatory impairment is beyond doubt [1-3]. It has recently been postulated that assisted ventilation could act through muscle rest in these patients with chronically overloaded respiratory muscles (*i.e.* chronic muscle fatigue) [16, 17]. Indeed, ROCHESTER *et al.* observed, in obstructive and restrictive patients under NPV, almost complete disappearance of diaphragmatic EMG, which they attributed to the relief by the respirator of the load chronically imposed upon the respiratory muscles [16]. Present and previous results from this laboratory [11, 15] suggest that arrest of contraction of the respiratory muscles under assisted ventilation may simply represent a behavioural response.

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Adaptation à l'assistance ventilatoire par pression positive intermittente par voie nasale pendant tout le nyctémère. D.O. Rodenstein, D.C. Stanesco, P. Delguste, G. Liistro, G. Aubert-Tulkens.

RÉSUMÉ: La réponse de la ventilation et de l'activité des muscles respiratoires à l'assistance ventilatoire par pression positive intermittente par voie nasale (nIPPV) a été étudiée chez un patient de 49 ans, porteur de séquelles de poliomyélite et placé depuis 20 ans sous ventilation assistée nocturne par pression négative périoracique (cuirasse, NPV). Le patient présentait un déficit ventilatoire restrictif sévère, avec des gaz du sang normaux au repos et après un effort modeste. Trois enregistrements ont été réalisés sous nIPPV, deux pendant la journée, patient éveillé, un pendant la nuit, et un quatrième enregistrement a été réalisé la nuit sous NPV. On a mesuré la ventilation minute (V_E), la fréquence respiratoire (f), l'EMG du diaphragme (DEMG) et/ou du sternocleidomastoïde (ScEMG), ainsi que les gaz du sang. Pendant la journée, sous nIPPV, le DEMG et/ou le ScEMG ont diminué de moins de 25% ($p < 0.005$) par rapport à la ventilation spontanée. Cependant, lorsque le patient a été prié de se relâcher, le DEMG a diminué de 62% ($p < 0.001$). La V_E augmentait de façon significative sous nIPPV, alors que les gaz sanguins restaient normaux. Pendant la nuit, sous nIPPV ou NPV le ScEMG est resté actif jusqu'à l'endormissement. Pendant le sommeil, et même pendant les périodes de veille intra-sommeil, le signal EMG a disparu pour réapparaître avec le réveil matinal. Sous NPV, mais non sous nIPPV, un ronflement a été enregistré. Nous postulons que l'activité électrique des muscles respiratoires sous ventilation assistée dépend de la réponse comportementale du sujet.

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