www.brn.cat

PERMANYER

BRN Rev. 2022;8(3):161-79

BRN Reviews

REVIEW

Main Procedures in the Intermediate Respiratory Care Units

Sarah Beatrice Heili Frades Zimmermann MD, PhD

Pneumology Service, Intermediate Respiratory Care Unit HUFJD-UCIR, Hospital Universitario Jiménez Díaz, Fundación Quirónsalud, Madrid, Spain

ABSTRACT

Respiratory intermediate care units (RICU) are a structure that in terms of complexity is between that of a hospitalization ward and the intensive care unit. However, given that its cost efficiency increases with complexity, the procedures that are carried out in it have also been increasing with it over time. We present the most frequent ones.

Keywords: RICU. Modes of ventilation. Assynchronies. Guided catheterisation. Ultrasound in RICU. Patient-ventilator interaction.

Correspondence to:
Sarah Beatrice Heili Frades Zimmermann
E-mail: sheili@fjd.es

Received in original form: 06-09-2022
Accepted in final form: 16-11-2022
DOI: 10.23866/BRNRev:2022-M0079
www.brnreviews.com

INTRODUCTION

In Spain, as there is a specialty in Intensive Care Medicine, unlike other European countries and the United States, training in respiratory resuscitation at the end of the pulmonology residency is scarce and, in general, insufficient to develop highly complex intermediate respiratory care units, which are the most cost effective[1]. In general, and mainly as a result of the pandemic, the Spanish Society of Pneumology and Thoracic Surgery (SEPAR) is making an intense effort to promote this subspecialty within pulmonology and to favor training and rotations in centers of excellence.

In France, resuscitation training, lasting three years, is done by the resuscitation DESC (Diplôme d'Etudes Spécialisées Complémentaires), therefore qualifying, which makes resuscitation a specialty in its own right (order of June 20, 2002, modified by the order of May 23, 2003)[2]. The resuscitation DESC gives access to exercise in all resuscitation and continuous monitoring services with a medical or multipurpose orientation. It also allows the practice of resuscitation as a specialty in the private sector. As with any medical exercise, the exercise of resuscitation is exclusive, that is to say, that at a given moment one can only practice one specialty, either resuscitation or the original specialty. We consider that two to three years are necessary to complete the training that allows the development of high complexity, that specialty shifts are necessary and a nurse-patient ratio that should not exceed 1:5. All the procedures described in this text are focused on this type of unit, one of which I have the pleasure of directing after completing that training in France.

RESPIRATORY SUPPORT

In the acute care setting, noninvasive ventilation (NIV) should be considered as the first-line approach to treat patients with hypercapnic respiratory failure and continuous positive airway pressure (CPAP) or NIV for respiratory failure due to acute cardiogenic pulmonary edema. Weaning or extubation can be facilitated by high flow nasal cannula (HFNC) and NIV, especially for high-risk or obese patients. The optimal noninvasive support strategy for de novo hypoxemic respiratory failure remains debated: HFNC and helmet support are promising techniques, but careful patient selection and clinical monitoring remain always warranted for the best balance between the benefits and risks of these approaches[3].

High-flow nasal therapy

Humidified high-flow therapy (HFT) is a noninvasive respiratory therapy, typically delivered through a nasal cannula interface, which delivers a stable fraction of inspired oxygen (FiO_2) at flow rates of up to 60 L·min–1. It is well-tolerated, simple to set up and ideally applied at 37°C to permit optimal humidification of inspired gas. Flow rate and FiO_2 should be selected based on patients' inspiratory effort and severity of hypoxemia. HFT yields beneficial physiological effects, including improved mucociliary clearance, enhanced dead space washout, and optimization of pulmonary mechanics. Robust evidence supports its application in the critical care setting (treatment of acute hypoxemic respiratory failure and prevention of post-extubation respiratory failure) and emerging data support HFT use during bronchoscopy, intubation and breaks from NIV or

CPAP. There are limited data on HFT use in patients with hypercapnic respiratory failure, as an adjunct to pulmonary rehabilitation and in the palliative care setting, and further research is needed to validate the findings of small studies. The COVID-19 pandemic raises questions regarding HFT efficacy in COVID-19-related hypoxemic respiratory failure and concerns regarding aerosolisation of respiratory droplets. Clinical trials are ongoing and healthcare professionals should implement strict precautions to mitigate the risk of nosocomial transmission.

A recent prospective physiological study[4] where a high-precision Millar® ultrathin pressure catheter[5] able to reach the alveolar environment under conventional oxygen therapy, and then under high flow oxygen therapy (HFO) 60 L/m highlighted four relevant observations under HFO therapy: 1) intrapulmonary positive end-expiratory pressure (PEEP) was demonstrated, reaching a mean of seven cmH_20; 2) inspiratory pressure decreased (PIP); 3) inspiratory time (IT) shortened, and; 4) expiratory time (ET) lengthened.

In absence of specific measurements, the last two observations could indicate enhanced expiratory resistance. This resistance could lead to the lower respiratory rate (RR) proved in the registry, a finding firmly demonstrated when HFO was applied in healthy volunteers by Brochard and coworkers[6]. In this paper, both inspiratory and expiratory resistance were measured accurately at different flow rates and increased significantly, reducing the RR, a fact that these authors warn should be taken into consideration in resistive patients (asthma, chronic obstructive pulmonary disease [COPD]). Then, the reduction in RR at higher flows would not represent a mechanical improvement as we might believe, but rather a mechanical overload assumed by the patient. Going back to the alveolar intrapulmonary pressure measurements[1], the demonstrated PEEP and the reduced PIP under extreme HFO, unleash *swings* conditioned by alveolar pressure gradient augmentation [PEEP-(-PIP)]. This shearing may translate in alveolar stress and strain situation and thus a potential patient-self-inflicted lung injury [P-SILI][7] that may be taking place while using such high flow rates in widely damaged lungs, as in acute respiratory distress syndrome (ARDS).

In a closer relation with COVID-19, CPAP or HFO should be proposed, as they have shown optimal results in reducing the rate of intubation and mortality[8]. However, just as NIV can induce ventilator-induced lung injury (VILI), it is probably imperative to title HFO to avoid P-SILI. This new concept must urgently be clarified since the Randomised Evaluation of COVid-19 thERapY [RECOVERY] trial[9] did not detect better outcomes with HFO versus CPAP, probably because of the known methodological limitations of the trial (timing of the study, selection of patients and arbitrariness in the choice of the device, as main problems) but we cannot rule out that these hidden phenomena may have influenced the results in an entity with an already very high respiratory drive. New larger physiological studies and probably new clinical trials are needed to conclude with certainty this important issue.

Mechanical ventilation

Respirators

In general, barometric ventilators are preferable because they have the capacity to compensate

for existing leaks and must have minimal information about the patient's response to ventilation, with data on respiratory mechanics, including at least the flow time and pressure time curves. Some devices also offer an estimation of the tidal volume considering the existing leaks, but it is desirable to use devices that have a return path to be able to measure the expired tidal volume, which will mark the degree of ventilation efficiency with greater precision. For invasive tracheostomy ventilation, we recommend wall-mounted gas resuscitation respirators with double tubes and a wide screen that have both ventilation modes and all the functionalities of an ICU respirator. Recently, single-tube respirators have entered the intermediate care units with force with mathematical algorithms capable of estimating the exhaled tidal volume very precisely and that also incorporate modalities that border on automation, as we will comment on later. The use of simplified devices, such as those for home use, has a limited application in cases of initiation of ventilation in acute respiratory failure because they do not offer the possibility of monitoring the degree of adaptation of the patient to the ventilator, asynchrony will not be identified, and they have a narrow margin in the control of thresholds and respiratory cycle times. In a study carried out to assess the estimations of tidal volumes calculated by devices for home use, it was found that most of them had a great inaccuracy, mainly when high pressures were applied in patients with high resistances, a fact that occurs frequently in cases of NIV in acute respiratory failure[10].

THE BEGINNING

NIV should preferably be done with an oral-nasal mask to reduce leaks through the mouth, and a transparent mask to visualize possible secretions or vomiting. Before adjusting the headgear, it is advisable to make a few seconds' approximations, adjusting the mask with the healthcare provider's own hand. An attempt is made to explain the procedure so that the patient understands its usefulness and avoids the anguish of the unknown. This process must be done with the necessary oxygen supply to maintain the fraction of inspired oxygen (FiO_2) indicated in each case. Only when it is certain that it is adapted to ventilation and that the device responds adequately to the respiratory cycles demanded by the patient, is the harness adjusted. The mask model must be suitably individualized, and the nasal bridge adequately padded. Sometimes it is preferable to admit small leaks to having to tighten the headgear too much due to difficulties in adapting the chosen model of mask, because it could reduce the acceptance of the treatment.

The first monitoring measures should be aimed at ensuring effective adaptation, achieving synchrony, and avoiding decoupling or failed impulses. Once this first phase has been achieved, the following steps will be aimed at improving gas exchange. An immediate normalization of arterial blood gases should not be sought, as this is generally not possible. The RR should be less than 25 breaths per minute and the estimated tidal volume greater than 300 ml. If it is a patient with exacerbated COPD, the presence of hypercapnia with acidosis would advise not exceeding an oxyhemoglobin saturation of 90-92%; permissive hypoxemia with the intention of controlling hypercapnia is not prudent, since hypoxemia in any scenario is a bad choice and does not contribute to adequate cardio hemodynamic response and muscular effort, therefore hyperoxia should be limited without

entering hypoxemia. Lastly, the appearance of secondary effects such as aerophagia, pressure ulcers from the mask, blood gas deterioration, agitation, etc., should be monitored.

Ventilation mode

NIV, invariably associated with the presence of leaks, must be done with pressure systems with leak compensation. Initially, better adaptation is achieved with the spontaneous breathing mode, thus facilitating efficiency and synchrony. The controlled breathing mode is reserved for cases of inhibition of the ventilatory drive or cases of proven adaptation. If NIV is started in controlled mode, the feared decoupling and patient-respirator asynchrony may appear. In terms of energy savings for the patient, the greatest efficacy of mechanical ventilation is achieved with the controlled breathing mode because it avoids the initial inspiratory effort to trigger the ventilator, but this mode is not easy to use and requires special expertise and specific training in neuromechanical coupling from the clinician, especially regarding the choice of the inspiratory time that must coincide with the neural one in what we will call the "*scanning procedure*", which we will explain later. The pressure levels must be low at the beginning. If a bilevel positive airway pressure (BIPAP) is used, an inspiratory pressure of around 10-12 cmH_2O and an expiratory pressure of around 5-6 cmH_2O can be programmed, with the FiO_2 necessary to achieve the desired level of oxygenation. From this point on, the changes will be made according to the curves, clinical parameters, RR and estimated tidal volume, and to the gasometric parameters. Inspiratory pressure can be increased slowly until an estimated tidal volume greater than 300 mL and a respiratory rate of less than 25 breaths per minute are achieved. If hypoxemia is severe, expiratory pressure can be slowly increased until an oxygen saturation (SaO_2) of 90% is achieved with an FiO_2 of less than 60%. After each change of parameters, the sensations perceived should be discussed with the patient. A rapid clinical-gasometric correction should not be attempted because this is not generally possible or recommended, since in the long run it will produce lung-kidney decoupling. The hypothetical rule that an increase in inspiratory pressure will be accompanied by a decrease in arterial blood gases ($PaCO_2$) may not be true in many cases and, rather, patient intolerances will occur. The appearance of gasometric deterioration in the first controls should lead to reconsideration of the treatment.

Apart from the conventional modes of pressure support and controlled pressure, there are new ventilatory modes capable of, through complex mathematical algorithms, automatically adjusting the patient's inspiratory and expiratory triggers in such a way that ventilation is notably simplified, and these modes are notably adjusted in addition to respiratory variability, improving the synchrony between the patient and the respirator.

There are intelligent modes, such as average volume assured pressure support (AVAPS) and intelligent volume assured pressure support (IVAPS), but their use now is focused on chronic patients with home ventilation and frequent associated sleep pathology. More advanced[11] modes such as Autotrack works very well in the acute settings; others as Nava or Intellivent currently belong to the environment of intensive medicine or the weaning phase in the intermediate respiratory care

unit (RICU) in tracheostomized patients, and their capabilities in non-invasive ventilation are still under evaluation.

Thresholds

The ventilator activation thresholds can be pressure or flow. The patient must generate a preselected negative pressure to activate the ventilator in the first case or start a determined inspiratory flow in the second. With pressure threshold, the respiratory muscles generate an isometric contraction, whereas the contraction is isotonic in the case of flow threshold. There is a variant of flow activation in which the ventilator identifies not only a flow signal but also a change in its profile, facilitating adaptation in certain cases[12]. In general, it is considered that the selection by flow threshold is better than by pressure threshold, but its difference in terms of clinical efficacy or adaptation to the ventilator has not been determined[13].

When the thresholds are programmed to be very sensitive, the so-called "auto-trigger" may appear, which consists of the appearance of an activation of the ventilator without a previous inspiratory impulse by the patient. It occurs mainly in situations of very low respiratory rates and with decreased ventilatory impulses. At the opposite extreme is the so-called "failed cycle", where the patient generates an inspiratory effort that does not reach the flow threshold, a situation that frequently occurs in cases of dynamic hyperinflation or if the inspiratory trigger is excessively demanding. Both the "auto-trigger" and the "failed cycle" are two asynchronies that can occur during assisted mechanical ventilation and have important clinical and pathophysiological consequences because they increase the work of breathing and can lead to NIV failure and inspiratory muscle damage.

Flows

By analyzing the pressure-volume curves, the work done by the patient and by the ventilator can be calculated. With this method, it has been possible to assess that reducing the sensitivity of the inspiratory threshold and the inspiratory flow increases the patient's workload, the opposite of programming a more sensitive threshold and a higher flow, with differences of up to 65% of work by the patient from one case to another[14]. The same conclusions were obtained in another study where patient comfort was assessed with comfort scales, with high flows being the ones best tolerated by patients[15].

By varying the pressurization ramp, which mainly affects the initial inspiratory flow, it is verified that the intermediate ramps had the best results in terms of tolerance and only the excessively low ramps changed the respiratory pattern, causing a decrease in tidal volume and increases in respiratory rate and work of breathing[16].

Pressures

As inspiratory pressure increases, the work exerted by the inspiratory muscles decreases, thus obtaining the desired effect of muscle rest. However, when pressure support is excessive, increased expiratory muscle recruitment is forced at the end of inspiration, and

diaphragm activity overlaps with that of the expiratory muscles, indicating that the patient is fighting the ventilator and the patient wants to end the inspiration he considers excessive in these cases. These data have been confirmed in patients with COPD by studying the pressure-time curves[17] and in normal subjects with hyperinflation caused by Starling's resistor by studying the activation of the diaphragm and the transversus abdominis muscle[18]. In these cases, in addition to the patient's intolerance, an increase in energy expenditure can be observed that would worsen the patient's clinical situation and an increase in the appearance of failed impulses at the beginning of the next cycle.

Respiratory cycle times

Under normal conditions, the respiratory cycle occupies one-third of the time in inspiration and two-thirds in expiration. Starting from this reference, the cycle must be able to maintain an adequate tidal volume and full expiration. For this, there will be a tendency to shorten the inspiratory time and thus allow prolongation of the expiratory time in cases of airflow obstruction and facilitate adequate pulmonary emptying, sufficient to avoid air trapping. From this point, increases in inspiratory time, without exceeding the ratio of 1:1, may be necessary in cases that require greater ventilatory efficiency. To avoid an excess of inspiratory time, the ventilators incorporate in their programming the "maximum inspiratory time" variable, which in the presence of leaks can become very high and a source of asynchrony, so if it is modifiable, it must be adjusted to a physiological time of 0.8 to 1 second.

Presumably, the inspiratory time provided by the ventilator should coincide with the neuronal activation time of the inspiratory muscles, but this coincidence does not always happen, and the ventilator concludes its inspiration well before or after the inspiratory effort has ended, which can add another source of asynchrony. If the inspiration provided by the ventilator ends before the neuronal activation, the sustained activation of the inspiratory muscles could cause a new activation of the ventilator within the same cycle (double *trigger*), an asynchrony that occurs in pressure support modes with trigger or threshold very sensitive expiratory time or in pressure-controlled mode with an excessively short set inspiratory time. On the other hand, in the pressure support modes with very insensitive trigger or expiratory threshold or in the controlled pressure mode with an excessively long set inspiratory time, the patient will not be able to cycle to expiration because the ventilator is in the inspiratory phase showing a plateau that indicates an "ineffective effort" in emptying the patient. The trigger or expiratory threshold is therefore a critical parameter when it comes to optimizing the neuromechanical coupling between the patient and the ventilator[19].

Asynchrony detection

Asynchrony during the inspiratory trigger phase

The patient's inspiratory effort initiates gas delivery by the ventilator upon reaching a certain threshold, *trigger,* programmed by the operator[20]. The *trigger* can be programmed to detect a change in pressure, flow, volume, or use the flow curve (this is the algorithm used

by the Philips Respironics auto-track system: the flow signal drawn by the ventilator's own software on the curve of the patient's flow, with a delay of 300 milliseconds on the latter and with a flow difference of 0.25 l/s).

Asynchrony due to inspiratory *trigger* can be expressed as autocycling (triggering of the ventilator in the absence of patient effort), *trigger delay* (delay time from when the patient exerts until the ventilator delivers gas flow) and ineffective efforts (muscular efforts of the patient that do not trigger the ventilator)[21].

Autocycling can occur as a result of expiratory leaks in the ventilator circuit, the presence of water in the circuit, and cardiac oscillations. The most common is expiratory leak, which activates cycling when the start condition occurs, corresponding to inspiratory tidal volume > expiratory tidal volume. Autocycling can considerably interfere with patient management, being able to decrease the partial pressure of CO_2 in $PaCO_2$ and affect inspiratory effort, being able to induce respiratory events of the absence of impulse and glottic closure due to overventilation, in addition to significantly impairing the tolerance.

Pressure and flow curves help detect this problem. The absence of an initial drop in pressure below the end-expiratory pressure is indicative of auto cycling, the absence of indentation in the pressure curve that expresses the voluntary nature of the cycle, and the reduction of the area under the expiratory curve are all indicators of this asynchrony.

This form of asynchrony can be minimized by increasing the pressure or flow threshold for ventilator triggering, increasing the patient's central drive (decreasing sedation), and eliminating expiratory leaks (mask fit and decreased peep)[22].

Trigger delay and failed effort have common causes. They are related to factors derived from the ventilator and due to the patient himself. Patient factors include dynamic hyperinflation, low central respiratory drive, and muscle weakness. Regarding the ventilator, high levels of assistance and expiratory asynchrony in the form of the delayed opening of the expiratory valve (mechanical inspiratory time greater than neural inspiratory time) are the factors associated with *trigger* delay and failed efforts[23-24].

In previous studies, it was said that the inspiratory effort was greater with the pressure *trigger* than with the flow trigger. However, with new generation devices this difference is negligible. However, the *auto-track* or flow signal form decreases *trigger* delay and ineffective efforts. To reduce the delay in triggering the ventilator and ineffective efforts, it is important to increase the pressure generated by the inspiratory muscles during the *trigger phase* (decreasing the level of sedation and correcting alkalosis), apply external PEEP to counteract auto-PEEP, decrease the pressure or flow threshold of the ventilator *trigger and use new generation ventilators, with a mean trigger delay time* of 100-120 milliseconds.

Asynchrony during the pressurization phase

Asynchrony during the pressurization phase will occur whenever the patient's flow demand

is not supplied by the flow provided by the ventilator. The time in which the set pressure is reached, called the ramp time (*rise time)*, can have a significant influence on the synchrony during this phase. There is no hard and fast rule to determine which ramp time is best, but we know that both long and excessively short times are associated with asynchrony[25].

Although a reduced inspiratory flow produces asynchrony with the appearance of short cycles, with duration < one half of the duration of the patient's normal cycle and swallow breathing - this is high-frequency, low-volume breaths that increase the work of breathing[26]-, an excessively high flow can cause tachypnea in the patient due to this same effect, so the choice of ramp must be individualized. For very fast flows, the effect under discussion seems to be mediated by the Hering Breuer reflex (reflex inhibition of inspiration when a certain threshold volume is reached). Excessively fast flow would activate the pulmonary stretch receptors, which, via the vagal route, inform the respiratory center, which would quickly cut off inspiration. Other authors attribute the increase in respiratory rate to a shortening of inspiratory time specifically motivated by the high flow provided (flow-related reflex termination of inspiration), and not mediated by the Hering Breuer reflex[27].

Double triggering

This is a form of asynchrony that can be observed when the patient's demand is very high, and the mechanical inspiratory time is short. It is identified as two consecutive inspiratory cycles without observing the expiratory phase between them. The patient's inspiratory effort continues after the ventilator's inspiratory time has elapsed, which causes, upon reaching the *trigger* threshold, a new *trigger* by the ventilator. The double *trigger* occurs mainly in the pressure control ventilation mode by programming an excessively short inspiratory time, but it also occurs in pressure support mode when setting an excessively sensitive expiratory *trigger*.

On the other hand, when in the pressure control ventilation mode, we program an excessively long inspiratory time, or in pressure support mode, when setting an excessively insensitive expiratory trigger, the ventilator does not allow cycling to expiration and a plateau is noted in the flow curve that will be longer the longer the inspiratory time programmed directly in pressure control mode is or indirectly in pressure support mode. This is another type of ineffective effort that has to do with asynchrony of cycling to expiration. The optimal neuromechanical inspiratory time travels between these two asynchronies and should be optimized by avoiding them in what can be called a "sweep procedure" if we modify this inspiratory time based on the observation of these two asynchronies[28].

Expiratory dyssynchrony

Expiratory dyssynchrony occurs when the mechanical inspiratory time precedes or exceeds the neural inspirtory time. In pressure support ventilation, the transition from inspiration to expiration, called "cycling", occurs when the patient's inspiratory flow (Vinsp)

decreases to a predetermined fraction of the peak inspiratory flow (Vinsp/Vpeak). Ideally, flow delivery should end when the patient's inspiratory effort ceases. In theory, synchronization can be obtained if the Vinsp/Vpeak ratio, that is, the expiration cycle or expiratory *trigger,* is equal to the ratio between the flow present at the end of the inspiratory effort (Vti) and the Vpeak (Vinsp/Vpeak = Vti/Vpeak). However, this may not happen, since the expiration cycle criterion is given, as mentioned above, by a certain value of flow or a percentage thereof with respect to the peak flow. When the mechanical and neural timings do not coincide, asynchrony occurs and the work of breathing increases[29,30]. We should program high cycling criteria, above 25%, in obstructive patients, who have prolonged time constants, and below 25% in restrictive patients, with shorter time constants. On some occasions, the inspiratory effort is of such a magnitude that it reaches the trigger threshold of the ventilator, and a double *trigger* occurs in the same breath. Similarly, an excessively insensitive expiratory *trigger* prolongs inspiration and causes prolonged respiratory times that in tachypneic subjects prevent the patient's demands from being met by the respirator, again appearing ineffective efforts. If this trigger is not set correctly and in the presence of significant inspiratory leaks, the ventilator will continue to deliver flow until Ti max is reached, which most of the time cannot be modified and is preset to an unphysiological value of three seconds. To abolish this asynchrony known as prolonged inspiration, it is necessary to adjust the mask, or reduce the inspiratory positive airway pressure (IPAP) or increase the expiratory trigger, making it more sensitive so that it exceeds the leak flow, or adjust the Ti max if possible. If none of this is possible, consider switching to pressure control mode that cycles by time[31].

Modern ventilators have the ability to vary the criteria from cycling to expiration, especially the flow line algorithms discussed above.

Interfaces and patient-ventilator interaction

There are six different types of interfaces on the market: oronasal mask, total mask, nasal mask, mouthpiece, nasal olives and *Helmet.* Of these, the oronasal mask is the most used in the situation of acute respiratory failure. Compared to nasal masks, they achieve higher airway pressures, are not affected by mouth breathing, have fewer leaks, and require less patient cooperation. On the contrary, they are less comfortable, the patient cannot speak, eat or drink during ventilation. If the erosion of the bridge of the nose occurs, we can resort to the full mask or the *helmet*[32].

Using the correct interface is crucial to the success of NIV. Reducing excessive air leak is an important aspect, since it produces asynchrony and decreases the pressures reached in the airway, reducing the effectiveness of the technique. Choosing the right size mask is convenient, but so is choosing the headgear that holds it. The fixation of the mask to the patient's face can be optimized with padding at the chosen interface. However, it is inevitable that some leakage will occur, and it is advisable to use respirators that can monitor and compensate for them[33].

In an RICU, it is necessary to have specially designed interfaces to be able to insert a bronchoscope or gastroscope without increasing leaks[34]. Another important aspect is the dead space of the mask (VDm). Apart from the static dead space of the interface (VDme), directly related to its volume, some authors add the dynamic dead space (VDdyn), which is due to the amount of exhaled gas that is inhaled again, limiting and compromising the alveolar ventilation. This VDdyn can be counteracted with sufficient flow during expiration. In this sense, it has been verified that the dead space volume of the mask has little repercussion if there is adequate pressurization during the expiratory phase. This flow efficiency during expiration could be optimized by using expiratory ports on the nasal bridge of the mask[35]. Another study compares the oronasal mask with the *helmet* in pressure support (PS) mode. The use of a *helmet* to provide SP increased inspiratory effort promoted the appearance of asynchrony, worsened CO_2 elimination, and increased dyspnea, compared with the oronasal mask[36].

Humidification and patient-ventilator interaction

For years, there has been a consensus on the need to condition inspired gases, and that this affects the maintenance of adequate ciliary function and rheological characteristics of respiratory mucus. The humidification of inspired gases is essential in the treatment of patients who need mechanical ventilation through an orotracheal tube and in non-invasive motion ventilation (NIMV).

To humidify and heat the inspired gas, we have heat-moisture exchangers (HME) and active humidifiers. Compared with active humidifiers, HME makes NIMV less effective in reducing inspiratory effort in patients treated for acute respiratory failure. This may be due to an increase in dead space, which has negative effects on gas exchange. On the other hand, they add resistance to the system due to the multiple membranes used for humidification and, finally, they have a non-negligible dead space and since for their effectiveness they must be located between the mask and the leak or the Y of the double tube, and this dead space affects also gas exchange and reduces effective alveolar ventilation. Finally, it has been shown that in the presence of leaks, they lose 50% of their effectiveness, so in non-invasive ventilation, where leaks are almost universal, their benefit is not clear[37].

Co_2 rebreathing and ventilator patient asynchrony

With the use of NIMV single-branch ventilators, there is the possibility of CO_2 rebreathing. If this occurs, the ventilatory drive increases and contributes to the appearance of asynchrony. Rebreathing can be minimized if the expiratory port is in the mask and not in the ventilator tubing, if oxygen is delivered through the mask itself, with a minimum expiratory positive airway pressure (EPAP) level (5 cmH_2O), or with anti-rebreathing devices, like the *plateau* valve. The major determinants for rebreathing CO_2 are the expiratory time and the flow through the circuit during expiration, so a minimum level of EPAP is necessary, as has been mentioned, to prevent rebreathing.

VASCULAR MANAGEMENT-CATHETERS

Arterial catheterization

Arterial line catheterization is generally considered to be a safe procedure with few serious complications and a major complication rate ranging between 1% and 5%[38]. There are very few absolute contraindications to the placement of an A-line, and they include an absent pulse, burns over the cannulation site, inadequate to the extremity, and Raynaud's syndrome. Most arterial catheters are placed in radial or femoral arteries with the radial artery being the preferred site due to lower risk of severe complications of ischemia, bleeding, and infection.

In critically ill and hemodynamically unstable patients, noninvasive blood pressure monitoring techniques may underestimate blood pressure; thus, the more intensive blood pressure monitoring via arterial catheterization may be beneficial[39]. Technological advances in contemporary design of catheter and monitoring systems now allow arterial lines to be used for more advanced hemodynamic monitoring, including real-time calculation of cardiac output, stroke volume, and evaluation of fluid responsiveness in suspected hypovolemic states. Ultimately, the beat-to-beat hemodynamic information provided by arterial line catheterization is only as valuable as the pulmonologist's interpretation of these data. We always place an arterial catheter because it does provide reliable information on hemodynamics and allows us to perform the necessary arterial blood gases, avoiding pain for the patient or unnecessary awakenings if nocturnal information on gas exchange is required. Logically it is necessary to know the technique perfectly, to have followed directed training and to know how to interpret the signals[40].

Central venous catheter catheterization

Central venous catheters (CVC) and dialysis catheters are inserted in three out of four critically ill patients' intensive care units, being less frequent in RICU. Complications included local insertion site complications, infections and thrombosis[41,42]. These adverse events are responsible for heavy morbidity and mortality and additional costs, although they can be avoided in the great majority of cases. Healthcare improvement programs and quality improvement strategies have been shown to be effective to prevent complications related to intravascular catheters[43], especially when there is local compliance with the measures.

Recommendations regarding catheter-related infection prevention included the preferential use of subclavian central vein, a one-step skin disinfection using 2% chlorhexidine-alcohol, and the implementation of a quality-of-care improvement program. Antiseptic- or antibiotic-impregnated CVC should likely not be used. Catheter dressings should likely not be changed before the seventh day, except when the dressing gets detached, soiled, or impregnated with blood. Ultrasound guidance should be used to reduce mechanical complications in case of internal jugular access, subclavian and femoral venous access. In the RICU it is always necessary to have at least two good vascular accesses, given that the patient's situation may suffer unforeseen

acute changes, since they are unstable patients and therefore require monitoring. But we monitor to interpret, decide, and treat and the treatment is often intravenous. When it is suspected that the patient will need parenteral nutrition, the peripheral accesses are of poor quality, or the patient has borderline hemodynamics or requires vasoactive or inotropic drugs. The staff of the unit must know how to place these accesses and train their residents.

IMAGING SUPPORT. LUNG SONOGRAPHY-FAST AND E-FAST EXAM

Lung sonography and fast exam

We will refer to ultrasound in a very general way. A very detailed approach to pulmonary sonography is published in the book by Frades H et al.[44].

In essence, in the RICU it is necessary to train in enlarged pulmonary ultrasonography and given the multidisciplinary origins of the patients (medical and surgical) it is very important to know how to perform the focused assessment with sonography for trauma (FAST) and extended FAST (eFAST), rapid evaluation of the chest for detection of pneumothorax, exam[45].

Primary ultrasound windows for the FAST examination include the following:

1. The right upper quadrant view (also known as the perihepatic, Morison pouch, or right flank view). This uses the liver as an ultrasound window to interrogate the liver as well as the hepatorenal space (Morison pouch) for free fluid. Slight cephalad movement of the transducer allows imaging of the right pleural space for free fluid. Care should be taken to carefully insonate the area between the dome of the liver and diaphragm to identify free fluid that may accumulate there. Caudal probe movement allows visualization of the inferior pole of the right kidney as well as the right paracolic gutter for free fluid assessment.

2. The left upper quadrant view (also known as the perisplenic or left flank view). This uses the spleen as a window to interrogate the spleen and the perisplenic space above the spleen, below the diaphragm, and the splenorenal recess. Scanning cephalad allows visualization of the left pleural space. Scanning caudad allows visualization of the inferior pole of the left kidney and the left paracolic gutter.

3. The pelvic view (also known as the retrovesical, retrouterine, or pouch of Douglas view). This allows assessment of the most dependent space in the peritoneum for free fluid. Analysis through a fluid-filled bladder (which can be filled, if necessary, by fluid placed through a Foley catheter or clamping the Foley catheter) may help analysis for pelvic fluid. When free fluid is present, it is noted most often posterior or superior to the bladder and uterus. The bladder should be scanned in its entirety in both the sagittal and transverse planes.

4. The pericardial view (also known as the subcostal or subxiphoid view). This uses

the left lobe of the liver as an acoustic window for analysis of the heart, particularly its right side. Both sagittal and transverse four-chamber planes may be used. The potential space of the pericardium is analyzed for the presence of any free fluid in anterior or posterior locations. Slight angulation posteriorly or inferiorly in this view allows visualization of the inferior vena cava (IVC) and hepatic veins, including their normal respiratory variability. A midline to slightly off midline longitudinal view or coronal view through the patient's sides can also allow analysis of the IVC.

5. The anterior thoracic view. The pleura normally apposes each other and slide on each other easily. Absence of this sliding and the potential separation of the pleura by a pneumothorax may be imaged typically in the second or third intercostal space with a higher frequency near-field transducer, although lower-frequency transducers may also be used. Other intercostal spaces may also be used for lung analysis. The identification of a lung point is highly specific for the diagnosis of pneumothorax and should be sought when time allows. A lung point represents the site where the lung adheres to the parietal pleura immediately adjacent to the pneumothorax.

Additional dedicated views may include the following:

6. The right and left pericolic gutter views. Longitudinal and transverse views through peritoneal windows inferior to the level of the ipsilateral kidney and next to the ipsilateral iliac crest may reveal free fluid surrounding the bowel. These windows may be of limited use because of the absence of an acoustic window, such as a fluid-filled bladder or a solid organ. Air-filled bowel may also limit these views. The presence of larger amounts of fluid may aid in visualization. The images may be obtained coronally or from an anterior approach.

7. The pleural space views. Each pleural space may be investigated via angulation and cephalad movement of the transducer along the ipsilateral flank. Abnormal fluid collections in the pleural space are visualized as anechoic collections above the echogenic diaphragm. At times, fluid that may be hemorrhagic, proteinaceous, or infectious will appear more echogenic or complex in nature. An upright or slight reverse Trendelenburg position of the patient may assist in the detection of pleural fluid.

8. The parasternal view. The parasternal window allows visualization of the heart in the long or short axis. These views are used in cases in which a patient's subcostal view is suboptimal.

9. The apical view. The apical view may allow visualization of pericardial fluid in the difficult patient by placing the transducer at the nipple line at the left fifth intercostal space and aiming it toward the spine or the right shoulder.

10. Inferior vena cava views. Multiple views of the IVC are accessible by using either a subxiphoid or lateral approach. The

lateral approach makes use of the liver as an acoustic window. The primary aim of IVC evaluation is to aid in the assessment of the intravascular volume status. IVC evaluation is particularly useful in those patients at the extreme ends of the spectrum: either hypovolemic (e.g., secondary to massive hemorrhage) or severely fluid-overloaded. IVC evaluation has also been shown to be useful in gauging fluid responsiveness in patients requiring volume resuscitation or transfusion of blood products.

MANAGEMENT OF COMPLEX PATIENTS EXCEPT WEANING FROM INVASIVE MECHANICAL VENTILATION

Pulmonary arterial hypertension monitoring and management

Patients with pulmonary arterial hypertension (PAH) who are admitted to the RICU pose a challenge to the multidisciplinary healthcare team due to the complexity of the pathophysiology of their disease state and the medication considerations that must be made to appropriately manage them.

Management of patients with PAH in the RICU is complex as it requires a careful balance to maintain perfusion while optimizing right-sided heart function. A comprehensive understanding of the underlying physiology and underlying hemodynamics is crucial for the management of this population. PAH is diagnosed with a right heart catheterization or Swan–Ganz catheter showing pulmonary arterial mean pressure greater than 25 mm Hg and not by echocardiogram. Treatment of patients with evidence of right heart failure due to pulmonary hypertension involves supportive care, correcting the underlying cause of the hemodynamic instability and supporting hemodynamic function of the right heart. Intubation of patients with pulmonary hypertension and right ventricular failure (RVF) should be avoided. Pulmonary vasodilators are used to reduce RV afterload by reducing pulmonary arterial pressures. Medications effective at reducing RV afterload, such as intravenous prostanoids, inhaled nitric oxide (iNO) cause improvements in cardiac output and oxygenation; iNO can be administrated via high-flow oxygen and non-invasive ventilation devices. Inotropes, such as Dobutamine and Milrinone are used to maintain cardiac output in the presence of cardiogenic shock from right heart failure due to pulmonary hypertension. Multidisciplinary work is necessary with the services related to the cause of this hypertension and cardiology/hemodynamics. If the patient is not eligible for admission to the ICU[46] or to the coronary care unit, they should be able to be managed in the RICU, especially if the cause is pulmonary (embolism, interstitial pathology, etc...). Ventral and arterial venous access is necessary.

Postextubation and postoperative patients and noninvasive support in the RICU

Although high-flow nasal oxygen seems to be an effective alternative to standard oxygen in patients with low risk of extubation failure in ICUs, the prophylactic use of NIV should be proposed as the first-line strategy

of oxygenation in patients with high risk of failure. By contrast, standard oxygen seems sufficient in postoperative patients and high-flow nasal oxygen should be used in patients with hypoxemia.

NIV may decrease the risk of intubation in postoperative patients with respiratory failure, but it could increase the risk of death by delaying reintubation in patients with post-extubation respiratory failure in the ICU[47].

SPECIAL TREATMENTS

Sedoanalgesia and vasoactive drugs

The Society of Critical Care Medicine[48] recommends sedation with either dexmedetomidine or propofol targeted to light levels of sedation for adults receiving mechanical ventilation and continuous sedation. Strict respiratory and hemodynamic monitoring is obliged. These drugs reduce the common delirium in critically ill patients. In our patients with a lot of dyspnea, mild perfusions (0.25 to 1 mg/h) of morphine may be necessary. It must always be remembered to stimulate intestinal activity with prokinetics and laxatives to avoid persistent constipation in patients. The adjustment of vasopressor and inotropic drugs will always depend on the patient's hemodynamic status according to the intensive medicine service, especially if the patient is eligible for admission to this unit. Otherwise, there is no reason not to administer it to the patient if it improves their prognosis and the conditions of monitoring and the presence of a specialist 24/24 hours are guaranteed.

ENDOSCOPIC PROCEDURES IN THE RICU

Flexible and rigid bronchoscopy

Bronchoscopy is the endoscopic examination of the tracheobronchial tree. Flexible bronchoscopy has largely replaced rigid bronchoscopy as the procedure of choice for most endoscopic evaluations of the airway. Rigid bronchoscopy, however, is indicated for the removal of large foreign bodies, which may be difficult to remove with the flexible bronchoscope, and in the evaluation of patients with massive hemoptysis. Flexible bronchoscopy is easily performed, is associated with few complications, and allows greater visualization of the tracheobronchial tree than rigid bronchoscopy. In tracheostomized patients, the tube provides easy access to the lower respiratory tract while still allowing ventilation. A size 8.0 mm is required to allow passage of the bronchoscope while allowing adequate ventilation (sometimes a size 7.5 tube may suffice). Several diagnostic and therapeutic procedures can be performed with the fiberoptic bronchoscope including bronchoalveolar lavage (BAL), biopsy of intrabronchial lesions, protected microbiology specimen brush (PSB) and cytology sampling, as well as a transbronchial biopsy.

The use of fiberoptic bronchoscopy (FB) in RICU has multi-modal indications (see Table 1).

Hypoxemia related to FB is associated with an increase in cardiovascular stress and workload. Despite this, clinically significant major arrhythmias are infrequent. Increases

Table 1. Multi-modal indications for FB in RICU

Clinical indications	Inspection	Sampling	Therapeutic
Aspiration	X	X	X
Infection	X	X	X
Lobar collapse/atelectasis	X	X	X
Airway management (i.e., difficult intubation)	X		X
Airway assessment (i.e., acute inhalation injury, burns, trauma, tracheostomy insertion, mass lesions, tracheoesophageal fistula)	X		X
Foreign body	X		X
Strictures and stenosis	X		X
Hemoptysis/hemorrhage	X		X

in systolic arterial pressure and heart rate during FB are associated with electrocardiogram (ECG) changes in 15% (ST-T changes in 4%, transient right bundle branch block in 3%). Unexpected ST changes have been reported in 21% of awake patients over 60 years of age[49]. RICU patients can have inotrope/vasopressor requirements and most frequently potentially an element of cardiovascular compromise. Bronchoscopy within 30 days of acute myocardial infarction is associated with 5% mortality and limited to patients with active ischemia at the time of bronchoscopy. In the absence of active ischemia, with good clinical justification, FB can be performed but is at the discretion of the clinician[50].

A potential complication of FB is increased intracranial pressure (ICP) during airway manipulation. It is important to take this into account, especially in patients of neurological and neurosurgical origin, which are very frequent in our units for airway management or weaning. Minor bleeding occurs in 0.19% and is severe in 0.26% of bronchoscopies[51]. Risk factors for abnormal coagulation include the use of anticoagulant therapy, liver disease, or history or family history of bleeding tendency. Hemoglobin, platelet, and coagulation studies should be performed always before FB.

In the rare cases of upper airway obstruction making ventilation and eventually intubation impossible, which cannot be solved through flexible bronchoscopy, an emerging rigid bronchoscopy may become necessary. The entire RICU team must be familiar with this procedure.

Assistance to complex endoscopic procedures in the RICU

Given the knowledge and training in Sedoanalgesia, cardiohemodynamic and respiratory support, complementary risk procedures such as upper gastrointestinal endoscopy, colonoscopies and transesophageal echocardiograms

can be performed on RICU patients, thus avoiding unnecessary hospital transfers that are not without risk. All these gestures deserve a notice to the ICU.

FUNDING

None to disclose.

CONFLICTS OF INTEREST

None to disclose.

ETHICAL DISCLOSURES

Nothing to disclose.

REFERENCES

1. Heili-Frades S, Carballosa de Miguel MDP, Naya Prieto A et al. Is It Really Efficient and Safe? Arch Bronconeumol (Engl Ed). 2019;55:634-41.
2. https://www.education.gouv.fr/bo/2003/25/MENS0301178A.htm
3. Ferreyro BL, Angriman F, Munshi L et al. Association of noninvasive oxygenation strategies with all-cause mortality in adults with acute hypoxemic respiratory failure: a systematic review and meta-analysis. JAMA. 2020;324:57–67.
4. Naya Prieto A, López Chang C, Fernández Ormaechea MI et al. High flow oxygen therapy (HFO) in patients with acute respiratory failure: hemodynamics, gas exchange and respiratory mechanics measurements in lung alveoli. ERJ Open Research. 2022;8(suppl 9);38
5. https://millar.com/Clinical/MikroCath/#quicktabs-mikro_cath_airway_pressure_cathe=0
6. Vieira F, Bezerra FS, Coudroy Ret al. High Flow Nasal Cannula compared to Continuous Positive Airway Pressure: a bench and physiological study. J Appl Physiol (1985). 2022 May 5.
7. Brochard L, Slutsky A, Pesenti A. Mechanical Ventilation to Minimize Progression of Lung Injury in Acute Respiratory Failure. Am J Respir Crit Care Med. 2017;195:438-42.
8. Heili-Frades S, de Miguel MPC, Prieto AN et al. The Role of Intermediate Respiratory Care Units in Preventing ICU Collapse during the COVID Pandemic. Int J Respir Pulm Med. 2020;7:147.
8. Perkins GD, Couper K, Connolly B. RECOVERY-Respiratory Support: respiratory strategies for patients with suspected or proven COVID-19 respiratory failure; continuous positive airway pressure, high-flow nasal oxygen, and standard care: a structured summary of a study protocol for a randomised controlled trial. Trials. 2020;21:687.
10. Lofaso F, Fodil R, Lorino H et al. Inaccuracy of tidal volume delivered by home mechanical ventilators. Eur Respir J. 2000;15:338-41.
11. Suarez-Sipmann F; Acute Respiratory Failure Working Group of the SEMICYUC. New modes of assisted mechanical ventilation. Med Intensiva. 2014;38:249-60.
12. Kondili E, Prinianakis G, Georgopoulos D. Patient-ventilator interaction. Br J Anaesth. 2003;;91:106-19.
13. Goulet R, Hess D, Kacmarek RM. Pressure vs. Flow triggering During Pressure Support Ventilation. CHEST. 1997;111:1649-53.
14. Ward ME, Corbeil C, Gibbons W, Newman S, Macklem PT. Optimization of respiratory muscle relaxation during mechanical ventilation. Anesthesiology. 1988;69:29-35.
15. Manning HL, Molinary EJ, Leiter JC. Effect of inspiratory flow rate on respiratory sensation and pattern of breathing. Am J Respir Crit Care Med. 1995;151(3 Pt 1):751-7.
16. Chiumello D, Pelosi P, Croci M, Bigatello LM, Gattinoni L. The effects of pressurization rate on breathing pattern, work of breathing, gas exchange and patient comfort in pressure support ventilation. Eur Respir J. 2001;18: 107-14.
17. Jubran A, Van de Graaff WB, Tobin MJ. Variability of patient-ventilator interaction with pressure support ventilation in patients with chronic obstructive pulmonary disease. Am J Respir Crit Care Med. 1995;152: 129-36.
18. Parthasarathy S, Jubran A, Tobin MJ. Cycling of inspiratory and expiratory muscle groups with the ventilator in airflow limitation. Am J Respir Crit Care Med. 1998;158:1471.
19. Tassaux D, Gainnier M, Battisti A, Jolliet P. Impact of Expiration "trigger Setting on delayed Cycling and Inspiratory muscles Workload". Am J Respir Crit Care Med 2005;172;:1283-9.
20. Jolliet P, Tassaux D. Clinical review: patient-ventilator interaction in chronic obstructive pulmonary disease. Crit Care. 2006;10:236.
21. Kondili E, Xirouchaki N, Georgopoulos D. Modulation and treatment of patient-ventilator dyssynchrony. Curr Opin Crit Care. 2007;13:84-9.
22. Prinianakis G, Kondili E, Georgopoulos D. Patient-Ventilator Interaction: An Overview. Respir Care Clin N Am. 2005;11:201-24.
23. Thille AW, Brochard L. Promoting patient-ventilator synchrony. Clin Pulm Med. 2007;14:350-9.
24. Georgopoulos D, Prinianakis G, Kondili E. Bedside waveforms interpretation as a tool to identify patient-ventilator asynchrony. Intensive Care Med. 2006;32:34-47.
25. Nilsestuen JO, Hargett KD. Using ventilator graphics to identify patient-ventilator asynchrony. Respir Care. 2005;50:202-32.
26. Chiumello D, Pelosi P, Taccone P, Slutsky A, Gattinoni L. Effect of different inspiratory rise time and cycling off criteria during pressure support ventilation in patients recovery from acute lung injury. Crit Care Med. 2003:;31: 2604-10.
27. Fernández R, Méndez M, Younes M. Effect of ventilator flow rate on respiratory timing in normal humans. Am J Respir Crit Care Med. 1999;159:710-9.
28. Herrero Huertas J, Laso Del Hierro F, Peláez Castro F et al. The Importance of Highly Specific Management in Prolonged Weaning. Arch Bronconeumol (Engl Ed). 2019;55:443-4.
29. Tokioka H, Tanaka T, Ishizu T et al. The effect of breath termination criterion on breathing patterns and the work of breathing during pressure support ventilation. Anesth Analg. 2001;92:161-5.
30. Tassaux D, Michotte JB, Gainnier M, Gratadour P, Fonseca S, Jolliet P. Expiratory trigger setting in pressure support ventilation: from mathematical model to bedside. Crit Care Med. 2004;32:1844-50.
31. Calderini E, Confalonieri M, Puccio PG, Francavilla N, Stella L, Gregoretti C. Patient-ventilator asynchronous during noninvasive ventilation: the role of expiratory trigger. Intensive Care Med. 1999;25:662-7.
32. Nava S, Ceriana P. Patient-ventilator interaction during noninvasive positive pressure ventilation. Respir Care Clin. 2005;11:281-93.
33. Elliott MW. The interface: crucial for successful noninvasive ventilation. Eur Respir J. 2004;23:7-8.
34. Murgu SD, Pecson J, Colt HG. Bronchoscopy During non-invasive Ventilation: Indications and Technique. Respir Care. 2010;55:595–600.

35. Saatci E, Miller DM, Stell IM, Lee KC, Moxham J. Dynamic dead space in face masks used with noninvasive ventilators: a lung model study. Eur Respir J. 2004;23:129-35.
36. Hess DR. Patient-ventilator interaction during noninvasive ventilation. Respir Care. 2011;56:153-65.
37. Lellouche F, Maggiore SM , Deye N , Taillé S , Pigeot J , Harf A , Brochard L. Effect of the humidification device on the work of breathing during non-invasive ventilation. Intensive Care Med. 2002;28:1582-9.
38. Cousins TR, O'Donnell JM. Arterial cannulation: a critical review. AANA J. 2004;72:267-71.
39. Shiver S, Blaivas M, Lyon M. A prospective comparison of ultrasound-guided and blindly placed radial arterial catheters. Acad Emerg Med. 2006;13: 1275-9.
40. Lamia B, Chemla D, Richard C, Teboul JL. Clinical review: interpretation of arterial pressure wave in shock states. Crit Care. 2005;9:601-6.
41. Günther SC, Schwebel C, Hamidfar-Roy R et al. Complications of intravascular catheters in ICU: definitions, incidence and severity. A randomized controlled trial comparing usual transparent dressings versus new-generation dressings (the ADVANCED study) Intensive Care Med. 2016; 42:1753–65.
42. Timsit J-F, Rupp M, Bouza E et al. A state of the art review on optimal practices to prevent, recognize, and manage complications associated with intravascular devices in the critically ill. Intensive Care Med. 2018;44:742–59.
43. van der Kooi T, Sax H, Pittet D et al. Prevention of hospital infections by intervention and training (PROHIBIT): results of a pan-European cluster-randomized multicentre study to reduce central venous catheter-related bloodstream infections. Intensive Care Med. 2018;44:48–60.
44. Frades H, Béatrice S, Peces-Barba Romero G. Ecografia Pleuro-Pulmonar Em Cuidados Intermédios Respiratórios. Editorial: Ergon Creación. 2014. ISBN: 978-84-15950-52-3.
45. John R. Richards, John P. McGahan. Focused Assessment with Sonography in Trauma (FAST) in 2017: What Radiologists Can Learn. Radiology. 2017; 283:30-48.
46. Broder N, Zolty R. Pulmonary Arterial Hypertension in the ICU. In: Oropello JM, Pastores SM, Kvetan V. eds. Critical Care. McGraw Hill. Accessed September 03, 2022. https://accessmedicine.mhmedical.com/content.aspx?bookid=1944&sectionid=143517315
47. Thille AW, Wairy M, Le Pape S, Frat JP. Oxygenation strategies after extubation of critically ill and postoperative patients, J Inten Med. 2021;1:65-70.
48. Devlin JW, Skrobik Y, Gélinas C et al. Clinical practice guidelines for the prevention and management of pain, agitation/sedation, delirium, immobility, and sleep disruption in adult patients in the ICU. Crit Care Med. 2018; 46:e825-73.
49. Davies L, Mister R, Spence DP et al. Cardiovascular consequences of fibreoptic bronchoscopy. Eur Respir J. 1997;10:695–8.
50. Dweik RA, Mehta AC, Meeker DP et al. Analysis of the safety of bronchoscopy after recent acute myocardial infarction. Chest. 1996;110: 825–8.
51. Facciolongo N, Patelli M, Gasparini S et al. Incidence of complications in bronchoscopy. Multicentre prospective study of 20,986 bronchoscopies. Monaldi Arch Chest Dis. 2009;71:8–14.