CASE REPORT

Heart lung interactions during recruitment maneuvers

– Alveolar and cardiac benefits

Flow Anesthesia | Recruitment maneuvers

Intraoperative ventilatory management in an obese patient

— A case report

As caregivers, you intend to restore or preserve a patient's health, and you also do your best to avoid possible complications related to the treatment.

Whenever patients are anesthetized, there is a risk for postoperative complications caused by a collapse of the alveoli. Far from being only a short-term side effect, anesthesia induced atelectasis has been shown to persist in patients' lungs long after they leave the operating room.[1]

Potential postoperative complications:[2]

- hypoxemia
- pneumonia
- local inflammatory response
- ventilator induced lung injury (VILI)

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Background

According to the literature, a significant number of patients develop atelectasis during the intraoperative period. Atelectasis can be induced by general anaesthesia, by the supine position and, in the case of laparoscopic procedures, by pneumoperitoneum. Atelectasis, a pulmonary stress factor that can cause ventilator-induced lung injury (VILI), is therefore a risk factor for the development of postoperative pulmonary complications (PPC). Alveolar collapse reduces lung compliance, and the resulting increase in driving pressure (DP) is an independent predictor of PPCs. Different ventilatory strategies during the lung protective ventilation stage have been proposed to reduce PPCs. In the individualized open-lung approach (iOLA), alveolar recruitment maneuvers (ARM) are performed to open collapsed alveolar units, and then positive end-expiratory pressure (PEEP) is adjusted individually to prevent their re-collapse *(see Figure 1)*. When successful, iOLA increases the percentage of functional lung volume and minimizes the pulmonary stress and strain that is known to trigger local and systemic inflammatory response.

Heart lung interactions

It is essential to understand the physiological impact of positive pressure on hemodynamics, and more specifically, during iOLA. Heart-lung interactions refer to the effect that changes in intrathoracic pressures and lung volumes have on the heart and blood circulation. In the context of hemodynamic monitoring, understanding this phenomenon can enable the clinician to predict, within certain limits, a patient's response to supportive treatment such as positive pressure mechanical ventilation (MV), fluid replacement, or the administration of vasoactive drugs. It is important to identify this interaction in patients under mechanical ventilation, since venous return and ventricular

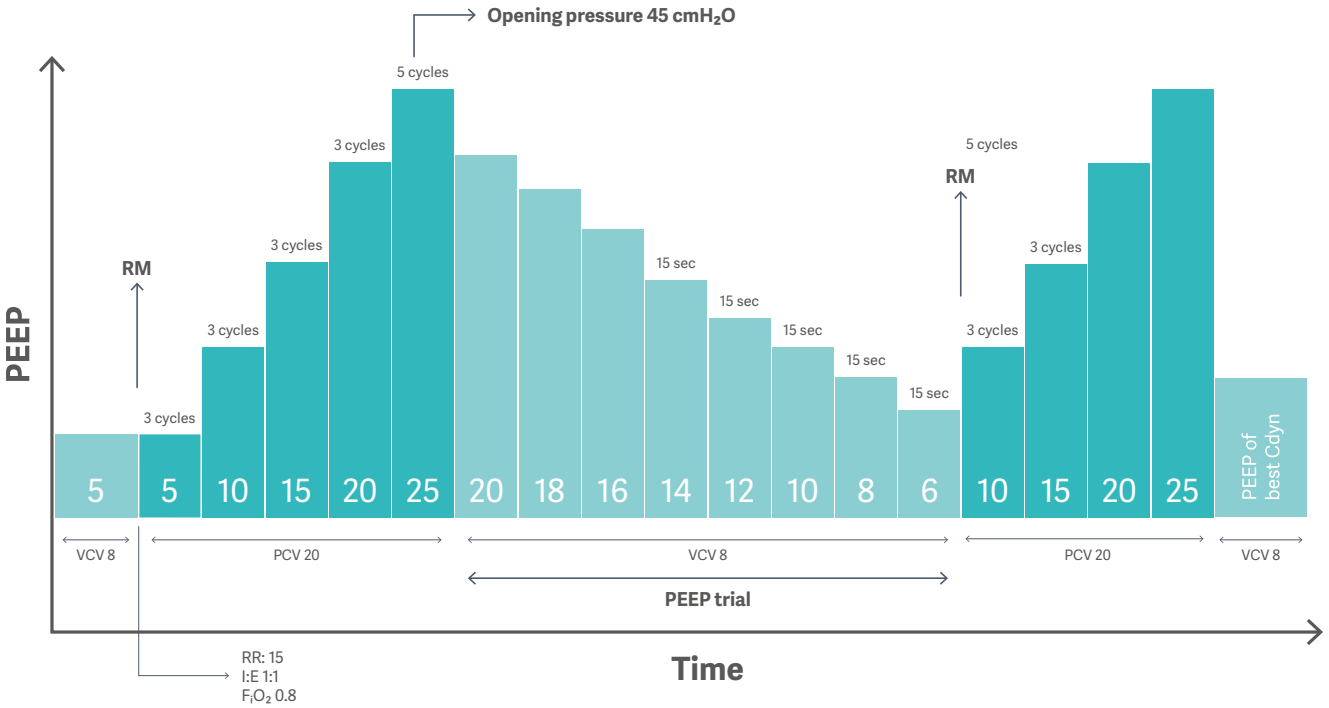


Figure 1. Stepwise alveolar recruitment maneuvers (ARM) performed according to i-OLA process.

"The effects of mechanical ventilation are in turn influenced by pulmonary mechanics and the patient's underlying circulatory status."†

preload and afterload dynamics are influenced by positive pressure ventilation. The effects of mechanical ventilation are in turn influenced by pulmonary mechanics and the patient's underlying circulatory status *(see Figure 2)*. Positive pressure mechanical ventilation can have different cardiovascular effects in each patient; for example, in a healthy patient with good cardiac function, the change from spontaneous to mechanical ventilation will hardly cause hemodynamic changes. However, in hypovolaemic patients and in patients with right heart failure of cardiac or pulmonary origin, the transition from one type of ventilation to another can lead to low cardiac output (CO).

Case report

Medical History

We present the case of a 61-year-old patient with no known drug allergies who was scheduled for gastric bypass surgery. Her personal history was significant for: ex-smoker, prediabetes, untreated high blood pressure, hepatic steatosis, hypothyroidism, type 3 obesity with a body mass index (BMI) of 47.45 kg/m² (weight 114 kg, height 155 cm) and obstructive sleep apnea syndrome (OSAS) treated night time continuous positive airway pressure (CPAP). Additional studies of interest: transthoracic echocardiography (TTE) that showed normal, non-dilated heart chambers, with mild left ventricular hypertrophy, good biventricular function, and absence of significant valvular disease, except for mild tricuspid regurgitation that prevents the estimation of pulmonary pressures.

General anesthetic management

The patient arrived in the operating room hemodynamically stable with baseline S_pO_2 of 95%. Standard monitoring (HR, non-invasive blood pressure, ECG) + bispectral index (BIS) + and Tofcuff (RGB Medical Devices) was performed. Premedication: fentanyl 50 mcg. Facemask pre-oxygenation was performed for 3 minutes under spontaneous ventilation to reach $FetO_2$ >90%. Anesthesia was induced with iv propofol 80 mg + fentanyl 150 mcg + lidocaine 80 mg + rocuronium 60 mg. The patient was ventilated with a facemask + oropharyngeal airway for 2 minutes until twitches were absent on train of four and BIS was <50.

Next, we inserted a Glidescope video laryngoscope to guide intubation with a 7.5 mm endotracheal tube, which was then connected to the anesthesia machine.

Hemodynamic monitoring

Left radial artery cannulation for invasive arterial pressure monitoring, hemodynamic monitoring via pulse contour analysis on a Pulsioflex monitor (Getinge), and measurement of arterial blood gas. Cardiac function was monitored with transesophageal echocardiography (TTE).

Ventilatory management

Tidal volume (VT) 6 to 8 ml/kg ideal weight, respiratory rate (RR) for $EtCO_2$ between 35 and 45 mmHg, minimum FiO_2 to achieve SpO_2 >90% and PEEP of 5 cmH_2O.

iOLA

Alveolar recruitment maneuvers (ARM)

Switch to pressure controlled ventilation (PCV) with a driving pressure of 20 cmH_2O and an RR of 15 rpm, I: E ratio 1: 1, FiO_2 0.8 and PEEP 5 cmH_2O.

During the recruitment phase, PEEP was increased by increments of 5 cmH_2O every 3 respiratory cycles to a PEEP of 25 cmH_2O, achieving an alveolar opening pressure of 45 cmH_2O for 5 respiratory cycles (total maneuver time: 68 seconds) *(see Figure 1)*. In our case, the hemodynamics shown by the Pulsioflex monitor showed stability before and during the ARM not requiring optimization with drugs or fluids.

Decremental PEEP titration trial

After the ARM, the PEEP titration trial phase was started. Ventilation was returned to volume controlled ventilation (VCV) with the same pre-ARM settings except for PEEP, which was set to 20 cmH_2O. During the PEEP trial, PEEP was decreased in decrements of 2 cmH_2O every 15 seconds until observing the highest dynamic compliance, Cdyn (when Cdyn starts to decrease or no longer increases). When same Cdyn is observed at several PEEP levels, the level that generates the lowest driving pressure (Pplat-PEEP) is chosen. Once optimal Cdyn has been determined, ARM is repeated and PEEP is adjusted to the optimal Cdyn *(see Figure 1)*.

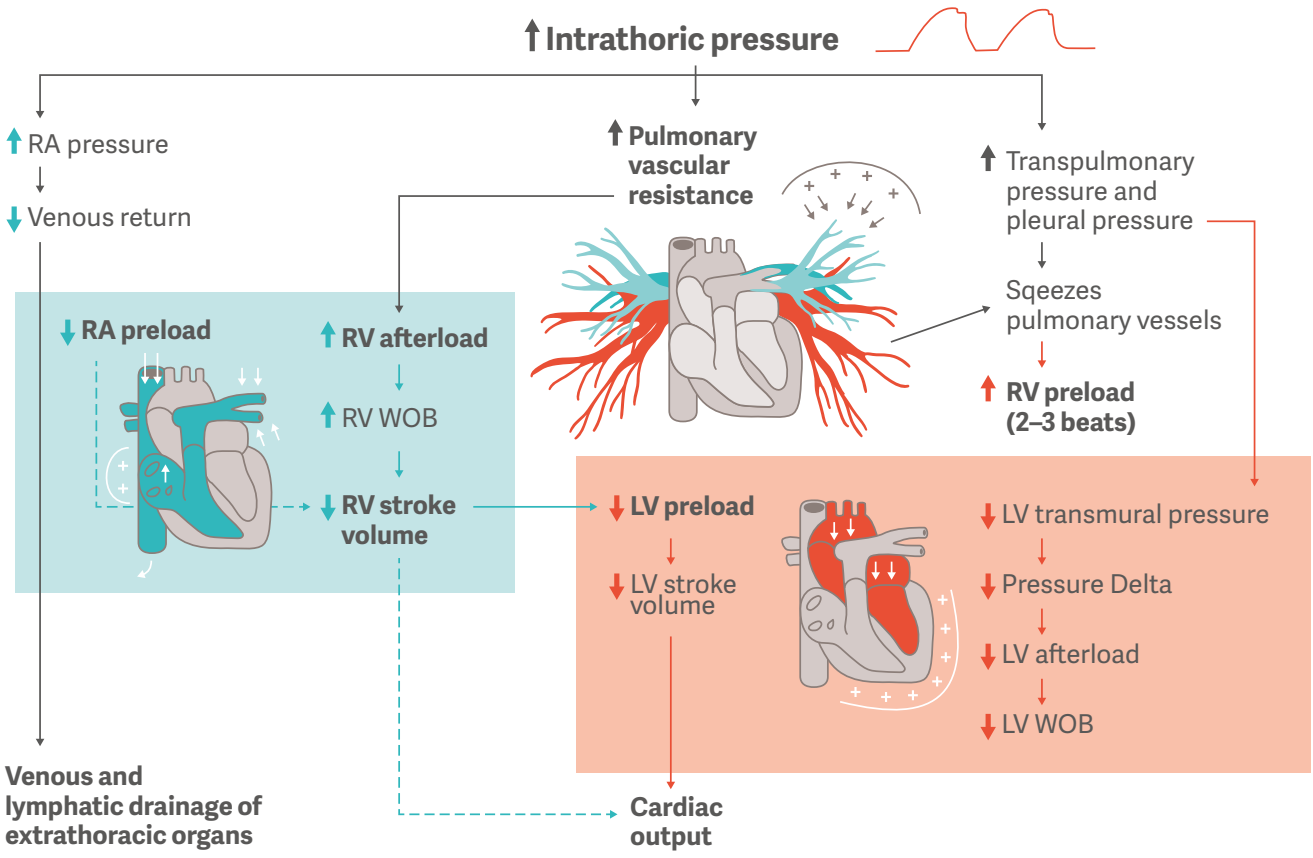


Figure 2. Effects of mechanical ventilation on pulmonary mechanics and the patients' circulatory system

Perioperative measurements

Table 1 shows the respiratory, hemodynamic, blood gas, and TTE results obtained during surgery.

Variables	Baseline	Pre- iOLA	Post-iOLA
Respiratory			
VT (ml)	500	500	500
RR, bpm	12	12	12
PEEP, cmH_2O	5	5	15
FiO_2	0.62	0.57	0.61
SpO_2, (%)	98%	98%	98%
Ppeak, cmH_2O	27	26	30
Plateau pressure, cmH_2O	22	21	25
Cdyn, ml/cmH_2O	29	33	54
Driving pressure, cmH_2O	17	16	10
$EtCO_2$ mmHg	39	41	36
Hemodynamics			
Mean arterial pressure, mmHg	67	66	86
Heart rate	70	75	61
Cardiac Index, $ml/kg/m^2$	1.7	2.2	2
Pulse pressure variation, %	54	11	11
SVRI (dyn x sec x m^2/cm^5)	2741	2352	1974
Cardiac Power index (CPI) (W/m^2)	0.36	0.7	0.5
Blood gas analysis			
pH	7.37	7.35	7,.37
$PaCO_2$, mmHg	46.9	43.1	43
PaO_2, mmHg	226.4	194.6	297
PaO_2/FiO_2	365	341	487
Lactate	13.6	12.3	11.1
Cardiac Power index (CPI) (W/m^2)	0.36	0.7	0.5
Echocardiography			
Right ventricle diameters (VD) – Basal – Medium – Longitudinal	 3.90 3.92 7.45	 3.83 3.89 7.52	 3.92 3.43 7.88
RV- FAC (%)	38%	37%	44%
m-TAPSE, cm	1.48	1.29	1.89
S' wave, cm/seg	9.09	9.30	10.2
RVOT VTI, cm	12.5	11.5	14
LVOT VTI, cm	19.8	23	24.3

SVRI: systemic vascular resistance index; RV-FAC: right ventricle fractional area change; m-TAPSE: modified tricuspid annular plane systolic excursion; S' wave: tissue peak systolic wave velocity (measured on right ventricle free wall at the level of annulus tricuspid insertion); RVOT VTI: Right ventricular outflow tract velocity time integral; LVOT VTI: left ventricular outflow tract velocity time integral.

Intraoperative individualized heart lung optimization

This case shows that applying iOLA with minimaly invasive hemodynamic monitoring is a safe strategy that improved both, oxygenation and ventilatory mechanics, resulting in higher Cdyn and lower driving pressure.

Discussion

In terms of hemodynamics, iOLA increased the cardiac index (CI) and mean arterial pressure (MAP). Until relatively recently, anesthesiologists have used elevated VT and low, or even zero, PEEP, particularly in obese and/or shorter than average patients. Today, we know that settings like this may lead to ventilator induced lung injury.[3–4] Various studies have shown that atelectasis is an independent risk factor for PPCs.[5] The risk of intraoperative atelectasis is higher in abdominal surgery, particularly laparoscopic surgery with pneumoperitoneum, and in obese patients, due to the need to increase intra-abdominal pressure. Several physiological studies have shown that the implementation of an iOLA (ARM + iPEEP) improves gas exchange (oxygenation, by reducing shunt and eliminating CO_2 by reducing dead space) and increases functional lung volume, which in turn increases lung compliance and decreases driving pressure.[6–13]

From a hemodynamic perspective, atelectasis, which can range from moderate local hypoventilation to complete atelectasis and pulmonary collapse, triggers hypoxic pulmonary vasoconstriction, a physiological reflex that promotes alveolar capillary collapse. This, together with atelectasis-induced changes in the geometry of pulmonary capillaries, leads to an increase in pulmonary arterial pressure that can increase impedance (afterload) in the right ventricular outflow tract and the risk of right ventricular failure.[14–15] Right ventricular failure is associated with extubation failure and increased morbidity and post-operative complications. In addition, right-sided heart changes will reduce left preload, leading to a corresponding decrease in cardiac output.[16]

In our patient, obesity, severe obstructive sleep apnea and/or OHS, and abdominal surgery, were factors that considerably increased the risk of developing these hemodynamic changes, due to the high prevalence of pulmonary hypertension and intraoperative atelectasis in patients with these co-morbidities. The recruitment maneuvers performed in the iOLA strategy, however, reopened collapsed alveolar capillaries and reduced right ventricle afterload, thus improving right ventricular contractility parameters (right ventricular fractional shortening, modified tricuspid annular plane systolic excursion, right ventricular outflow tract velocity time integral, and S' wave or tricuspid annular systolic velocity). Mechanical ventilation itself, and iOLA in particular, helped reduce both left ventricular transmural pressure and left ventricular afterload by increasing intrathoracic pressures, and also increased both stroke volume and cardiac index. In this way, individualized ventilatory management together with advanced hemodynamic monitoring based on arterial pulse wave and echocardiography allowed us to optimize intraoperative fluid therapy management and the use of vasoactive drugs, thereby reducing the risk of post-operative complications.

Finally, individualizing PEEP will minimize the risk of selecting an inappropriate PEEP level after ARM. Setting PEEP below optimum levels increases the risk of returning to the baseline situation of alveolar collapse. Excessively high PEEP will lead to alveolar distension. This will not only increase the risk of VILI, but also cause the collapse of the alveolar capillaries, leading to hemodynamic instability that must be corrected by administering fluids or vasoactive agents.

Conclusion

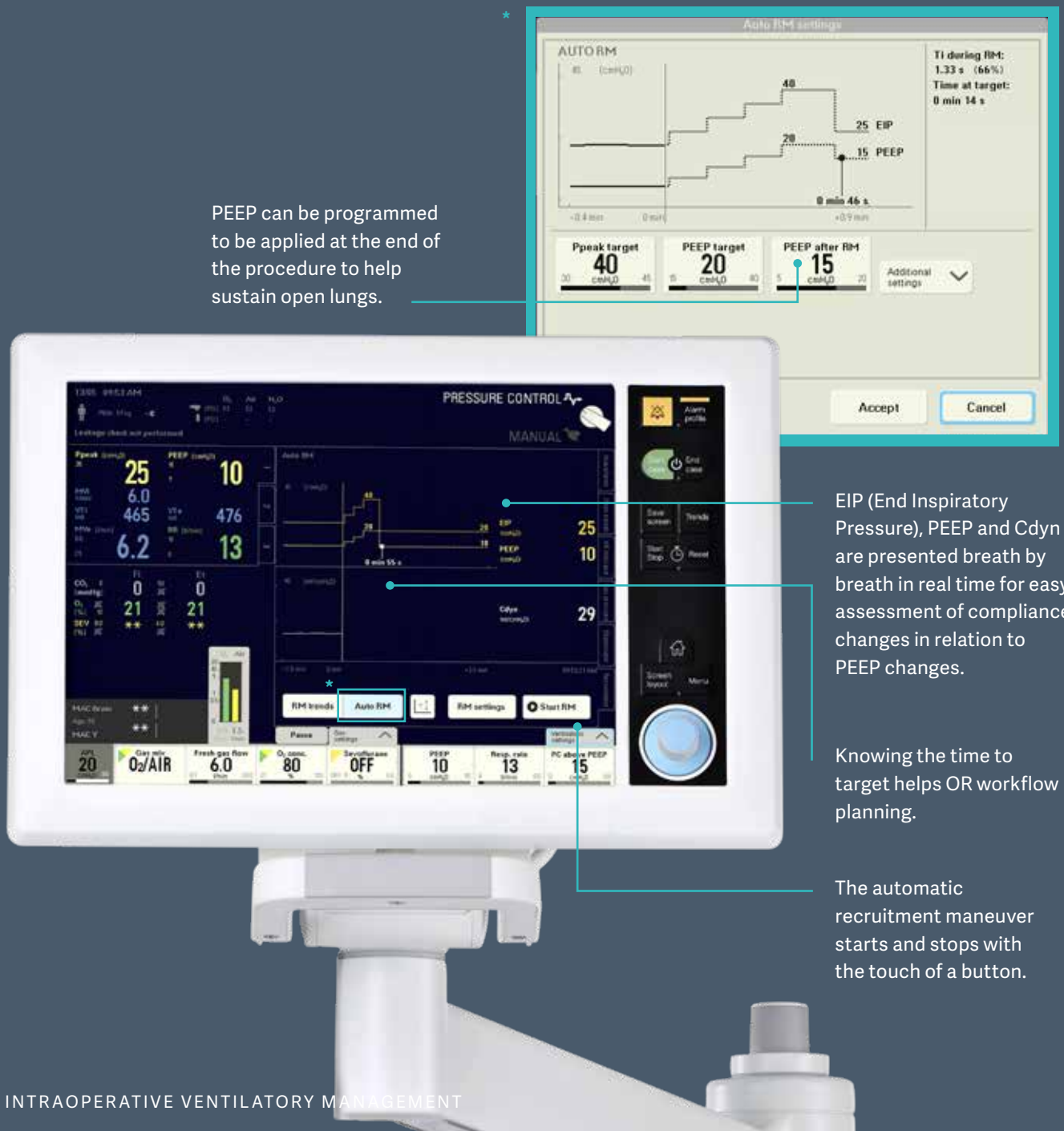
Applying iOLA during the intraoperative period improved both oxygenation parameters and pulmonary mechanics. It also improved the patient's hemodynamics status, as evidenced by an improvement in cardiac output and stroke volume in continuous monitoring and in TTE-derived ventricular function parameters.

Automatic and stepwise lung recruitment

Lung recruitment with Getinge's anesthesia system (Flow-i and Flow-e) allows you to choose between an automatic or manual maneuver. Whichever you choose, it's designed for stepwise recruitment. This feature aims to gently open the alveoli to make a lasting difference — for you and for your patients.

In the automatic recruitment maneuver (RM), a stepwise increase in pressure is applied for a time period set by the user. It's designed to reduce the occurrence of hemodynamic compromise. Getinge's anesthesia system (Flow-i and Flow-e) measures and displays the dynamic compliance in real time, which is used to find the optimal lowest PEEP that keeps the lungs open.

With RM trends, you can tailor the settings for your individual patient and can perform lung recruitment manually.



PEEP can be programmed to be applied at the end of the procedure to help sustain open lungs.

EIP (End Inspiratory Pressure), PEEP and Cdyn are presented breath by breath in real time for easy assessment of compliance changes in relation to PEEP changes.

Knowing the time to target helps OR workflow planning.

The automatic recruitment maneuver starts and stops with the touch of a button.

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