Teaching Pearls in Noninvasive Mechanical Ventilation

Key Practical Insights

Antonio M. Esquinas
Editor

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To my family…, great motivation in my live

Preface

In the last decades, we have developed a broad knowledge base in noninvasive mechanical ventilation that supports the daily use of this technique in different settings and medical specialties. We have been able to establish solid knowledge and apply practical protocols as well as technological advances. From a critical perspective, one of these essential elements that have allowed us to ensure a correct application is supported by excellence in training and education plans in noninvasive mechanical ventilation carried out with the support of clinical teachers. In this original and first book, **Teaching Pearls in Noninvasive Mechanical Ventilation**, we offer the first original book whose bases is a teaching based on the critical analysis of selected clinical cases that represent the most common and real situations of use of noninvasive mechanical ventilation.

The structure of the book and chapters is based on the teaching that provides the critical analysis of the most common clinical cases present on a daily basis of clinical practice. This original book structure makes it ideal to be a reference book in training and education plans of pulmonary as well as critical care and sleep medicine fellowship programs, universities, and postgraduate courses in noninvasive mechanical ventilation. This book comes from the solid idea that teaching based on the "critical analysis" of the "clinical case" is the first and basic element to ensure the best transmission of knowledge and correct application. Besides, this book is conceived as a key tool for proper teaching for professors or teachers in the field of **Noninvasive Mechanical Ventilation**.

If you want to learn, teach (Marcus Tullius Ciceron)

Murcia, Spain
March 2021

Antonio M. Esquinas

Contents

Part I Clinical Cases in Noninvasive Ventilation: Interfaces, Methodology

Abbreviations

AASM	American Academy of Sleep Medicine
ABG	Arterial blood gas analysis
ACBT	Active cycle of breathing technique
AChR-Ab	Autoantibodies against the acetylcholine receptor
ACPE	Acute cardiac pulmonary edema
ADH	Anti-diuretic hormone
AECOPD	Acute exacerbation of chronic obstructive pulmonary disease
AE-IPF	Acute exacerbation of IPF
AF	Atrial Fibrillation
AFC	Alveolar fluid clearance
AG	Anion gap
AHI	Apnea hypopnea index
AHRF	Acute hypoxemic respiratory failure
AI	Asynchrony index
AI%	Asynchrony index
AIH	Apnea hypopnea index
AKI	Acute kidney injury
ALI	Acute lung injury
ALS	Amyotrophic lateral sclerosis
ALT	Alanine aminotransferase
APACHE II	Acute Physiology and Chronic Health Evaluation II
APCV	Assist pressure control ventilation
APE	Acute pulmonary edema
ARDS	Acute respiratory distress syndrome
ARF	Acute respiratory failure
ASA	American Society of Anesthesiologists
AST	Aspartate aminotransferase
ASV	Adaptive servoventilation
ATS	American Thoracic Society
AUC	Area under the curve
AVAPS	Average volume-assured pressure support

Bf	Bronchofiberoscopy
BGA	Blood gas analysis
BiPAP	Bilevel positive airway pressure
BiPAP-S	BiPAP spontaneous mode
BMI	Body mass index
BNP	Brain natriuretic peptide
BP	Blood pressure
bpm	Beats per minute
C	Compliance
CAP	Community acquired pneumonia
CCHS	Congenital central alveolar hypoventilation syndrome
CF	Cystic fibrosis
CHRF	Chronic hypercapnic respiratory failure
CI	Confidence interval
CKD	Chronic kidney disease
CLD	Chronic liver disease
cm H_2O	Centimeter of water
CMO	Comfort measures only
CMT	Charcot–Marie–Tooth disease
CO_2	Carbon dioxide
COPD	Chronic obstructive pulmonary disease
CPAP	Continuous positive airway pressure
CPE	Cardiogenic pulmonary edema
CPH	Chronic pulmonary hypertension
CPT	Chest physiotherapy
CRF	Chronic respiratory failure
CRP	C-reactive protein
CRX	Chest radiograph
CSA	Central sleep apnea
CT	Computed tomography
CTPA	Computed tomographic pulmonary angiography
CURB-65	Confusion, urea, respiratory rate, blood pressure – 65 years of age
CVP	Central venous pressure
CWP	Centimeters of water pressure
CXR	Chest X-Ray
DAD	Diffuse alveolar damage
DE	Diaphragmatic excursion
DEX	Dexmedetomidine
DLCO	Carbon monoxide diffusion capacity
DLT	Double lumen tube
DMD	Duchenne muscular dystrophy
DNI	Do-not-intubate
DNR	Do not resuscitate
DOT	Domiciliary oxygen therapy
DP	Driving pressure

DRG	Dorsal respiratory group
DT	Diaphragm thickness
e	Elastance
EAdi	Electrical activity of diaphragm
EADi/Edi	Electrical activity of the diaphragm
ECCO2R	Extracorporeal CO_2 removal
ECG	Electrocardiogram
ECMO	Extracorporeal membrane oxygenation
ED	Emergency Department
EEN	Effective enteral nutrition
EF	Ejection fraction
EPAP	Expiratory positive airway pressure
ERS	European Respiratory Society
ES	Excessive sleepiness
OSAS	Obstructive Sleep Apnea Syndrome
IAH	Index of apnea-hypoapnea
PSG	Polysomnography
RSD	Respiratory sleep disorder
ESICM	European Society of Intensive Care Medicine
ESS	Epworth sleepiness scale
EVLW	Extra-vascular lung water
Flow	Gas flow
FBS	Fiberoptic bronchoscopy
FEF	Forced expiratory flow
FEV1	Forced expiratory volume in the first second
FEV1/FVC ratio	Forced expiratory volume in the first second/forced vital capacity
FiO_2	Fraction of inspired oxygen
FM	Face-mask
FMV	Face-mask ventilation
FOB	Fiberoptic bronchoscopy
FRC	Functional residual capacity
FVC	Forced vital capacity
GCS	Glasgow Coma Scale
GOLD	Global Initiative for Chronic Obstructive Lung Disease
GORD	Gastro-oesophageal reflux disease
GPB	Glossopharyngeal breathing
HACOR	Heart rate, Acidosis, Consciousness, Oxygenation, and Respiratory rate
HAP	Hospital-acquired pneumonia
Hb	Hemoglobin
HCO_3^-	Bicarbonate
HDU	High Dependency Unit
HES	Hypercapnic encephalopathy syndrome
HFNC	High flow nasal cannula

HINPPV	High-intensity noninvasive positive pressure ventilation
HR	Heart rate
HRCT	High-Resolution Computed Tomography
HRQoL	Health-Related Quality of Life
Htc	Hematocrit
IAP	Intra-abdominal pressure
IBP	Invasive blood pressure
IBW	Ideal body weight
IC	Inspiratory capacity
ICP	Intracranial pressure
ICS	Inhaled corticosteroid
ICSD-3	International Classification of Sleep Disorders, Third Edition
ICU	Intensive Care Unit
I:E Ratio	Ratio of inspiratory and expiratory time
IGHMBP2	Immunoglobulin helicase μ-binding protein 2
ILD	Interstitial lung disease
IMV	Invasive mechanical ventilation
IPAP	Inspiratory positive airway pressure
IPF	Idiopathic pulmonary fibrosis
IPPB	Intermittent positive pressure breathing
ISS	Injury Severity Score
IT	Inspiratory time
IV	Intravenous
IVS	Interventricular septum
KMS	Kelly-Matthay Scale
L/min	Liter per minute
LA	Left atrial
LABA	Long-acting β_2-agonist
LAMA	Long-acting muscarinic antagonist
LMWH	Low molecular weight heparin
LoS	Length of stay
LRTI	Lower respiratory tract infection
LTMV	Long-term mechanical ventilation
LTOT	Long-term oxygen therapy
LUS	Lung ultrasound
LUS-ReS	Lung Ultrasound Reaeration Score
LV	Left ventricle
LVR	Lung volume recruitment
MAC	Manually assisted cough
MDT	Multidisciplinary team
MEP	Maximal expiratory pressure
mg/h	Milligram per hour
MI	Mechanical insufflations
MIC	Maximum insufflation capacity
MI-E	Mechanical Insufflation-Exsufflation

MIP	Maximal inspiratory pressure
mL/kg	Milliliters to kilograms
mm/Hg	*Millimeters* of mercury
mmol/L	Millimoles per liter
MODS	Multiple organ dysfunction syndrome
MOF	Multiple organ failure
MRF	Maugeri Respiratory Failure questionnaire
MV	Mechanical ventilation
NAVA	Neurally adjusted ventilatory assist
NC	Nasal cannula
NEX	Distance of nose tip earlobe and processus xyphoideus
NIMV	Noninvasive mechanical ventilation
NIOV	Noninvasive open ventilation
NIPSV	Noninvasive pressure support ventilation
NIV	Noninvasive ventilation
NIV-BF	Noninvasive positive pressure facilitated bronchofiberoscopy
NMBAs	Neuromuscular blocking
NMD	Neuromuscular disease
NPPV	Noninvasive positive pressure ventilation
NPV	Negative pressure ventilation
NREM	Non-rapid eye movement
NT-proBNP	N-terminal pro-brain natriuretic peptide
NVS	Noninvasive ventilatory support
O_2	Oxygen
O_2-LT	Oxygen long-term therapy
O_2T	Oxygen therapy
OCS	Oral corticosteroids
OD	Oxygen desaturation
ODI	Oxygen desaturation index
OHS	Obesity-hypoventilation syndrome
OR	Odds ratio
OSA	Obstructive sleep apnea
P/F-PaO_2/FiO_2	Ratio of PaO_2 to fraction of inspired oxygen
P/F ratio	Partial pressure of arterial oxygen/fraction of inspired oxygen ratio
P/I Index	Ratio of EADi peak value and EADi inspiratory AUC
$PaCO_2$	Partial pressure of arterial carbon dioxide
$PaCO_2$	Arterial carbon dioxide tension
Pal	Alveolar pressure
PaO_2	Arterial oxygen partial pressure
PAV	Proportional assist ventilation
Paw	Airway pressure
PAWP	Pulmonary arterial wedge pressure
PBW	Predicted body weight
PC-BIPAP	Control pressure – Bilevel positive airway pressure

PCA	Patient control analgesia
PC-BiPAP	Pressure control bilevel positive airway pressure
PCF	Peak cough flow
pCO_2	Carbon dioxide tension
PCV	Pressure control ventilation
PEEP	Positive end-expiratory pressure
PEEPi	Intrinsic positive end-expiratory pressure
PEF	Peak expiratory flow
PE	Pulmonary embolism
$PetCO_2$	End-tidal CO_2
PFTs	Pulmonary Function Tests
pH	Potential of hydrogen
PH	Pulmonary hypertension
pNIV	Portable noninvasive ventilation
pO_2	Peripheral oxygen saturation
PPC	Postoperative pulmonary complications
PPE	Personal protective equipment
Ppl	Pleural pressure
PPV	Positive pressure ventilation
PR	Pulmonary rehabilitation
PVR	Pulmonary vascular resistance
PS	Pressure support
PSG	Polysomnography
P-SILI	Patient self-induced lung injury
PSV	Pressure support ventilation
$PtcCO_2$	Transcutaneous carbon dioxide
pts	Patient/s
PVA	Patient–ventilator Asynchrony
PVD	Patient ventilator dyssynchrony
PY	Pack year
Raw	Airway resistance
RCT	Randomized clinical trial
REM	Rapid eye movement
RF	Respiratory failure
RHDCU	Respiratory high-dependency care unit
RICU	Respiratory Intensive Care Unit
ROM	Range of motion
RR	Respiratory rate
RRT	Renal replacement therapy
RV	Residual volume
RVent	Right ventricle
RVSP	Right ventricular systolic pressure
S/T Mode	Spontaneous/Timed Mode
SaO_2	Oxygen saturation
SAPS II	Simplified Acute Physiology Score II

SAPS	Simplified Acute Physiology Score
SAPS3-CNIV	Simplified Acute Physiology Score 3-Customized NIV
$SatO_2$	Arterial oxygen saturation
SB	Spontaneous breathing
SD	Swallowing disorders
SDB	Sleep disordered breathing
SGRQ	St George's Respiratory Questionnaire
SID	Strong ion difference
SMA	Spinal muscular atrophy
SMARD1	Spinal muscular atrophy with respiratory distress type 1
SOFA Score	Sequential Organ Failure Assessment Score
SPN-CPAP/PS	Spontaneous - Continuous positive airway pressure or Pressure support ventilation
SpO_2	Peripheral oxygen saturation
SRBDs	Sleep-related breathing disorders
SrH	Sleep-related hypoventilation
SrHDs	Sleep-related hypoventilation disorders
SRI	Severe Respiratory Insufficiency questionnaire
StO_2	O_2 saturation
SVA	Subject ventilator asynchrony
T90	Time spent SpO_2 <90%
TAPSE	Tricuspid annular plane systolic excursion
TB	Tuberculosis
TBI	Traumatic brain injury
$TcCO_2$	Transcutaneous carbon dioxide
TEE	Transesophageal echocardiography
Ti	Inspiratory time
TI	Thickening fraction
TLC	Total lung capacity
TRV	Tricuspid regurgitation velocity
TTE	Transthoracic echocardiography
TV	Tidal volume
Tv	Tricuspid valve
UIP	Usual interstitial pneumonia
Va/Q	Ratio of ventilation to perfusion
VAP	Ventilator-associated pneumonia
VAPS	Volume-assured pressure support
VATS	Video-assisted thoracoscopic surgery
VC	Vital capacity
VCI	Vena cava inferior
VCV	Volume-controlled ventilation
VDd	Dead space of the mask
VDdyn	Dynamic dead space
VDph	Physiologic dead space
VIDD	Ventilator-induced diaphragmatic dysfunction

VILI	Ventilator-induced lung injury
VPF	Ventilatory pump failure
VPW	Vascular pedicle width
VRG	Ventral respiratory groups
VS	Versus
Vt	Tidal volume
WBC	White blood cell count
WOB	Work of breathing
WSS	Woodhouse-Sakati syndrome
ΔP	Pressure change
ΔV	Volume change
μL	Microliter
% pred	Percent of predicted value

List of Videos

is important to note that without increasing the pressure in the pressure—time scalar, there is movement of flow in the flow—time scalar. This mode can be used in patients with correct respiratory drive

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Part I
Clinical Cases in Noninvasive Ventilation: Interfaces, Methodology

Chapter 1
Facemask and Total Face Mask

Edoardo Piervincenzi, Giorgio Zampini, and Daniela Perrotta

1.1 Introduction

Avoiding endotracheal intubation in adult and pediatric population has undiscussed advantage.

Nowadays there is a great development by the companies of new masks and physicians have a wide selection of interfaces available.

At the same time, there are few recommendation about which interface are better than other in each clinical situation, and data about tolerance and efficacy are lacking especially for paediatric patients.

Every ICU anyway, should have several types of mask/interface to provide a tailored therapy on each patient to provide the best possible comfort and efficacy during NIV therapy administration.

Facemask (oro-nasal mask) and full-face mask are both valid instruments to provide non-invasive positive pressure ventilation therapy.

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Also the helmet has shown over the years to be a valid alternative to supply NIV with some precautions in the setting of the mechanical ventilator.

In this chapter, the main aspects of the NIV delivered through masks will be analyzed, NIV will be intended as a ventilation with an inspiratory and an expiratory pressure level both generated by the mechanical ventilator. CPAP therapy or HFNC will not be treated as topics in this paper [1–6].

1.2 NIV-Mask

1.2.1 Caratheristics

A first differentiation must be considered between vented and non-vented mask, this will lead to the use of different circuits, different ventilators with intrinsic characteristics; all aspects that have repercussion on NIV supply (see Fig. 1.1).

In ICU/PICU usually are preferred non-vented mask with double tube circuits due to large diffusion of ICU-ventilators instead of Home Care Ventilator.

But in some patient especially in chronic ones, Home Care-Ventilators are more tolerated and offer a lot of interesting options with particular ventilation modalities.

It is superfluous to point out that unvented interfaces cannot absolutely be used on single-limb circuits because it would cause enormous damage due to an impossible expiration.

Likewise, the vented interfaces cannot be used on double-limbs circuits due to the enormous pressure losses that would result.

The non-vented Oro-nasal or full-face masks with ICU ventilator is more effective in dyspnoeic patients due to a lower amount of risk of rebreathing CO_2 (see Figs. 1.2 and 1.3).

Fig. 1.1 Different connector for vented single limb circuit (**a**) and for non-vented double limb circuit (**b**)

Fig. 1.2 Example of nasal mask

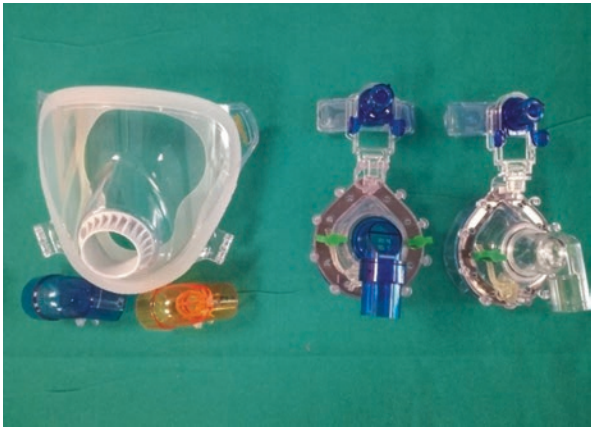

Fig. 1.3 Example of oronasal: fullface masks

The nasal masks or nasal pillows can be used only in cooperative non-dyspnoeic adult patient or in small infants that have still nasal breathing, the patient usually better accepts these masks than the other interfaces, but the power in gas exchange improvement is low.

In collaborating non-dyspnoeic adult patient however, any interface that is comfortable for the patient can be reasonably used only if, however, we know its characteristics and strategies to get better its effectiveness.

The success or not of NIV therapy depend on the capability to reduce amount of WOB and to increase alveolar minute ventilation.

Leaks and patient-ventilator asynchrony are the two main negative determinants responsible for the failure of the NIV.

An interesting data from literature is that seems to have more weight on the outcome the choice of a correct interface rather than the ventilation mode.

In addition, the poor tolerance, for claustrophobia, for an uncomfortable interface that develops too much pressure on the skin inevitably leads to an excessive discomfort and therefore to the interruption of the treatment.

Patient comfort play a key-role in ensuring continuous therapy without interruption thus allowing effective alveolar recruitment and adequate rest of the respiratory muscles.

Therefore, is crucial to choose masks that have adequate fixing systems capable of distributing the pressures and possessing well adherent but at the same time soft cushion (see Fig. 1.4).

From a physiological point of view, any type of mask increase the dead space but peep level correctly set could gain a better CO_2 washout and cut down the risk of rebreathing.

Saatci et al. have investigated the properties in terms of dead space of several facemask.

The different design of each mask could influence not only the absolute but also the dynamic dead space [7]. R. Fodil et al. tested several numbers of interfaces comparing them in term of dead space and his clinical impact.

The different interfaces available have shown to be all, each with its own small differences and peculiarities, a good way to delivering NIV therapy. The airway pressure, neuromuscular drive, inspiratory muscle effort, work of breathing (WOB), arterial blood gases does not shown great difference [8].

With the appropriate setting of the mechanical ventilator, also the differences in terms of WOB can be effectively overcome.

This paper, with an elegant fluid-dynamics study, show how the effective dead space called interface dead space it's not equivalent to real interface volume.

This means that, in this bench study, with a normal adult tidal volume the bigger interface does not entail a significant exhaled gas rebreathing.

The authors explain this phenomenon by demonstrating that when the interface has a large internal volume, as in the case of the helmet, each single breath influences the variation of internal gases by a small percentage and convective flux inside

Fig. 1.4 Different type of headgears

has a relative role. When the interface has smaller internal volume (much more near to Vt), a predominant role in the variation of gas composition is assumed by the convective flow that develops inside and the real death space is almost the same to internal volume (see Table 1.1).

If this is true in adult, with a tidal volume in the order of hundreds of milliliters, it may not be equally true in the child although there is no currently experimental strong data to verify it.

In this paper the authors suggest that, for extremely low VT, maybe the better interface to reduce CO_2 and to prevent gas rebreathing are Helmet. Unfortunately, the Helmet, with a low tidal volume ventilation has enormous problems in term of sensivity of inspiratory trigger and in synchronization.

Davide Signori et al. have shown, in another paper, how the use of non-vented double-tube interfaces significantly reduce CO_2 rebreathing during NIV and the presence of a flow-by amplified this effect [10].

In a bench-study by Conti el al. has been studied in details the synchronization and the interaction between patient and ventilator with different paediatric interfaces during PSV in a mixed obstructive restrictive model.

PSV ventilation present several problems of asynchrony/interaction especially for high respiratory frequencies.

From this study emerge that Helmet has the worst patient interaction, especially during high respiratory frequency (>30 rr) even in a normal lung model in PSV.

All the results available to date seem to indicate the use of a mask as a preferential delivery strategy in the NIV, in the pediatric population, albeit with the theoretical limits linked to rebreathing as explained above [9].

Finally, as emerged from the PEMVECC, to date, there are no strong date to recommend method or timing for NIV in paediatric population.

Despite the bench studies, there are not enough RCTs to define if an interface is better than another one.

As recommended in adult, it should be used the more fitting interface with less percentage of leaks monitoring patient-ventilator synchrony to improve efficacy and comfort [11, 12].

Table 1.1 Characteristics of principal interface to erogate NIV

	Nasal mask	Oro-nasal mask	Full face mask	Helmet
Statical dead space	–	–/+	+	+++
Claustrophobia	–	–/+	+	++
Secrection clearance	++	+	+	–/+
Aspiration risk	–	+	+	+
Flow resistance	++	–/+ (depends on prevalence of nasal breathing or not)	–/+ (depends on prevalence of nasal breathing or not)	–
Patient-ventilator interaction	–/+	++	++	–

In adult such as in paediatric population, success of NIV is even closely related to underlying disease.

In fact, is particularly effective in cardiogenic oedema or in acute exacerbation of chronic respiratory failure, it is much less effective in respiratory failure secondary to oncologic disease or to ARDS.

NIV is also used efficiently as a ventilatory therapy for respiratory distress occurring after extubation both in adults and in children.

The keystone of a successful treatment is a well adaptation of the patient with the NIV: few asynchronies, good comfort and small leaks associated to a good reduction in WOB and in an improvement in gas exchange.

If all this is not achieved in a rather short time the NIV is in the adult that in the child will have high failure rates that will inevitably lead to intubation.

1.3 Bronchopulmonary Displasia

Northway et al. descripted BPD in 1967 for the first time as a diffuse lung disease in a premature lung.

Low volumes and a progression to chronic disease heterogeneous with infiltrates and hyperinflation areas with sponge-like or cystic lesion inside characterized the disease.

BPD now is considered a chronic lung disease typically of premature child with an abnormal distribution of small pulmonary vessels with a hyper-reactive arteriolar tone. These anatomo-pathological modifications lead to pulmonary hypertension and right ventricular hypertrophy.

BPD is still the most frequent disease for premature infant born before the 30th gestational week and persist as chronic respiratory disease in childhood.

To date, there are lacking data from literature about consequences and late outcomes of this disease in childhood, however we know how these children are much more susceptible to respiratory infective events and how they often need MV to overcome these exacerbation.

Going on with age, the disease tends to develop fewer exacerbations episodes even if, from the latest data in the literature, it seems to be responsible for permanent pathological changes in the lungs like pulmonary hypertension, asthma-like symptoms and a permanent compromised lung function [13–15].

Clinical Case

BPD patient 2 years and 4 months old.

Chronic therapy since 4 months ago with Sildenafil, PEG on 2018.

No requirement of O_2 home therapy.

N1H1 pulmonary infection on January 2019.

At presentation on ED in March 2019, there was bilateral crackles, a SpO_2 of 90% and on chest x-ray hyper-insufflation, atelectasis zones and air broncho-gram sign.

Viral PCR on tracheal aspirate positive for coinfection from Adenovirus and Metapneumovirus.

Was diagnosed a superior right lobar and left medium-basal pneumonia.

EGA on room air: pH 7,29, PaO_2 41, $PaCO_2$ 52, BE −4, Lac 1.2.

First line therapy was high flow nasal oxygen with FiO_2 40% at 2 L/kg/min, aerosol therapy with beta2 agonist and ipratropium. In addition, was started a broad-spectrum antibiotic therapy with Amoxicillin/clavulanate plus Claritromicin ev.

EGA after 2 h of oxygen therapy: pH 7,28, PaO_2 65, $PaCO_2$ 54, BE −4.2, Lac 1.0.

Despite therapy after 24 h, there was no improvement of EGA and respiratory mechanics; moreover, indexes of phlogosis and neutrophilia raised up so it was decided to admit the patient in PICU.

Was performed an Echocardiography that shown increased right side pressure so we started a full face NIV, diuretics therapy and Sildenafil ev. beyond the therapies already in progress.

NIV treatment was started with an oro-nasal face alternating it with a full-face mask every 8 h to prevent pressure sore in PRVC controlled ventilation with peep level of 7 cmH_2O, a FiO_2 of 40% and a target volume of 7 mL/kg with a Servo-u ventilator.

EGA after 6 h of NIV: pH 7.36, PaO_2 95, $PaCO_2$ 41, Be −1.3, Lac 0.9.

Two days after we received the result from microbiological tracheal colture positive for E. Coli and antibiotic therapy was changed with Meropem instead of Amoxicilline/clav.

After 4 days, in anticipation of a shift of the patient to a sub-intensive respiratory therapy unit, the ventilator strategy was modified in an S/T ventilation (ipap 16 epap 6 with a FiO_2 of 40% 35 rr) with an home care ventilator TRILOGY 200®.

The patient has been ventilated with a vented oronasal interface cycling NIV with HFNC allowed a progressive re-autonomization of spontaneous respiratory activity.

During the whole period in PICU, active respiratory physiotherapy was performed to increase airway secretions clearance prevent respiratory muscle atrophy and improve recovery.

Another 3 days of non-invasive mechanical ventilation in PICU allowed an improvement of the exchanges such as to be able to discharge the patient in the respiratory sub-intensive ward with a successful ventilation weaning after another 2 days of HFNC therapy and 2 on low flux oxygen nasal cannula.

Patient was successfully discharged from Hospital with a SpO_2 of 93% and a perfectly compensated pH without needing of oxygen.

Key Teaching Points
Strategy in NIMV with mask.

- The choice of right size interface for each patient is the first step to guarantee better tolerance and an optimal ventilator assistance.
  An incorrect mask size can lead to huge leaks hard to manage (compensate) even for ventilators with NIV algorithm.
  Moreover too big or too small mask, displacing easily, cause discomfort for the patient and less tolerability.
  Even the knows of ventilator available to provide NIV in ICU is fundamental to a successful treatment, each one has own strengths and weaknesses characteristic in NIV therapy.
- Do not try to make a mask fit by tightening the headgear, this will only lead to a lower tolerance of the mask
- Even the best mask after some hours creates discomfort due to the constant pressure applied on the facial tissues up to the formation of real pressure sore especially with high peep level.
  To ensure a long-term NIV tolerance it is important a constant daily rotation between at last two different interface available having different shape and then different pressure surface.
- In chronic pulmonary disease (in children as well as in adults), the assisted ventilation modalities are to prefer. These patient has already less respiratory muscle reserve because they are already chronically fatigued and even few days of controlled ventilation could compromise muscle function make weaning process much more difficult if not impossible.
- Physical rehabilitation and respiratory physiotherapy have a crucial role to help in airway secretion clearance and to maintain adequate physical conditioning to start respiratory weaning when clinical condition improve.
- A gradual passage from much more assisted to less assisted NIV and then from NIV to CPAP or HFNC is mandatory to successfully weaning process, especially in chronic disease. This variegate subset of illness has in common the difficulty of restoring the initial condition, tending to worsen after every single episode of exacerbation.

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Chapter 2
Helmet

Giorgio Zampini, Edoardo Piervincenzi, and Daniela Perrotta

2.1 Introduction

The management of respiratory distress in adults and in children is challenging for intensivists and pediatricians; proper treatment is crucial to avoid death and long-term disabilities. When respiratory distress is confirmed, its treatment requires correction and improvement of gas exchange, followed by the diagnosis of the underlying causes and complications [1].

Several studies have showed that Helmet CPAP has a high efficiency in resolving respiratory distress. This was mostly due to the effect of CPAP on alveolar extension, which causes an increase of the alveolar surface responsible for blood gas exchange.

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2.2 Helmet Interface

The helmets are made by a soft transparent plastic hood built on a hard plastic ring. The base of the plastic ring is connected whit a silicon/polyvinyl chloride soft collar that provides a pneumatic seal at the neck, while the hood contains the patient's entire head.

The collar provides a good seal without major compression at contact points. The lack of pressure points on the face avoids skin necrosis and pain, reduces discomfort, and improves patient tolerance.

The seal around the neck allows the use of the helmet also in patients with difficult anatomical situations that commonly do not allow the use of a facemask.

Different companies produce various types and sizes of helmets, each provided with various fixing and safety features. The choice of the right size generally depends on the circumference of the neck.

The helmet can be used as an alternative interface to the face mask during the NIV (Fig. 2.1) or it can be used to deliver an air flow with high oxygen concentration and a PEEP if simply connected to an air blender (Fig. 2.2).

When this is used instead of the face mask it is connected via an inspiratory branch and an expiratory branch to the mechanical ventilator. If you switch from a NIV therapy with a mask to a helmet it is good, as underlined by numerous works, to increase the inspiratory pressure by a 20%. this because the interface of the helmet has a greater volume and different structural characteristics.

There are little difference in intrinsic characteristics between helmet designed for CPAP (bigger inner volume and softer hood) and helmet designed for NIV (smaller inner volume and harder hood to transmit better the delta pressure).

Fig. 2.1 Helmet for NIV therapy connected with two branch to the mechanical ventilator

Fig. 2.2 Helmet for CPAP therapy connected with one branch to the mechanical ventilator or gas blender

Table 2.1 How set gas flow to reach desired FiO_2 with a gas blender

Total Flow L/min	5	5	5	10	10	10	10	10	15	15	15	15	15
Air L/min	4	3	2,5	9	8	7	6	5	14	12	11	9	7,5
O2 L/min	1	2	2,5	1	2	3	4	5	1	3	4	6	7,5
FiO2%	37 %	53 %	61 %	29 %	37 %	45 %	53 %	60 %	30 %	37 %	42 %	53 %	60 %

Total Flow L/min	20	20	20	20	20	25	25	25	25	25	25	25	30	30	30	30	30	30	30
Air L/min	18	17	16	14	13	22	20,5	19	17,5	16	14	12,5	26,4	24,5	22,5	21	19	16,5	15
O2 L/min	2	3	4	6	7	3	4,5	6	7,5	9	11	12,5	3,6	5,5	7,5	9	11	13,5	15
FiO2%	29 %	33 %	37 %	45 %	49 %	30 %	35 %	40 %	45 %	49 %	56 %	60 %	30 %	35 %	41 %	45 %	50 %	57 %	60 %

Total Flow L/min	35	35	35	35	35	35	35	40	40	40	40	40	40	40	45	45	45	45	45	45	45
Air L/min	30,5	28,5	26,5	24,5	22	17,5	17,5	35	32,5	30,5	28	25	22	20	39,5	37,5	34,5	31,5	28,5	25	22,5
O2 L/min	4,5	6,5	8,5	10,5	13	15,5	17,5	5	7,5	10,5	12	15	18	20	5,5	8	11	13,5	16,5	20	22,5
FiO2%	31,5%	36 %	40 %	45 %	50 %	56 %	60 %	31 %	36 %	41 %	45 %	51 %	56 %	60 %	31 %	35 %	40 %	45 %	50 %	56 %	60 %

Total Flow L/min	50	50	50	50	50	50	50	55	55	55	55	55	55	55	60	60	60	60	60	60	60
Air L/min	44,5	41	38	35	32	28,5	25	49	45,5	41,5	38	34,5	31	27,5	53,5	49,5	45	41,5	38	34,5	30,5
O2 L/min	5,5	9	12	15	18	21,5	25	6	9,5	13,5	17	20,5	24	27,5	6,5	10,5	15	18,5	22	25,5	30
FiO2%	30 %	35 %	40 %	45 %	50 %	55 %	60 %	30 %	35 %	40 %	45 %	50 %	55 %	60 %	30 %	35 %	40 %	45 %	50 %	55 %	60 %

The volume delivered for each act, the respiratory frequency, the sensitivity of the cycling, the PEEP and the FiO_2 will be regulated by the mechanical ventilator options during bilevel ventilation.

During CPAP therapy we set only PEEP level, FiO_2 and liters per minute delivered into the helmet, the respiratory rate and the tidal volume is regulated by the patient.

Considering the significant increase in dead space and the very high compliance of the device may result in inconsistencies between the set volume and the volume actually delivered. This is shown even more clearly when the volumes set are very small [2].

For this reason the use of the helmet is currently reserved for NIV set in assisted ventilation with two pressure levels or CPAP therapy.

When for therapeutic purposes it is only necessary to set a higher FiO_2 together with a PEEP, it is possible to connect the helmet through its inlets port directly to the air blender.

The total air flow and the oxygen flow are regulated as indicated in the table to reach the total air flow and the desired oxygen concentration (Table 2.1).

Another port provide for expiratory exit; a threshold valve is mounted here to generate PEEP.

In addition, there is a pressure release valve which opens in case of sudden absence of air flow to prevent asphyxia in case of technical malfunction.

2.3 Bronchiolitis

Among etiological causes of respiratory distress in childhood, bronchiolitis is the most common etiology in infants <1 year of age admitted to the hospital [1, 3].

Several studies showed that bronchiolitis represents the greatest worldwide cause [4, 5] of infant hospitalization and the 17.1% of all non-elective pediatric ICU admissions [3].

Moreover, it is estimated that 1–3% of hospitalized infants will require treatment in an intensive care unit, especially when risk factors are present [5], and 7–9% of these infants require ventilatory support [6, 7].

The most common symptoms include coughing, wheezing, difficulty eating and sleeping, and apneas.

The principal underlying cause of Bronchiolitis is the respiratory syncytial virus (RSV) infection. It mostly affects children from 0 to 2 years [8] and 1–3% of the worldwide infant population is hospitalized for bronchiolitis during winter months [4, 5]. Management of bronchiolitis mostly involves supportive care that include rehydration and oxygen supplementation [9].

Inflammation of the infant's airways induces an increase in small airway resistance, causing increased ventilatory work [10].

In addition, the predominance of fast twitch muscle fibers in respiratory muscles accelerates fatigue and respiratory failure [11].

The most recent guidelines for management of infants with bronchiolitis and/or other causes of respiratory distress in hospitals emphasize the importance of oxygen therapy, respiratory support, and maintenance of hydration in hypoxia [12].

Respiratory support has traditionally been the cornerstone of intensive care settings and is usually provided by noninvasive techniques or intubation and mechanical ventilation [13–15].

The main common and effective noninvasive respiratory support methods in children with bronchiolitis and/or other etiologic causes, are the high- flow nasal cannula (HFNC) and CPAP, due to its ability to increase functional residual capacity with a reduction of apnoic episodes [16–18].

Both methods are efficient in improving the clinical conditions of patients with mild-to-moderate respiratory distress, although clinical response to helmet CPAP seems to be more efficient and rapid compared with that of HFNC [19].

During CPAP administration, the patient's airway is maintained throughout the respiratory cycle at a selected constant pressure (CPAP) higher than the atmospheric pressure. This method improve respiratory mechanics and gas exchange in patients without neuromuscular diseases, and represent a good supportive therapy in patients with various forms of respiratory distress.

CPAP acts through improving arterial oxygenation and respiratory mechanics and reducing the patient's respiratory drive and effort.

Because the inspiratory effort creates a negative pressure inside the thorax, the ventricle afterload decrease. Accordingly, a decrease in inspiratory effort implies a reduction in the left ventricle afterload. Therefore, venous return and ventricle sizes are reduced with a parallel drop in the wall tension and myocardial oxygen consumption.

In patients with non-hydrostatic pulmonary edema, CPAP could improve gas exchange and respiratory mechanics, thereby increasing the end-expiratory lung volume and preventing alveolar collapse.

The alveolar extension provides a greater gas exchange surface, which improves the respiratory mechanics of ventilation and results in a consequent decrease of $PaCO_2$ in blood gas analysis [20].

Case Report

A 10 months old- infant, 3.5 kg, with negative familiar and medical history, arrived in Emergency Department complaining fever and dry cough from 3 days. At the moment of arrival she presented SpO_2 88% and a blood gas analysis with pH 7.36, pCO_2 44, pO_2 60, Na+ 136; K+ 4.6, Cl- 106, glycaemia 124; Lac 0.8; Hb 11.1; EB -0.5; HCO3- 24.3.

The chest X-ray showed widespread thickening of the bronchial walls and of the peribronchovascular interstitium which is associated with the presence of two shaded areas of increased density localized respectively in the right para-cardiac site and left basal, of possible atelectasis significance.

The patient was admitted in the Intensive Observation Unit and HFNC 2 L/kg/min., hydration, aerosol and antibiotic therapy were promptly started.

During the night an impairment of respiratory mechanic occurred with an increase of respiratory and cardiac rate, fever (T 38.6 C), wheezing and respiratory distress with nasal flaring, chest retractions and increase of respiratory effort.

The physical exam highlighted bilateral and diffuse crackles and a persistent several hypoxemia, despite maximal HFNC therapy, was detected at blood gas analysis.

The chest X-ray was repeated and showed that the areas of hypodiafania with an atelectasis significance appear increased. The widespread thickening of the bronchial walls remains bilaterally. Pleural cavities free from effusion. Cardio-mediastinal image within the limits, in axis.

The infant was admitted in Pediatric Intensive Care Unit and Helmet CPAP 40 L/min FiO_2 50% and peep valve set on 10 cm/H_2O was started.

CPAP was connected to a blender and an active humidification system that deliver a mixture of medical gas at the temperature of 32 °C.

The significant improvement in gas exchange, vital signs and sedation protocol, was reported in the Table 2.2.

Table 2.2 Patient improvement after Helmet CPAP therapy

Timing	pH	PaO_2	P/F	$PaCO_2$	Therapy	BP	HR	RR	Sedation
Pre CPAP	7.34	63	157	51	HFNC 40%	80/55	145	55	Morfine 0.01 mg/kg/h
1 h CPAP	7.36	117	228	51	CPAP 50% PEEP 10 cmH_2O	120/60	95	35	Dexdor 0.7 mcg/kg/h
12 h CPAP	7.42	128	256	44	CPAP 50% PEEP 10 cmH_2O	115/70	98	35	Dexdor 0.7–1.4 mcg/kg/h
24 h CPAP	7.40	178	356	42	CPAP 50% PEEP 10 cmH_2O	109/60	83	30	Dexdor 1.4 mcg/kg/h

Key Teaching Points

- NIV/CPAP erogated with Helmet is safe, well tolerated and effective to improve gas exchange and respiratory system workload.
- Humidity and temperature is two fundamental point to evaluate during CPAP with high fresh gas flow, especially in children, and if it is possible it should be used always an humidification system.
- When Helmet is used instead a mask to deliver NIV remember to set a 15–20% higher pressure to avoid CO_2 rebreathing and optimize respiratory workload
- Helmet is not a good interface to provide NIV in children due to excessive patient ventilator asynchronies and difficult triggering.

Hydration support was maintained with 14 mL/kg/h.

Waiting for microbiological results, corticosteroid and antibiotic therapy with ceftriaxone and clarithromycin were started.

Microbiological tests for Chlamydia Pneumoniae, Mycoplasma Pneumoniae and Bordetella spp. resulted negative, while nasopharyngeal aspirates were positive for Rhinovirus.

Serial radiological controls were made during the hospitalization in the intensive care unit and they showed a progressive improvement of pulmonary ventilation with a persistent upper right lobar hypodiafania and diffuse accentuation of the peribronchus-vascular texture.

Helmet CPAP showed a good patient's tolerance that allowed to a prolonged therapeutic effect with a significant beneficial effect on respiratory mechanics and gas exchange.

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Chapter 3
Mouthpiece Ventilation

Jennifer Obi and Stephen M. Pastores

Case Report
A 34-year-old man with severe spinal injury following a motor vehicle accident and a 2-year history of nocturnal non-invasive ventilation (NIV) use via nasal mask was admitted to the hospital after being noted to be increasingly somnolent. In the emergency department he was found to be hypercapnic and hypoxemic. His mother reported excessive mouth leak. On examination, the patient was drowsy but easily arousable. A continuous face mask NIV was started with marked improvement. Once stabilized, he was discharged with a follow-up visit in the outpatient pulmonary clinic. He continued to use the nasal ventilation. At follow up review, respiratory acidosis reoccurred despite diurnal use of NIV.

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Question: What is the appropriate next step in the management for this patient?
Answer: Continue NIV with intermittent daytime mouthpiece ventilation (MPV) alongside overnight NIV via nasal or face mask.

Following institution of MPV, control of respiratory failure was achieved. Most importantly, independent living was maintained. Intermittent MPV is practical and effective where the limits of ventilator tolerance have otherwise been reached. MPV may reduce the need for tracheostomy ventilation and this case serves as a reminder of the increasing NIV interface options available to clinicians.

3.1 Introduction

The mouthpiece ventilation (MPV) was introduced in the 1950s as a ventilatory mode that can be used as daytime ventilatory support in combination with other ventilatory modalities and interfaces for nocturnal noninvasive respiratory support.

Alveolar hypoventilation is a major complication of many neuromuscular diseases (NMDs). It occurs initially during sleep and subsequently extends into the daytime [1]. Nocturnal noninvasive positive pressure ventilation is the standard mode of initial management of alveolar hypoventilation in NMDs [2]; however as respiratory muscles weakness progresses, the ventilator-free breathing time is reduced significantly. When the number of hours of ventilator use per day exceed an arbitrarily defined threshold (e.g. >16 or 20 h), many practitioners consider transitioning to invasive ventilatory support via tracheostomy.

MPV has been used as an alternative to tracheostomy ventilation for patients requiring continuous ventilatory support for over 60 years. However, there is still a poor understanding of this method's benefits compared with other modalities. This chapter aims to highlight the indications, benefits and drawbacks of MPV.

3.2 Types of Mouth Piece Interfaces

There are two types of oral NIV interfaces: standard narrow mouthpieces with various degrees of flexion, which are held by the patient's teeth and lips; and custom-molded bite-plates (Fig. 3.1). Oral interfaces are used, especially in North America, for long-term ventilation of patients with severe chronic respiratory failure due to severe neurological dysfunction.

The mouth piece is placed between the patient's lips and held in place by lip-seal oral NIV interfaces. Intermittent MPV is practical and effective where the limits of ventilator tolerance have otherwise been reached. MPV may reduce the need for

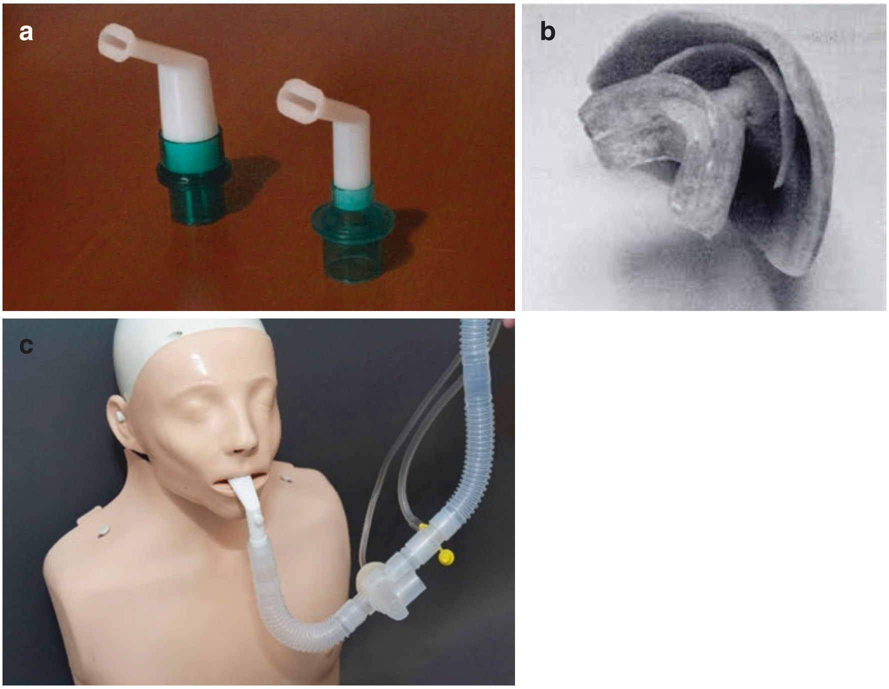

Fig. 3.1 Mouthpiece interfaces: (**a**) 22 and 15 mm angled mouthpieces with adaptors. (**b**) Custom-molded bite-plates. (Reprinted with permission from Copyright Clearance Center.) (**c**) Mouthpiece interface connected to ventilator circuit

tracheostomy ventilation and this case serves as a reminder of the increasing options routinely available to NIV clinicians.

3.3 Indications

MPV is mainly indicated for patients with NMD and chronic respiratory failure when they develop daytime hypercapnia despite optimized nocturnal ventilatory support or when they manifest deteriorating daytime respiratory status with increasing ventilator dependence. In individuals with adequate bulbar muscle function but chronic respiratory muscle insufficiency, intermittent MPV can be an effective alternative to tracheostomy.

Majority of patients considered for MPV have already been using mechanical ventilation for several years. However, the experience of MPV is quite different and some patients may feel uncomfortable and express reluctance to continue. Hence, the application of MPV requires active participation from the patient, increased nursing time and longer periods of training [1].