EVALUATION OF EFFECT OF ALVEOLAR RECRUITMENT MANEUVER DURING INTRAOPERATIVE MECHANICAL VENTILATION

DISSERTATION SUBMITTED FOR

DOCTOR OF MEDICINE

BRANCH X (ANAESTHESIOLOGY)

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CHENNAI.

BONAFIDE CERTIFICATE

This is to certify that this dissertation titled **"EVALUATION OF EFFECT OF ALVEOLAR RECRUITMENT MANEUVER DURING INTRAOPERATIVE MECHANICAL VENTILATION"** is a bonafide record work done by **Dr. RAMA RANI K.** under my direct supervision and guidance, submitted to the Tamil Nadu Dr. M.G.R. Medical University in partial fulfillment of University regulation for MD, Branch X–Anaesthesiology.

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DECLARATION

I, **Dr. RAMA RANI K.,** solemnly declare that this dissertation titled **"EVALUATION OF EFFECT OF ALVEOLAR RECRUITMENT MANEUVER DURING INTRAOPERATIVE MECHANICAL VENTILATION"** has been done by me. I also declare that this bonafide work or a part of this work was not submitted by me or any other for any award, degree, diploma to any other University or board either in India or abroad.

This is submitted to The Tamil Nadu Dr. M. G. R. Medical University, Chennai in partial fulfillment of the rules and regulation for the award of Doctor of Medicine degree Branch X–Anaesthesiology to be held in April 2013.

Place: Madurai **Dr. RAMA RANI K.**

Date:

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INTRODUCTION

At the beginning of the last century, Pasteur described postoperative pulmonary atelectasis, analyzed postoperative pulmonary complications and noted: `when the true history of postoperative lung complications comes to be written, active collapse of the lung, from deficiency of inspiratory power, will be found to occupy an important position among determining causes'. (1; 2; 3). In fact, development of atelectasis begins with induction of general anesthesia and continues into the postoperative period. This contributes to significant complications and additional healthcare costs. (1)

Pulmonary gas exchange is affected in general anesthesia. This is due to increased ventilation perfusion mismatch that occurs even in healthy individual. This increase in intrapulmonary shunt is mainly caused by formation of atelectasis during general anesthesia. Within minutes of the induction of general anesthesia, alveolar collapse occurs in most of the patients, even with healthy lungs. (4) (5) (6) Atelectasis commonly develops in the most dependent parts of the lungs. (1) Atelectasis develops irrespective

of the mode of induction, intravenous or inhalational anesthesia. Once induced with anesthetics, they develop in spontaneously breathing individual as well as in mechanically ventilated individual paralyzed with muscle relaxants. (1) (7)

Studies have proved that ketamine, is the only anesthetic agent that does not produce atelectasis, provided that no muscle relaxants have been used. (1) (8) On a CT cut near the diaphragm the area with atelectasis is around 5% to 6% of the total lung area. But, the lung tissue with regions of atelectasis can easily exceed 15% to 20%. The amount of tissue that is collapsed is even larger, the atelectatic area consisting of mainly lung tissue and the aerated lung consisting of only 20% to 40% tissue, the rest being air. Thus, 15% to 20% of the lung is regularly collapsed at the base of the lung during uneventful anesthesia, before any surgery has even been done! The amount of atelectasis decreases toward the apex, which is mostly spared (fully aerated). (9)

AIM OF THE STUDY

The study was undertaken with the aim to **evaluate the effect of alveolar recruitment maneuver on intra operative arterial oxygenation in patients undergoing laparotomies under general anaesthesia.**

MECHANISM OF ATELECTASIS

The causes of atelectasis can be explained by a variety of factors. Three different but interrelated mechanisms have been implicated in the formation of atelectasis. A reduction in the Trans mural pressure that keeps the alveoli open, results in compression atelectasis. When the uptake of gas by blood exceeds its entry into the alveolus, absorption atelectasis results. When the surfactant decreases, surface tension within the alveolus increases resulting in loss-of-surfactant atelectasis. Hence, atelectasis during anesthesia and the postoperative period may be caused by any of these factors. (1)

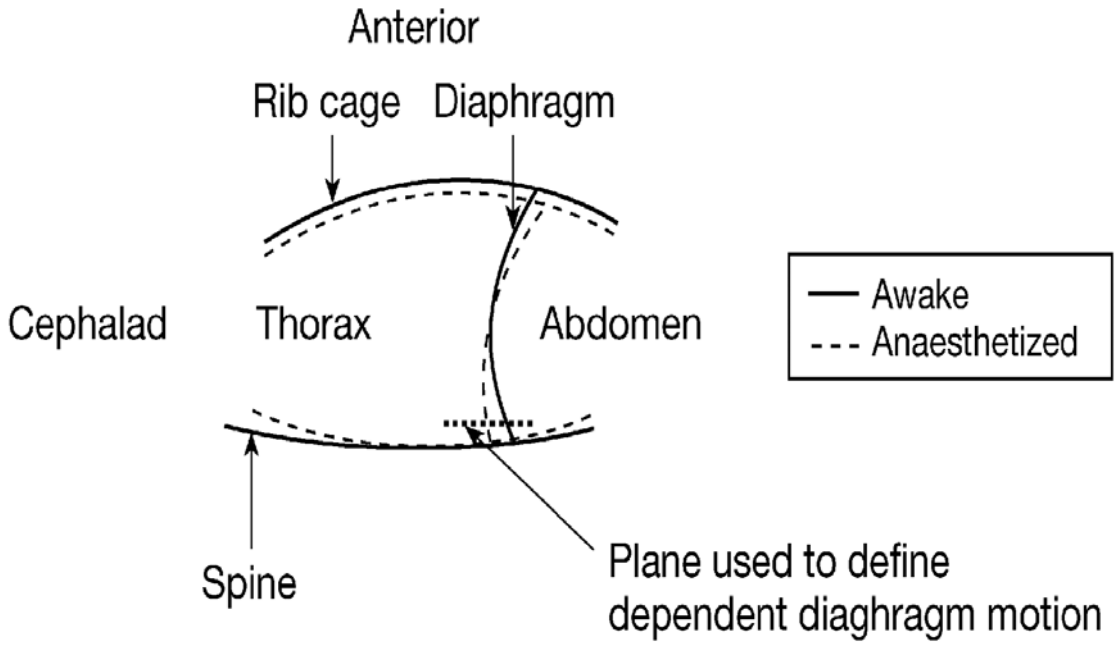
Compression atelectasis - normally there is some amount of pressure within the alveolus that keeps it open. A drop in this pressure to an extent to cause alveolar collapse results in compression atelectasis. Loss of inspiratory muscle tone with compression of lung tissue is an important factor in atelectasis formation. Normally, the thorax and abdomen is divided into two separate cavities by the diaphragm. In an awake spontaneously breathing individual, it creates different pressures in each cavity.

But, this effect is lost with loss of muscle tone on induction with anesthetic agents. The diaphragm moves upwards especially in the dorsal dependent areas. This leads to conduction of pressure across the diaphragm, often to such an extent that it causes compression of the adjacent lung tissue. This is termed compression atelectasis. (10)

Several lines of evidence support a role of the diaphragm in this setting. The classic study by Froese and Bryan showed in spontaneously breathing normal volunteers, diaphragmatic motion is altered when the volunteers are paralyzed with muscle relaxants. (1) (11). The authors concluded that while the dorsal areas of the diaphragm had the greatest cephalad shift in the supine position in spontaneously breathing individual, the non-dependent part of the diaphragm was shifted maximally after muscle paralysis and positive pressure ventilation. Krayer and colleagues also made similar observations. Alterations in the movement of the diaphragm with anesthesia were confirmed by CT scans. (1) (12). When the diaphragm becomes lax or paralyzed, as during anesthesia, the greater intra-abdominal pressure is conducted more easily into the thoracic cavity.

The intercostal muscle tone also helps in maintaining functional residual capacity. Hence, the use of muscle relaxants during anesthesia leads to fall in functional residual capacity. Inhalational anesthetic agents also contribute to the reduction in functional residual capacity by decreasing the intercostal muscle activity, especially in the pediatric age group. (10)

Positive pressure ventilation leads to pooling of intravascular blood in the abdominal cavity. This adds to the diaphragmatic compression of the basal lung tissues. This was observed by Hedenstierna et al in his study. (10)



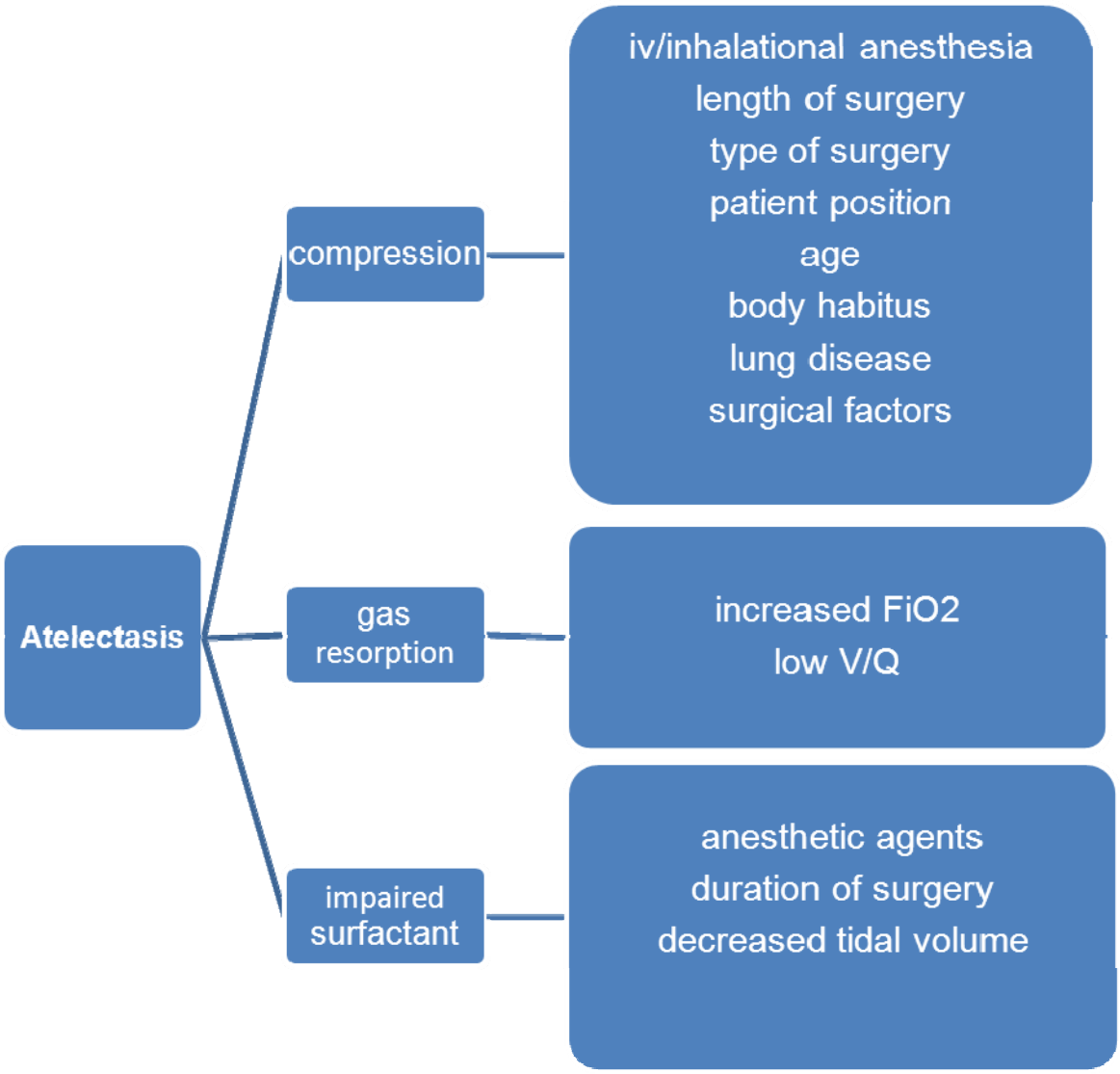
Cranial shift of the diaphragm and a decrease in transverse diameter of the thorax contribute to lowered functional residual capacity during anesthesia. Decreased ventilated volume (atelectasis and airway closure) is a possible cause of reduced lung compliance. Decreased airway dimensions by the lowered functional residual capacity should contribute to increased airway resistance. CT based evidence of formation of atelectasis on induction of anesthesia, with the role of positive end expiratory pressure in distending the alveoli suggested that the atelectasis was due to compression of lung tissue.

Absorption atelectasis- Absorption atelectasis is sometimes called gas atelectasis. Complete airway occlusion causes gas trapping in the lung tissues distal to the point of occlusion. No further ventilation occurs at these sites but perfusion continues to occur. Passive diffusion of gases continues to occur from these alveoli into the mixed venous blood perfusing these areas due to the pressure gradient that exists between them. The alveolar pressure, initially close to atmospheric pressure, gradually drops with continued uptake of gas into the circulation, finally resulting in alveolar collapse.

The rate of absorption of gas, hence the rapidity of formation of atelectasis from such lung areas also depends on the fraction of inspired oxygen (FIO_2). (1; 15)Critical V_A/Q ratio is a point of balance between the rate of entry of gas into the alveolus and its uptake into the pulmonary circulation. When this balance is affected such that gas uptake into the blood is higher than the inspired gas, or in other words when this ratio falls below the critical value, the alveoli will collapse. (1; 16)This shows that alveolar collapse is dependent on gas uptake which in turn depends

on fraction of inspired oxygen. So, higher the FiO_2 more is the alveolar collapse. (1)

Loss-of-surfactant atelectasis - surfactant acts on the alveolar surface to reduce its surface tension stabilize the alveoli, hence prevent its collapse. Anesthesia depresses the alveolar stabilizing property of the surfactant leading to alveolar collapse. (10)A vicious cycle develops with atelectasis leading to further decrease in the surfactant function. Various methods have been tried to increase the surfactant, maintain its distribution, and thereby preserve its protective action. Some of these methods are to increase the tidal volume sequentially to total lung capacity, hyperventilation, etc. (10; 17; 18)


Atelectasis
compression
gas resorption
impaired surfactant
iv/inhalational anesthesia
length of surgery
type of surgery
patient position
age
body habitus
lung disease
surgical factors
increased FiO2
low V/Q
anesthetic agents
duration of surgery
decreased tidal volume

Other Factors Modulating the Formation of Atelectasis

AGE–Beyond infancy, atelectasis is independent of age, with children and young people showing as much atelectasis as elderly patient (19)

BODY MASS INDEX-Patients with greater BMI develop larger areas of atelectasis than those with low or normal BMI. There is increased closing volume particularly affected by the position of the patient as supine or Trendelenberg resulting in decreased functional residual capacity. Similarly, the reduced functional residual capacity that occurs during pregnancy also potentiates atelectasis (19)

PREEXISTING LUNG DISEASE- Atelectasis does not affect much smokers and patients with lung disease particularly chronic obstructive lung disease. Although these patients have an impaired gas exchange even in the awake state, this is mainly due to ventilation perfusion mismatch. Chronic air trapping, destruction of lung tissues and enlargement of terminal and respiratory

bronchioles along with pulmonary vasoconstriction is the cause for ventilation perfusion mismatch. Air trapping in the distal lung units prevents their collapse. The mechanical properties of the lungs and its interaction with the chest wall change to produce a high compliance state. However, atelectasis can develop late in the disease process in patients with chronic obstructive lung disease due to resorption in the low V_A/Q units. (10)

EFFECTS OF POSITION-Functional residual capacity is known to reduce by 0.5 to 1litre even in healthy adults by change in the position from in upright to supine.(18)An additional decrease of 0.5-0.7 litre is observed in anesthetized individual. Nearly 15-30 degree of head down tilt aggravates the fall in the functional residual capacity by increasing the cephalad shift of the diaphragm. Changes have been observed in the dependent and non-dependent lung in lateral decubitus position. Functional residual capacity is more in the non-dependent lung than in the dependent lung which has more compressed lung units. However, the total functional residual capacity increases minimally irrespective of the size of the dependent lung. In the prone position, proper positioning of the

anesthetized patient so as to avoid abdominal compression, allows a more uniform distribution of ventilation leading to an increase in the functional residual capacity. But atelectasis does not decrease in the prone position. (10)

TYPE OF ANESTHESIA- Both intravenous and inhalational modes of induction are capable of producing atelectasis. This occurs in patients who are on spontaneous or mechanical ventilation with or without paralysis. (1; 7; 10) An exception to this rule is ketamine, in the absence of neuromuscular blockade as it maintains muscle tone. (1; 8; 10)

Regional anesthesia also affects ventilation, reducing inspiratory capacity and expiratory reserve volume. The extent of these changes depends on the type of regional technique used and also on the level of motor blockade. Higher the blockade more is the changes. However, neuraxial blockade commonly produces minimal changes in pulmonary gas exchange, maintaining arterial saturation well within normal limits. Closing capacity and functional residual capacity are unaffected. (10)

IMPACT OF TIME- atelectasis occurs within minutes of induction of general anesthesia with maximum decrease in functional residual capacity.

SURGICAL FACTORS- surgical manipulation as in surgical packing, tissue retraction in thoracic and abdominal surgeries is associated with a higher incidence of atelectasis. Peripheral surgeries as in surgeries of the limb have lesser areas of atelectasis.

TIDAL VOLUME- Use of low tidal volumes in the absence of positive end expiratory pressure or alveolar recruitment strategies may result in significant atelectasis.

EFFECTS OF ATELECTASIS

Several pathophysiologic effects have been observed with atelectasis:

i) DECREASED COMPLIANCE- there occurs a decrease in pulmonary compliance and a consequent increase in work of breathing associated with worsening of systemic oxygenation with atelectasis. The increased work of breathing in decreased compliance is because of the increased trans pulmonary pressure required to achieve a given tidal volume.(10)

ii) IMPAIRED OXYGENATION- atelectasis increases V_A/Q mismatch with low V_A/Q ratios, wherein perfusion exceeds alveolar ventilation. This results in impairment of oxygenation.

iii) INCREASED PULMONARY VASCULAR RESISTANCE- pulmonary vascular resistance is minimal at functional residual capacity. When atelectasis develops with areas of regional hypoxia there is a decrease in alveolar and mixed venous oxygen tension. This results in hypoxic pulmonary vasoconstriction. Impairment in the right ventricular function and increase in the

micro vascular leakage has been observed as a result of increased pulmonary vascular resistance.(10; 21; 22)

iv) LUNG INJURY- high lung volumes cause volutrauma; low lung volumes by means of repetitive small airway closure results in lung injury. Atelectasis potentiates lung injury by causing release of inflammatory mediators. In the absence of positive end expiratory pressure and impaired lung compliance, an increase in the serum cytokine concentrations (Tumor necrosis factor – alpha, interleukin -1) has been observed. Shearing stresses caused by repetitive airway closure can be minimized by preventing atelectasis.(10)

PREVENTION OF ATELECTASIS DURING ANESTHESIA

Several interventions can help prevent atelectasis or even reopen collapsed tissue, as discussed in the following paragraphs.

POSITIVE END-EXPIRATORY PRESSURE

Application of PEEP has been tried in various studies to increase arterial oxygenation by opening collapsed lung tissue. This is more often due to increased airway pressures associated with positive end expiratory pressure. But, some atelectasis persists in most patients. However, on discontinuation of positive end expiratory pressure the alveoli recollapse immediately. Within 1 minute after cessation of positive end expiratory pressure, the collapse is as large as it was before the application of positive end expiratory pressure. (9)(19)(23) .However, in a study conducted in patients with acute lung injury it was shown that recruitment strategies opened collapsed alveoli and that without adequate positive end expiratory pressure alveoli were unstable and susceptible to derecruitment (10)

MAINTENANCE OF MUSCLE TONE.

Atelectasis develops even at induction due to loss of muscle tone. Hence, anesthetics that allow maintenance of respiratory muscle tone will prevent formation of atelectasis. Ketamine does not impair muscle tone and does not cause atelectasis. So far this is the only anesthetic tested that does not cause collapse. However, atelectasis appears even with ketamine anesthesia once muscle relaxants are used.

Stimulation of the phrenic nerve was tried to restore respiratory muscle tone. This method reduced the atelectatic area, but the overall effect was small. The other disadvantage in this technique is that it is too complicated for routine use during anesthesia and surgery (9)(19)(23)

RECRUITMENT STRATEGIES.

Recruitment maneuver opens up the atelectatic alveoli by sufficient inspiratory pressures and keeps them open with the help of positive end expiratory pressure. A combined initial pressure beyond the opening pressure of the collapsed alveoli with enough positive end expiratory pressure to stabilize the newly opened units

reduces atelectasis and improves lung compliance, hence increasing the lung available for gas exchange.

Sigh maneuver, or a double tidal volume (Vt) is one of the modes of recruitment. On increasing the airway pressures to 30 cm H_2O only nearly half the collapsed alveoli open up. On further increasing pressures to 40cm of H2O complete reopening of all collapsed lung tissue occurs. Such large tidal volumes are inhaled even during a maximum spontaneous inspiration, and it can thus be called a vital capacity maneuver. Hence airway pressures of +40 cm H_2O in healthy lungs, maintained for not more than 7 to 8 seconds may re-open all previously collapsed lung.(9)(19)(23)

MINIMISING GAS RESORPTION.

Ventilation with pure oxygen is not protective against atelectasis. It was found that even after recruiting collapsed alveoli by any of the recruitment maneuvers there was rapid reappearance of the atelectasis when 100% oxygen was used. Whereas, when 40% oxygen in nitrogen was used for ventilation of the lungs, atelectasis reappeared slowly, and 40 minutes after the recruitment maneuver only 20% of the initial atelectasis had reappeared. Thus,

moderate fraction of inspired oxygen (e.g., FiO_2 of 0.3 to 0.4) should be used for ventilation during anesthesia as long as it is sufficient to maintain arterial oxygenation. (19)(23)

Preoxygenation during induction of anesthesia also has an effect on the formation of atelectasis. Although the safe period against hypoxia is increased in cases of difficult intubation by preoxygenation, there is an additional risk of atelectasis that occurs on breathing 100% oxygen even for a few minutes before and during commencement of anesthesia. By totally avoiding preoxygenation atelectasis formation during induction can be minimized but the apnea period is shortened. Hence, by using lower fractions of inspired oxygen at the time of induction, throughout the maintenance of general anesthesia and at the time of extubation the amount of formation of atelectasis can be reduced. Preoxygenation with 100% Oxygen can also be provided without producing atelectasis if undertaken with continuously increased airway pressure, as with continuous positive airway pressure (CPAP).This technique might provide the greatest safety without atelectasis formation but it requires a tight system and might be complicated in clinical practice.(19)(23)

POSTANESTHETIC OXYGENATION.

Similar to preoxygenation, post anesthetic oxygenation also promotes formation of atelectasis. At the end of the surgery, 100% oxygen is routinely used for ventilation. This procedure is often combined with suctioning of the airway tree. Both oxygenations with suctioning add on to the atelectasis in postoperative ward. Post anesthetic oxygenation (100% Oxygen) 10 minutes before termination of anesthesia together with a vital capacity maneuver at the end of anesthesia did not protect against atelectasis at the end of anesthesia.[79]Derecruitment of previously opened lung tissue is the probable cause. Instead, a recruitment maneuver with lower concentration of oxygen at the time of extubation reduces atelectasis formation. (19)(23)

RESPIRATORY FUNCTION

DURING GENERAL ANESTHESIA

General anesthesia is associated with impairment of pulmonary function and oxygenation in previously normal lungs. .This extends into the post-operative period resulting in significant pulmonary complications ranging from1% to 2% following minor surgery to up to 20% following thoracic and abdominal surgery. Impaired tissue oxygenation even in the intraoperative period increases surgical site infections

General anesthesia induces changes in the pulmonary function in the following sequence:

loss of muscle tone, change in the balance between respiratory muscles and elastic tissue in the lung leads to a fall in functional residual capacity followed by reduced compliance and an increase in respiratory resistance. The decrease in functional residual capacity is associated with the formation of atelectasis (made worse with the use of high concentrations of inspired oxygen) and airway closure. There occurs ventilation perfusion mismatch which in turn impedes oxygenation of blood and removal of carbon dioxide. (19)

i) LUNG VOLUME

The volume of air that remains in the lungs after a normal passive exhalation is termed as functional residual capacity. Normal functional residual capacity is 3000-4000ml.Functional residual capacity decreases by up to 20% with induction of anesthesia, regardless of intravenous or inhalational mode of induction. Functional residual capacity is the physiological reserve of the lung. The balance of forces between the lung and chest wall determines the functional residual capacity. A disturbance in the above process reduces the chest and lung volume. This has been observed in anesthesia due to loss of tone of the respiratory muscles. Diaphragm shifts cranially causing decrease in functional residual capacity, alveolar collapse, increase in the shunt fraction and finally hypoxaemia. (19)(24)

Factors such as position of the patient, type of surgery, intraperitoneal insufflation and use of neuromuscular blockers also determine thoracic compliance and lung volumes through their effects on the position of the diaphragm and intra-abdominal pressure (24)

ii) ATELECTASIS AND AIRWAY CLOSURE

Along with atelectasis intermittent airway closure, which is increased in anaesthetized state, adds to the low (Va/Q) units. The reduced ventilation in the lower half of the lung, just above the atelectasis is reasonably explained by airway closure. Airway closure and atelectasis together explains nearly 74% of the impaired arterial oxygenation. (19)

iii) COMPLIANCE AND RESISTANCE

Total thoracic compliance = 100ml/cm H_2O:

Lung compliance = 200ml/cm H_2O

Chest wall compliance = 200ml/cm H_2O

Airflow resistance is normally around 1 cm H_2O/L/sec

Thoracic compliance decreases and airway resistance increases in anesthesia due to reduction in the lung volumes. The total static compliance of the thorax reduces from an average of 95 to 60ml/cm of cm H_2O during anesthesia. (19)

iv) DISTRIBUTION OF VENTILATION AND BLOOD FLOW DURING ANESTHESIA

Redistribution of inspired gas away from dependent to nondependent lung regions occurs in anesthetized supine humans.

Almost no ventilation occurs in the dependent areas of the lung due to atelectasis. Use of positive end expiratory pressure restores overall functional residual capacity toward or beyond the awake level, hence returns gas distribution toward the awake pattern by reopening of closed airways and recruitment of collapsed, dependent lung regions.(19)

Perfusion increases down the lung, from the nondependent to the dependent regions, with some reduction in the lowermost region. The lowermost portion of the lung, which is atelectatic as evidenced by simultaneous CT, is still perfused. Positive end expiratory pressure forces perfusion into dependent areas of the lung increasing flow to the atelectatic areas. Positive end expiratory pressure reduces cardiac output by decreasing venous return in hypovolemia (19)

Hypoxic pulmonary vasoconstriction is blunted by most anesthetics, thereby enhancing any ventilation-perfusion mismatch.

Ideally, ventilation should match perfusion perfectly (V/Q = 1) at the alveolus, but there is always some mismatch .When blood flows through areas of low V/Q ratio, oxygenation is incomplete, resulting in reduced levels of oxygen in the arterial blood

(hypoxaemia). During anesthesia, ventilation –perfusion mismatch occurs commonly. This is because of the fall in the functional residual capacity, which in turn changes the position of the lung on the compliance curve. The apices move to the most favorable part of the curve while the bases are located on a less favorable part at the bottom of the curve. In areas of atelectasis and airway closure due to lack of ventilation, the V/Q ratio reaches zero. Such areas are referred to as **shunt**. Blood flowing through these areas will not have any change in the partial pressure of oxygen resulting in significant hypoxaemia. Oxygen status cannot be improved by simply increasing the inspired oxygen concentration, as the inspired gas does not reach the alveolus due to poor ventilation. The well-ventilated parts of the lung cannot compensate for the area of shunt because hemoglobin is fully saturated at a normal pO_2. Increasing the pO_2 of this blood will not increase the oxygen content substantially. In the case of shunt, therefore, adequate oxygenation can only be re-established by restoring ventilation to these areas using measures such as physiotherapy, positive end expiratory pressure or Continuous positive airway pressure, which clear blocked airways and re-inflate areas of collapsed lung.(19)(24)

At the other extremes of V/Q mismatch, an area of lung receiving no perfusion will have a V/Q ratio of ∞ (infinity) and is referred to as **alveolar dead space**, which together with the anatomical dead space makes up the physiological dead space. Such ventilation is wasted ventilation, as there is no blood available for gas exchange. (19)(24)

v) RESPIRATORY DRIVE

A dose dependent depression of the central and peripheral chemoreceptor response to carbon dioxide and oxygen is observed with anesthetic agents. Attenuation of the hypoxic response may be attributed to an effect on the carotid body chemoreceptors. The effect of anesthesia on respiratory muscles is nonuniform. Rib cage excursions diminish with deepening anesthesia. The normal ventilatory response to carbon dioxide is produced by the intercostal muscles. Thus, the reduced ventilatory response to carbon dioxide during anesthesia is due to impeded function of the intercostal muscles. (19)(24)

ADVANTAGES OF INCREASING INTRAOPERATIVE ARTERIAL OXYGENATION

Surgical site infections is a common and sometimes, serious complication of surgery. Though infections are typically not detected until some days after surgery, multiple factors at the time of surgery also influence the incidence of infections. These factors are:

a) the surgical site
b) complexity of surgery
c) co-existing illness
d) use or nonuse of prophylactic antibiotics
e) the patient's temperature during surgery
f) Patient's hydration status
g) the extent of post-operative pain relief
h) Partial pressure of oxygen in the tissue.

Destruction by oxidation, or oxidative killing, is the most important defense against surgical pathogens and depends on the partial pressure of oxygen in contaminated tissue. Oxidative damage is mediated through free radicals derived from molecular oxygen.

These free radicals are toxic to the bacteria. The enzyme NADPH-linked oxygenase, required for the generation of free radicals, depends on the oxygen tension in the tissue, which ranges from 0 to 300mm of Hg in the neutrophils. In the areas of tissue damage, as in surgical trauma there is a compromise in the local blood flow due to direct injury to the vessels. Surgery being a pro-coagulant state further contributes to this by promoting thrombosis within the vessels. Hence, the wound tissues are often hypoxic with their partial pressure of oxygen being less than 30 mm of Hg. By this it could be inferred that the oxygen tension, often at levels greater than that required to maintain saturation, plays an important role in protecting tissues against infection. (25) (26)

Other advantages that have been observed with increased intra operative arterial oxygen are decrease in the post-operative nausea and vomiting, improvement in the intestinal perfusion and better post-operative cognitive scores.

ABOUT DRAGER FABIUS PLUS (27)(28)

Dräger fabius plus was employed in the study for mechanical ventilation intra operatively.

This machine has an electronically controlled, electrically driven piston ventilator that provides facilities to ventilate patients of all age groups in any of the common modes of ventilation. Advanced features such as fresh gas decoupling, which maintains constant tidal volumes regardless of fresh gas flow are present. The volume of the breathing system excluding the hoses and reservoir bag is 1.7litres. The Fabius plus can be equipped with two vaporizers for volatile anesthetic agents. The combination of its high-precision ventilator and its leak-tight breathing system design makes the Fabius plus well suited for low-flow ventilation. It comes with a standard reusable soda lime canister of 1.5litres capacity for carbon dioxide absorption that can be mounted on a height adjustable swivel arm. In addition, it has an auxiliary oxygen flow meter. A Standardized Dräger user interface gives way for easy and intuitive operation.

It allows a fresh gas flow of up to 12litres/minute. However, it can provide oxygen up to 75litres/min at 87 psi with the use of

oxygen flush. The ventilatory modes available on the anesthesia delivery machine in our set up are: spontaneous breathing, manual ventilation, volume controlled ventilation and pressure controlled ventilation.

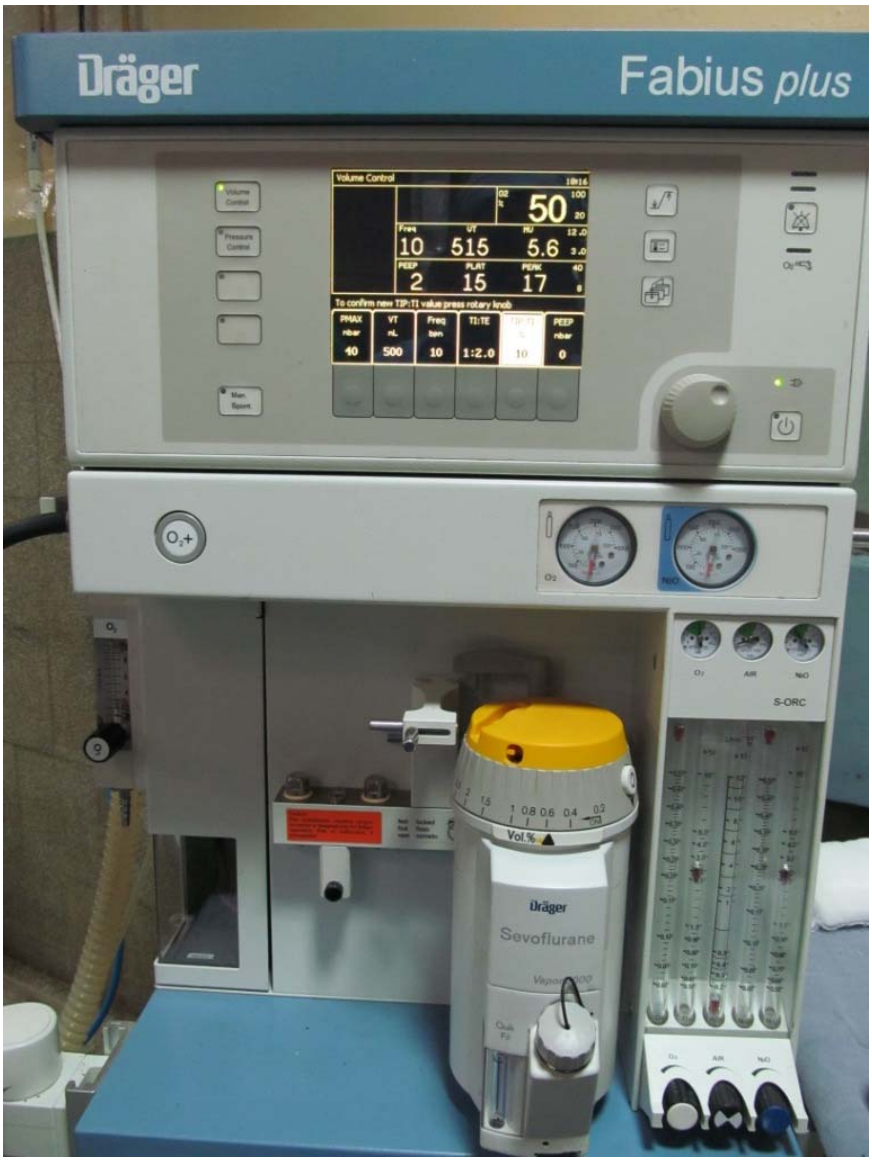
The frequency of respiration can be set from 4 to 60 breaths per minute. Tidal volumes ranging from 20ml to 1400ml can be set in the volume control mode. The end expiratory pressure can be set from 0cm of H_2O to +20cm of H_2O. The peak inspiratory pressures depend on the set PEEP values ranging from PEEP + 5 to 65cm of H2O.The limiting pressures can be varied from 15 to 70 cm of H_2O. It provides for an inspiratory flow rate of 10 to 75litres/min in both volume and pressure control modes. The ratio of inspiration time to expiration time (Ti: Te) can be varied from 4:1 to 1:4.An inspiratory pause of up to 50% can be set.

The Fabius plus features an integrated, high-contrast 6.5-inch monitor (with color option) which displays all relevant ventilation parameters and a pressure curve. Continuous monitoring of fraction of inspiratory oxygen, respiratory rate, expiratory tidal volume and minute ventilation, positive end expiratory pressure, peak and

plateau pressure is possible. . Gas flow rates are monitored via conventional glass flow meter.

Safety measures: Hypoxic mixture of fresh gas flow is prevented by sensitive oxygen ratio controller. In case the oxygen flow drops below 200ml/min, supply of nitrous oxide is cut off. Audible and visual alarms are activated in case of fall in the oxygen pressure below 20±4 psi. In cases of crisis as failure of power due to electricity and battery, manual ventilation is still possible with intact fresh gas and agent delivery. A safety relief valve that opens at high pressures (75±5cm of H_2O) is present.

DRAGER FABIUS PLUS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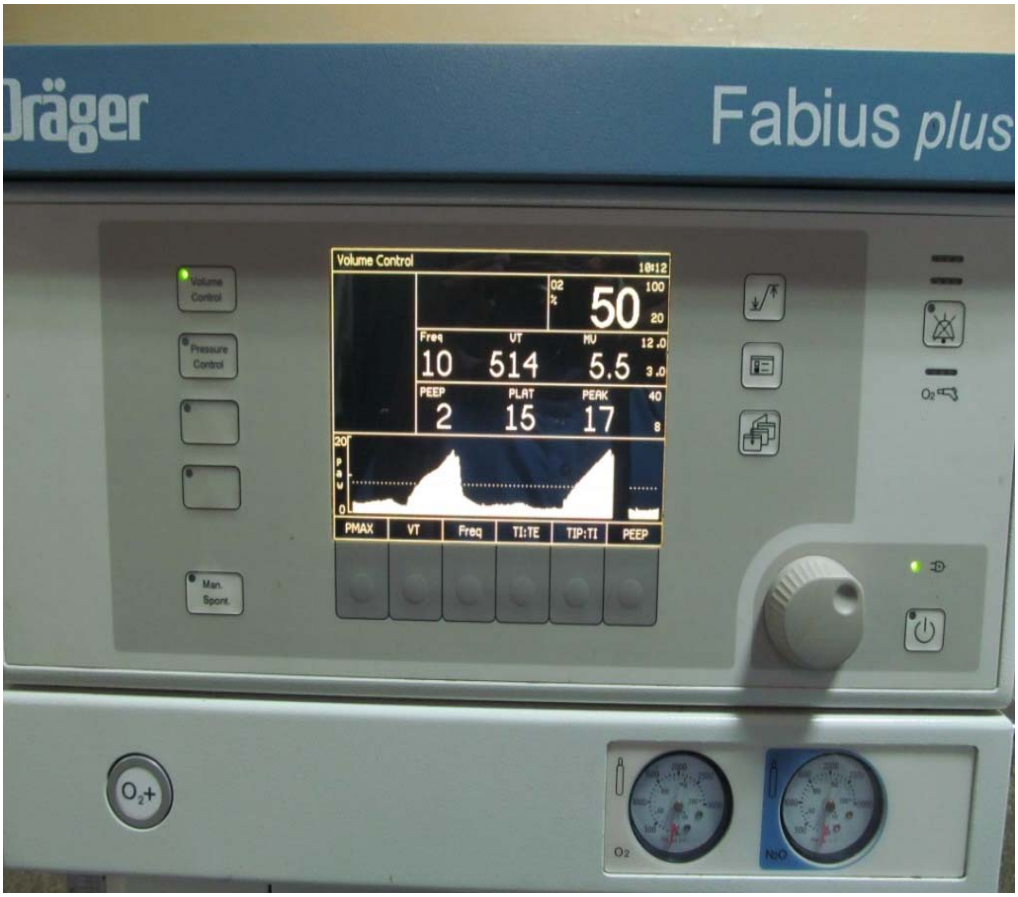
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O2
N2O

ASSESSMENT OF ARTERIAL OXYGEN CONTENT

One of the main factors determining oxygen delivery to cells is the oxygen content of the blood. Oxygen diffuses passively across the alveolar capillary membrane to reach the blood in the pulmonary capillaries. Most of this oxygen binds to the hemoglobin and is transported as oxy-hemoglobin, whereas only a minimal amount is carried by the blood in the dissolved form. The partial pressure of oxygen in the arterial blood is influenced by several physiological disturbances.

Routinely, pulse oxymetry is used to determine oxygen saturation which gives an idea about the oxygen status of the arterial blood. It is a simple, non-invasive method. Arterial blood gas analysis though invasive is a better method to assess the oxygen tension. Partial pressure of oxygen in the arterial blood or PaO_2 alone speaks little about the efficiency of gas exchange that occurs across the alveolar capillary membrane because it does not estimate the physiological shunt. By assessing the shunt fraction, a rough idea about the severity of the lung pathology is obtained which in

turn guides the oxygen requirements of the patient. Physiological shunt can be calculated by different methods. The classic shunt equation is the best method up to date used to calculate the physiological shunt but it is more complex requiring samples of mixed venous blood. Alveolar- arterial gradient can also be used. (29)A simpler index of arterial oxygenation efficiency corresponds to ratio of partial pressure of arterial oxygen to the fraction of inspired oxygen. Hence, the ratio PaO_2/FiO_2 was used to assess the arterial oxygen content in this study. (29) Efficacy of positive end expiratory pressure in converting areas of atelectasis into functional alveolar-capillary units is assessed by an increase in the PaO_2/FiO_2 ratio. This ratio fails to rise; rather a fall may be seen in case of excessive positive end expiratory pressure resulting in over distension of alveoli leading to ventilation – perfusion mismatch. (30)

As room air contains 20% oxygen, a healthy adult will have a partial pressure of arterial oxygen around 80 to 100 mm of Hg. Using this the PaO_2/FiO_2 ratio of 400 -500 mm of Hg was obtained PaO_2/FiO_2ratio of less than 200 most often indicates a shunt greater than 20% .A notable limitation of PaO_2 at low FiO_2 ratio is this that

it does not take into account changes in $PaCO_2$ at low FiO_2which tends to have a considerable effect on the ratio. (29)

AIRWAY PRESSURES AND COMPLIANCE

A few words about certain parameters recorded in the study.

Peak inspiratory pressure: the pressure used to deliver the tidal volume by overcoming resistance offered by the non-elastic airways and the elastic pulmonary parenchyma. It is measured usually at the end of inspiration. It is the maximum pressure measured during one respiratory cycle. Changes in airway pressures are affected by changes in tidal volume, airway resistance, peak inspiratory flow rate and compliance. It is directly related to tidal volume and peak inspiratory flow rate. (31)Keeping tidal volume constant, an increase in airway resistance or a decrease in compliance, or both causes an increase in the peak inspiratory pressure. (30)

Plateau pressure: the pressure needed to maintain lung inflation in the absence of air flow. (31)By doing inflation hold maneuver there occurs a drop in the proximal airway pressure to reach a plateau which is called as plateau pressure. Plateau pressure

is unaffected by changes in airway resistance, but is inversely related to lung compliance. (30)

Hence, decreased compliance is associated with increase in both the peak and plateau pressures whereas an increased peak pressure with no changes in the plateau pressure is suggestive of increased airway resistance. The difference between the peak and plateau pressures is a measure of changes in airway resistance. (30)

Thus the proximal airway pressures can be used to evaluate the mechanical properties of the lungs during mechanical ventilation (30)

Compliance: denotes distensibility of the lungs, i.e. the property of elastic recoil of the lungs. Lung compliance is the degree of lung expansion or change in volume per unit pressure change or the work of breathing. (31) (32) Stiffer lungs as in pulmonary fibrosis have poorer compliance and increased work of breathing. Compliance also varies within the lung according to the degree of inflation. At low volumes, initial lung inflation is difficult leading to poor compliance and increased work of breathing and at high volumes, there occurs limitation of chest wall expansion again

increasing the work of breathing. Compliance is found to best in the mid-expansion range. (33)

Compliance is affected by various factors.

Age- in neonates and infants the immature development of alveoli reduces lung compliance, in contrast their cartilaginous ribs contributes to very compliant chest wall. The lung compliance improves with alveolar maturity and also chest compliance decreases slowly with age. (34)

Posture-thoracic compliance is lower in supine position due to upward displacement of diaphragm. (34)

Body mass index-in an obese individual compliance is poor which is worsened by effects of posture (34)

Anesthesia-several factors influence compliance under anesthesia usually decreasing the compliance is decreased. Some of the factors are:

a) supine position
b) airway closure
c) changes in intrathoracic blood volume
d) accumulation of fluid
e) direct effect of drugs

f) altered muscle tone

g) external pressure

Diseases-in atelectasis, the lung is relatively stiff or non - compliant. There is greater pull inwards with a low functional residual capacity, further increasing atelectasis. In emphysema, a high compliance state, functional residual capacity is greater due to lack of elastic recoil of lungs. (31) (34)

Compliance can be subdivided into static or dynamic compliance. Static compliance reflects the elastic properties of lung and chest wall. It is measured in the absence of airflow. (31) (32)

Static compliance = Tidal volume/ (Plateau pressure – PEEP)

Dynamic compliance reflects the airway resistance, both elastic and nonelastic. It is measured during normal tidal breathing. As it is measured in the presence of airflow airway resistance, in turn peak airway pressure is an important determining factor. (31) (34)

Dynamic compliance=Tidal volume/ (Peak pressure –PEEP)

Changes in plateau pressure and static compliance are associated with similar changes in peak airway pressure and dynamic compliance. Atelectasis increases plateau and peak airway pressures. Hence, a decrease in the static and dynamic compliance is observed in atelectasis. But, changes in peak airway pressure, as in conditions of increased airway resistance, cause changes in the dynamic compliance without any change in plateau pressure or static compliance. (31)

Positive end expiratory pressure–increases the baseline airway pressure to greater than the atmospheric pressure. Positive end expiratory pressure is found to increase proximal airway pressures as the entire pressure waveform is displaced upwards. (30) (31) Positive end expiratory pressure acts as a stent for distal airways and counterbalances the compressive forces produced by the elastic recoil of the lungs. (30)By alveolar recruitment it increases the functional residual capacity, improves ventilation, decreases ventilation perfusion mismatch, and finally improves

oxygenation and decreases the work of breathing. Hence positive end expiratory pressure is indicated intra pulmonary shunt decreased lung compliance and decreased functional residual capacity. (30) (31) Positive end expiratory pressure is titrated and often set between the upper and lower inflection point. (31)

Associated complications: Positive end expiratory pressure has the potential to decrease venous return and hence cardiac output but this effect depends on the compliance characteristics of the individual. (30) (31)

Barotrauma can result when on increasing the positive end expiratory pressures increases the mean airway pressures above 30cm of H_2O or peak inspiratory pressures above 50cm of H_2O. (31)

Impaired cerebral venous drainage due to increase in the intra thoracic pressure during positive end expiratory pressure can increase intra cerebral pressure. (31)

Renal blood flow may also be impaired affecting the normal kidney functions. (31)

REVIEW OF LITERATURE

1. A comparison between 'Open lung' ventilation and conventional ventilation was done by T.N. Weingarten et al. In Recruitment Maneuver group patients received tidal volume of 6 ml/kg and 12 cm H_2O positive end expiratory pressure. In the control group, conventional ventilation was employed with no recruitment maneuver. Patients received tidal volume of 10 ml/kg and zero end-expiratory pressure. The study was conducted in elderly patients beyond 65 yrs. of age posted for laparotomies under general anesthesia. Pre and post -operative serum was sampled for interleukins IL-6 and IL-8.In the case group, arterial oxygenation increased during surgery without compromise in the hemodynamic status whereas after surgery, it was found to be comparable with the control group. Dynamic compliance improved in the case group. Resistance of the airway was found to be reduced in the case group than in the control group. A similar rise in the interleukin levels was found in both the groups which

suggested that the systemic inflammatory response to surgery was not affected by ventilatory strategy. (35)

2. Tusman et al employed strategies to recruit alveoli in his study. During general anesthesia, atelectasis could be prevented by increasing the inspiratory pressures to initially open up the collapsed airways followed by a positive end expiratory pressure to keep them open. Patients were divided into 3 groups based on whether they received no positive end expiratory pressure; an initial control period without positive end expiratory pressure followed by positive end expiratory pressure of 5 cm H_2O; and positive end expiratory pressure of 15 cm H_2O with high tidal volume (18 ml/kg, or peak inspiratory pressure 40 cm of H_2O) maintained for 10 breaths, followed by stepwise reduction of positive end expiratory pressure and tidal volume. A significant increase in arterial oxygen content was observed in patients with alveolar recruitment i.e. the third group during general anesthesia. However, the increase in oxygenation was not similar in patients in the second group receiving positive end expiratory

pressure of 5 cm H_2O alone. The authors concluded that during general anesthesia the collapsed alveoli could be recruited by high initial pressures and that at least 5cm of positive end expiratory pressure is needed to keep the recruited alveoli open. An additional finding was absence of barotrauma or other pulmonary complications that could be caused by increased airway pressures.(36)

3. Anesthesia has gross effects on the pulmonary function of patients with high body mass index. One of the causes for this is formation of atelectasis. Henrik Reinius et al studied the effect of general anesthesia. He used 3 different strategies to improve lung function. A prospective randomized study was conducted in thirty patients (body mass index 45 +/- 4 kg/m^2) posted for gastric bypass surgery .They were divided into three groups: (1) positive end-expiratory pressure of 10 cm H_2O (2) a recruitment maneuver with 55 cm H_2O for 10 s followed by zero end-expiratory pressure, (3) a recruitment maneuver followed by positive end expiratory pressure. Transverse and spiral CT scans were taken to record the

development of atelectasis. Arterial blood gas analysis was done to know the status of oxygenation before and after the maneuver. The conclusion was that recruitment maneuver followed by positive end expiratory pressure reduced atelectasis and improved oxygenation in morbidly obese patients. Positive end expiratory pressure or a recruitment maneuver alone was not comparably effective**.** (37)

4. Patients undergoing laparoscopic bariatric surgery were tested for the effect of ventilatory strategy on arterial oxygenation. After carbon dioxide insufflation, the study group received 4 consecutive tidal volumes of sufficient magnitude to increase peak airway pressures to 50 cm of H_2O .Following this a baseline PEEP of 12cm of H_2O was kept. The control group followed a standard pattern of ventilation with positive end expiratory pressure of 4 cmH2O. Francis X. Whalen et al found that dynamic compliance and arterial oxygenation can be improved in intubated patients. These effects were short-lived lasting only in the intraoperative

period. All the beneficial effects on oxygenation disappeared with extubation. (38)

5. Marta Coussa et al studied the efficacy of use of positive end expiratory pressure in morbidly obese patients as a measure to prevent atelectasis. 23 highly obese adults (body mass index>35kg/m2) were randomly assigned to one of two groups. In the first group patients were spontaneously breathing pure oxygen with a continuous positive pressure of 10cm of H_2O. Once induced with anesthetic agents, these patients were ventilated with a face mask with a positive end expiratory pressure of 10 cm H2O. In the control group, the same induction was applied but without continuous positive airway pressure or positive end expiratory pressure. Ct scans were taken and ABG analysis done prior to induction and immediately after intubation. Atelectasis developed more rapidly after intubation in the control group compared to the positive end expiratory pressure group .The study concluded that in morbidly obese patients, atelectasis formation is largely prevented by positive end expiratory pressure applied during

the anesthetic induction and is associated with a better oxygenation (4)

6. Marco Rusca et al also tested the use of positive end expiratory pressure at the time of induction of anesthesia in patients breathing 100% oxygen. Continuous positive pressure of 6 cm of H_2O during spontaneous ventilation and a positive end expiratory pressure of 6 cm of H_2O manual ventilation via face mask prior to intubation in the study group and no Continuous positive airway pressure or positive end expiratory pressure in the control group. A similar conclusion was derived that atelectasis formation is prevented by application of positive end expiratory pressure during the anesthesia induction despite the use of large oxygen concentrations, resulting in improved oxygenation. (39)

7. Leonid Minkovich et al conducted a study with ninety-five patients requiring elective cardiac surgery with cardiopulmonary bypass. The study group received inflation pressures of 35 cm H_2O sustained for 15 seconds before

separation from Cardiopulmonary bypass and at 30 cm H_2O for 5 seconds after admission to the intensive care unit (ICU). The primary outcome was the ratio of arterial oxygen tension to inspired oxygen fraction measured at the following specific time intervals: after induction of anesthesia, 15 minutes after separation from cardiopulmonary bypass, after admission to the intensive care unit, after 3 hours of positive-pressure ventilation, after extubation, and before intensive care unit discharge. Consecutive vital capacity maneuvers resulted in better arterial oxygenation extending from the immediate postoperative period to approximately 24 hours after surgery at the time of intensive care unit discharge. (40)

8. A prospective case control study was conducted by B. A. Claxton et al in 78 patients scheduled for on pump cardiac surgery. The patients were randomly assigned to 3 groups of twenty six each based on the ventilation strategy the received. Group 1 received no PEEP with conventional ventilation

Group 2 patients were ventilated with conventional ventilation methods with additional PEEP of 5cm of H_2O until they were extubated in the ICU

Group 3 patients were ventilated with incremental tidal volumes up to 18ml/kg and PEEP up to 15 cm of H_2O such that a peak airway pressure of 40cm of H_2O was achieved. This was repeated ten times followed by a baseline PEEP of 5cm of H_2O till the patient was extubated in the ICU.

Oxygenation was found to improve in group 3 where in the alveoli were recruited at 30 and 60 minutes following bypass. There was a statistical significant difference in the oxygenation when compared with the other groups. However, beyond 60 minutes all the groups were comparable. Group 2 did not have any significant improvement in the oxygenation with 5 cm H_2O of PEEP. No adverse effects were noted during the study. Hence, the authors concluded arterial oxygen tension in patients following bypass surgeries could be improved by recruiting the alveoli (41)

9. Talab HF et al conducted a study in 66 adult obese patients with a body mass index between 30 and 50 kg/m2 scheduled to undergo laparoscopic bariatric surgery. The patients were randomly assigned to 3 groups. According to the recruitment maneuver used, the zero end-expiratory pressure (ZEEP) group (n 22) received the vital capacity maneuver (VCM) maintained for seven to eight seconds that was applied soon after intubation without positive end expiratory pressure. The positive end-expiratory pressure 5 group (n 22) received the Vital capacity maneuver maintained for 7–8 s applied immediately after intubation along with 5 cm H_2O of positive end expiratory pressure; and the positive end expiratory pressure10 group (n 22) received the vital capacity maneuver maintained for 7–8 s applied immediately after intubation plus 10 cm H2O of positive end expiratory pressure. The authors observed that intraoperative alveolar recruitment with a Vital Capacity Maneuver followed by positive end expiratory pressure 10 cm H2O is effective at preventing lung atelectasis and is associated with better oxygenation, early discharge from post anesthesia care unit

with minimal post- operative lung complications in patients with BMI more than 30 kg/m^2 .scheduled for laparoscopic bariatric surgery (42)

10. Atelectasis was evaluated by computed tomography (CT) in 13 ASA I–II patients undergoing elective surgery. CT scans were obtained before and 15 min after induction of anaesthesia. Then, recruitment of collapsed lung tissue was performed as a “vital capacity maneuver” (VCM, inspiration with Paw=40 cmH2O for 15 s), and a CT scan was obtained at the end of the VCM. Thereafter, positive end expiratory pressure=0 cmH2O was applied in group 1, and positive end expiratory pressure=10 cmH2O in group 2. Additional CT scans were obtained after the vital capacity maneuver. Oxygenation was measured before and after the vital capacity maneuver. Neumann et al concluded that positive end expiratory pressure=10 cmh2o reduced atelectasis formation after a vital capacity maneuver, when FiO2=1.0 was used. Thus, a Vital Capacity Maneuver followed by positive end expiratory pressure of 10 cmH2O should be considered when

patients are ventilated with a high FiO2 and gas exchange is impaired. (43)

11. Rothen et al employed strategies to open up atelectatic areas in 16 healthy adults. Ten patients in the first group received incremental airway pressures of 10, 20, 30 and 40 cm of H_2O.The remaining six patients in the other group received 3 cycles of lung inflation until peak airway pressures reached 30cm of H_2O.Further increase up to 40cm of H_2O was done in the second group. CT scans was taken at different levels of airway pressures. Atelectatic areas were found to be lower at higher airway pressures with minimal atelectasis at 40 cm of H_2O. They concluded that, in anesthetized patients, Ventilation by standard tidal volume or double tidal volume did not reduce the areas of atelectasis. Only a vital capacity maneuver was found to open up most of the collapsed lung tissues.(44)

MATERIALS AND METHODS

A prospective randomised double blind study was done to evaluate the effect of recruitment maneuver on intraoperative arterial oxygenation in patients undergoing laparotomy under general anaesthesia. The study was carried out in 40 adult patients at Govt. Rajaji Hospital, Madurai after getting ethical committee approval. The patients were divided into two groups of 20 each.

INCLUSION CRITERIA:

- Age group 20-55 years
- Both sexes
- ASA I, II
- Elective Laparotomies

EXCLUSION CRITERIA:

- Patients with significant pulmonary disease either restrictive or obstructive like active asthma
- COPD
- Previous lung surgery
- significant cardiac dysfunction

Pre anesthetic evaluation:

1. History
2. Clinical examination
3. Relevant investigations – Hemoglobin, Blood sugar, Renal function tests, Electrolytes, ECG, Chest x ray
4. Informed consent from patients

METHODS

A standardized anesthetic technique was used. All patients were premedicated with inj. glycopyrrolate 0.2mg i.m. 45 minutes before surgery. Basic monitors as pulse oxymetry, NIBP, ECG, Capnography were connected. All patients were induced with inj. thiopentone 5mg/kg, Inj. fentanyl 2mics/kg, Inj. succinylcholine 1.5mg/kg i.v. and intubated. Inj. fentanyl and Inj. atracurium were used for intraoperative maintenance in titrated doses. Sevoflurane 0.6-1% was used for maintenance. Patients were reversed with Inj. neostigmine40mics/kg and Inj. glycopyrrolate 20mics/kg i.v. and extubated on table.

Patients were randomized to receive one of the two ventilation strategies. In both groups after tracheal intubation an

inspired oxygen fraction (FiO2) of 0.5, Inspiratory to expiratory time ratio of 1:2 was set.

In **control group**, patients were ventilated with a tidal volume of 10ml/ kg at a rate of 10 breaths/minute with zero end expiratory pressure.

In **recruitment maneuver group**, patients were ventilated with tidal volume of 8ml/kg and a positive end expiratory pressure of 4 cm of H_2O at a respiratory rate of 10breaths/minute. In this group, lung recruitment was achieved by sequential increases in positive end expiratory pressure in three steps from 4 to 10 cm H2O (for 3 breaths), 15 cm H2O (for 3 breaths), and 20 cm H2O positive end expiratory pressure (for 10 breaths). After recruitment, the level of positive end expiratory pressure was maintained at 12 cm H2O throughout the entire operation. Lung recruitment was repeated at 30 and 60 min after the first recruitment and hourly thereafter

PARAMETERS MONITORED:

The following parameters were recorded at 0, 5, 15, 30, 60, 90, 120 minutes and at end of surgery:

a. Pulse rate
b. blood pressure
c. arterial oxygen saturation
d. end tidal carbon dioxide
e. peak and plateau airway pressures
f. positive end expiratory pressure
g. expiratory tidal volume
h. Minute ventilation
i. Dynamic compliance (tidal volume /Peak inspiratory pressure-positive end expiratory pressure)

Arterial blood gas analysis was done pre-operatively before premedication and was taken as the baseline value, at 1 hour of intubation and again at 30minutes after extubation in the recovery room. The PaO_2/FiO_2 ratio was calculated by using these values

In case of any fall in mean arterial pressure below 20% of the baseline, after ensuring adequate hydration titrated doses of

ephedrine was planned to be used. Hence, in addition to the above parameters, intraoperative blood loss, use of crystalloids, colloids and vasopressors were noted.

ANALYSIS OF DATA

The observations were recorded for both the groups as shown in the master chart.

STATISTICAL TOOLS

The information collected regarding all the patients in the study was recorded in a Master Chart. Data analysis was done with the help of SPSS for Microsoft Windows Version 15.The values for mean with their standard deviations were obtained. ANOVA was employed to compare the means between the two groups. ‘p’ value less than 0.05 was considered significant.

OBSERVATION AND RESULTS

The following characteristics are presented as mean, standard deviation and p value.

TABLE 1: Age distribution (in years)

Age	**Recruitment maneuver group**	**Control group**
Mean	42.7	40.00
SD	8.316	9.021
p value	0.638 not significant	

The mean age of the Recruitment maneuver Group was 42.7 years and the control Group was 40years.There was no statistically significant difference (p = 0.638).

TABLE 2: Duration of surgery (in minutes)

Duration	**Recruitment maneuver group**	**Control group**
Range	60 – 120	45 – 120
Mean	86.5	89.5
Standard deviation	22.94	26.15
p value	0.702 not significant	

Duration of surgery in recruitment maneuver group was 86.5 ± 22.94 min whereas in the control group it was 89.5+/-26.15min with the difference between them not being significant.

TABLE 3: Bodyweight of the patients (in kg)

Body weight	**Recruitment maneuver group**	**Control group**
Mean	49.25	49.75
Standard deviation	3.89	5.15
p value	0.731 not significant	

Patients in both the groups were of similar body weights.

TABLE 4: Variation of Positive end expiratory pressure over time during surgery

(in cm of H_2O)

	Baseline	**5min**	**15min**	**30min**	**60min**	**90min**	**120min**	**End of surgery**
Recruitment maneuver group	4.000 (0.00)	12.000 (0.00)	12.000 (0.00)	12.000 (0.00)	12.000 (0.00)	12.000 (0.00)	12.000 (0.00)	12.000 (0.00)
Control group	0.100 (0.307)	0.700 (0.470)	1.000 (0.561)	1.250 (0.550)	1.555 (0.615)	1.583 (0.514)	1.666 (0.516)	1.550 (0.604)
p value	**<0.001**	**<0.001**	**<0.001**	**<0.001**	**<0.001**	**<0.001**	**<0.001**	**<0.001**

The set positive end expiratory pressure values were significantly higher in the recruitment maneuver group.

Variation of Positive end expiratory pressure over time during surgery

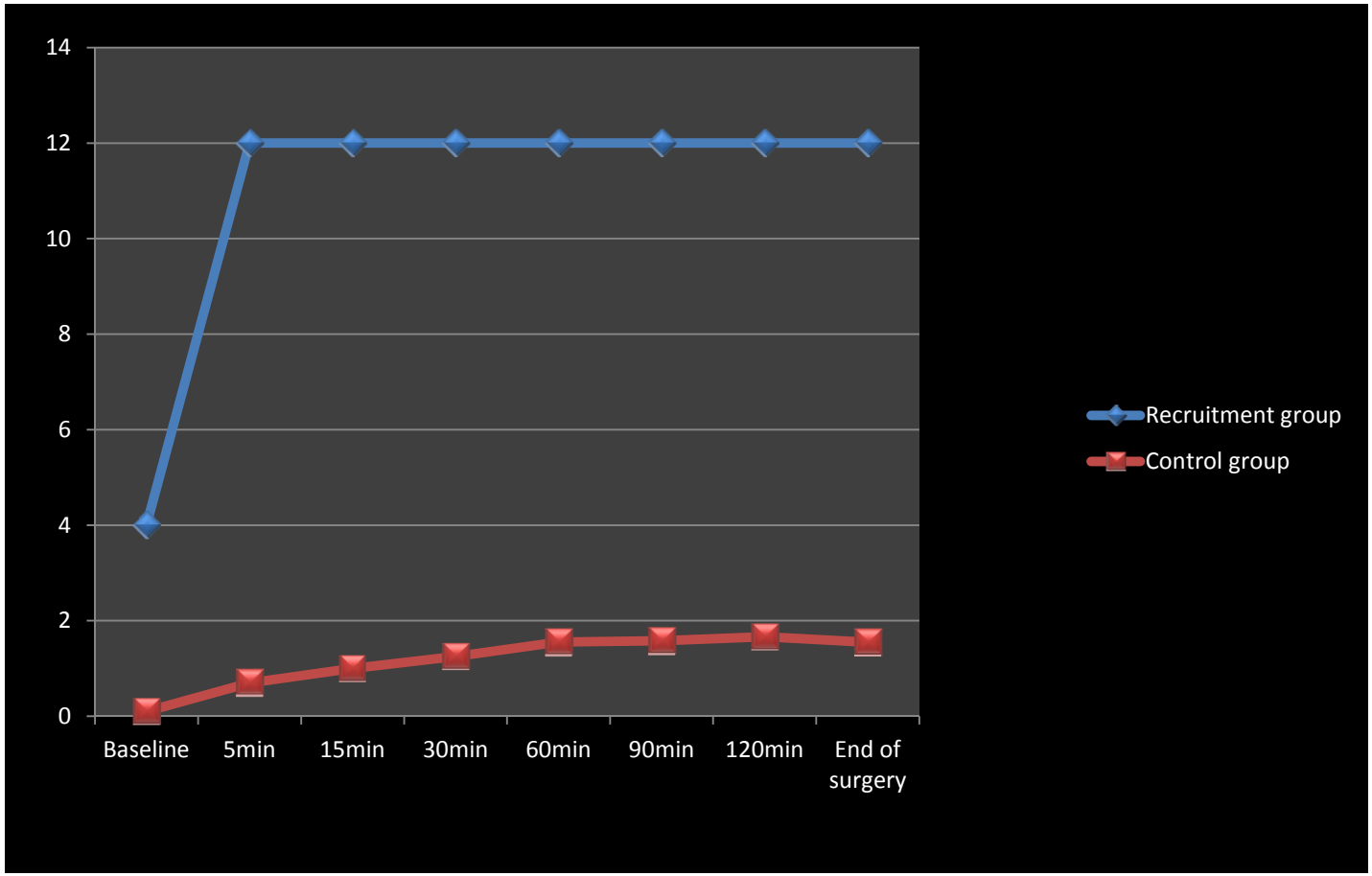

TABLE 5: Variation of peak inspiratory pressure over time during surgery

(in cm of H_2O)

	Baseline	**5min**	**15min**	**30min**	**60min**	**90min**	**120min**	**End of surgery**
Recruitment maneuver group	13.300 (0.733)	19.850 (1.663)	21.250 (1.019)	21.550 (0.9445)	21.850 (0.933)	22.400 (1.173)	22.500 (1.290)	22.150 (1.039)
Control group	14.800 (0.951)	14.700 (1.031)	14.740 (1.020)	14.800 (0.951)	14.670 (0.907)	14.750 (0.622)	15.170 (0.753)	14.850 (0.813)
p value	**<0.001**	**<0.001**	**<0.001**	**<0.001**	**<0.001**	**<0.001**	**<0.001**	**<0.001**

Variation of peak inspiratory pressure over time during surgery

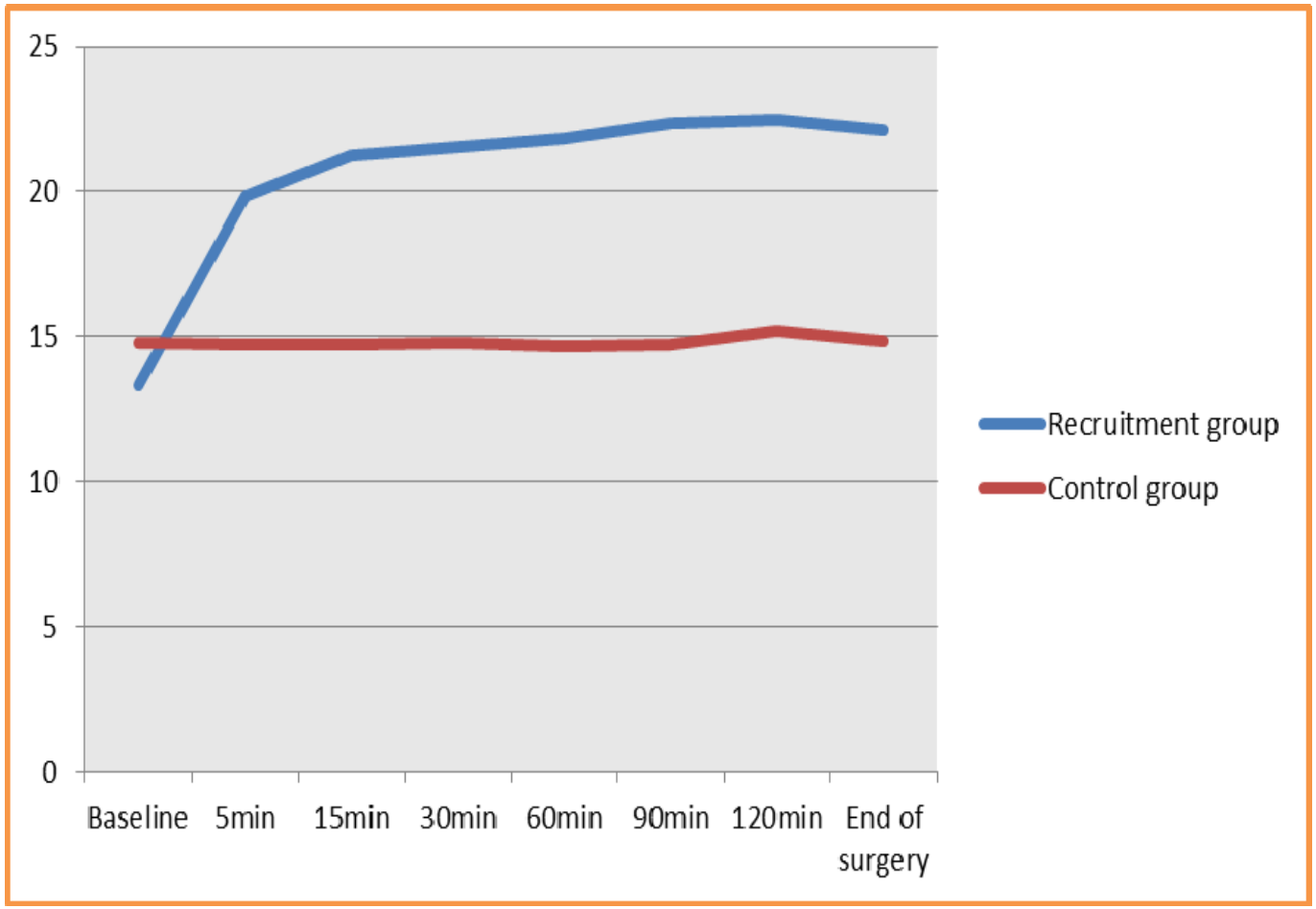

TABLE 6: Variation of plateau pressure over time during surgery (in cm of H_2O)

	Baseline	**5min**	**15min**	**30min**	**60min**	**90min**	**120min**	**End of surgery**
Recruitment maneuver group	11.75 (0.786)	18.500 (1.820)	20.050 (1.276)	20.600 (0.882)	20.750 (0.910)	21.100 (1.197)	21.000 (1.414)	20.900 (1.020)
Control group	13.500 (1.051)	13.350 (1.424)	13.100 (0.968)	13.300 (0.979)	13.390 (1.037)	13.170 (0.835)	13.500 (1.049)	13.300 (1.081)
p value	**<0.001**	**<0.001**	**<0.001**	**<0.001**	**<0.001**	**<0.001**	**<0.001**	**<0.001**

Both plateau pressures and peak inspiratory pressures were more in the recruitment maneuver group than in the control group and these values were found to be significant.

Variation of plateau pressure over time during surgery

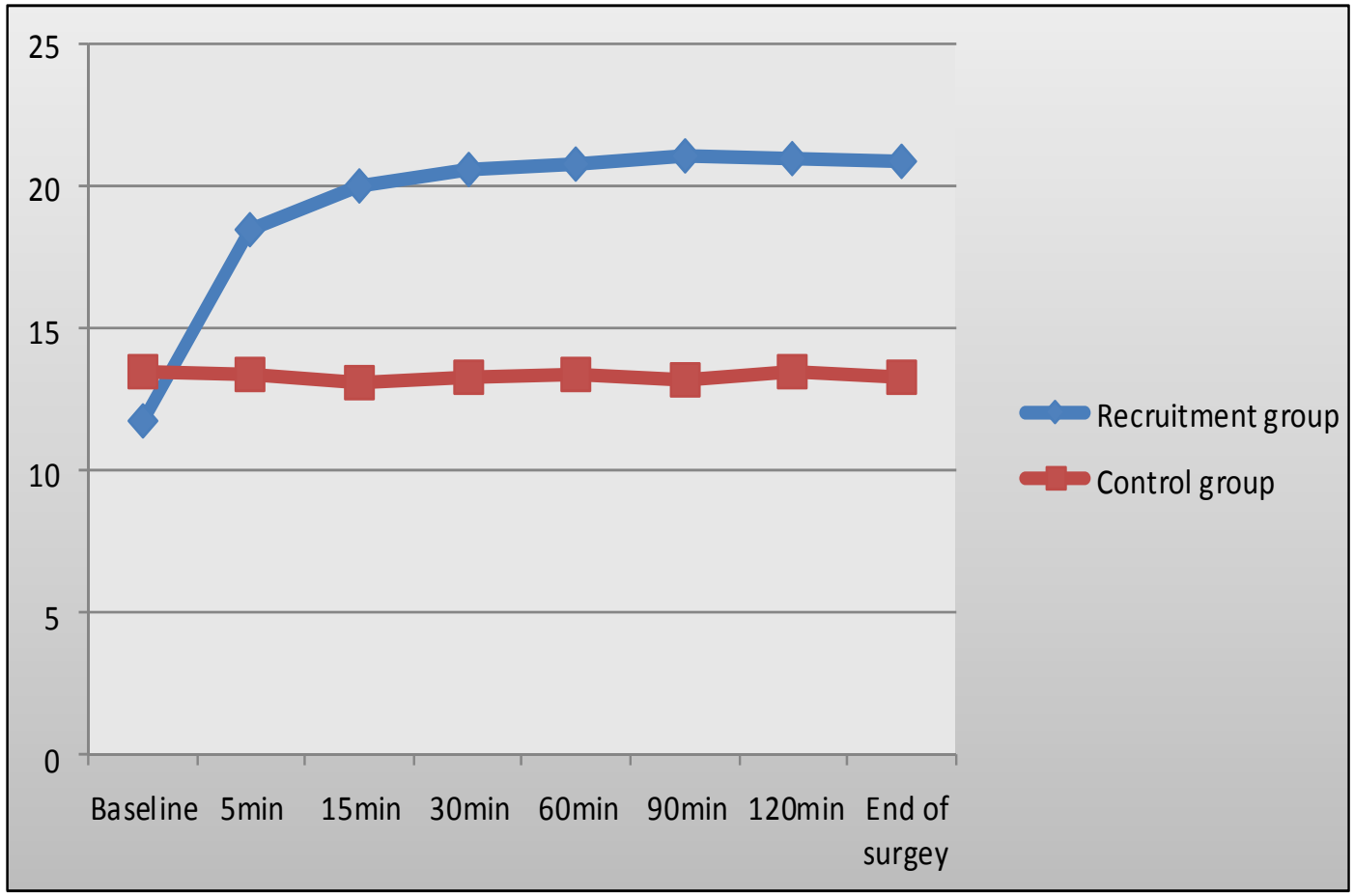

TABLE 7: Variation of Expiratory Tidal volume over time during surgery (in ml)

	Baseline	**5min**	**15min**	**30min**	**60min**	**90min**	**120min**	**End of surgery**
Recruitment maneuver group	394.75 (31.726)	434 (44.71)	408.75 (41.003)	457.5 (51.999)	463.75 (50.702)	431.5 (45.829)	460 (58.31)	451 (47.117)
Control group	497.25 (51.694)	504.25 (59.322)	508 (54.541)	501.75 (48.943)	507.22 (58.784)	505 (51.079)	503.33 (40.825)	507.5 (57.927)
p value	<0.001	<0.001	<0.001	0.009	0.02	0.002	0.201	0.002

Variation of Expiratory Tidal volume over time during surgery

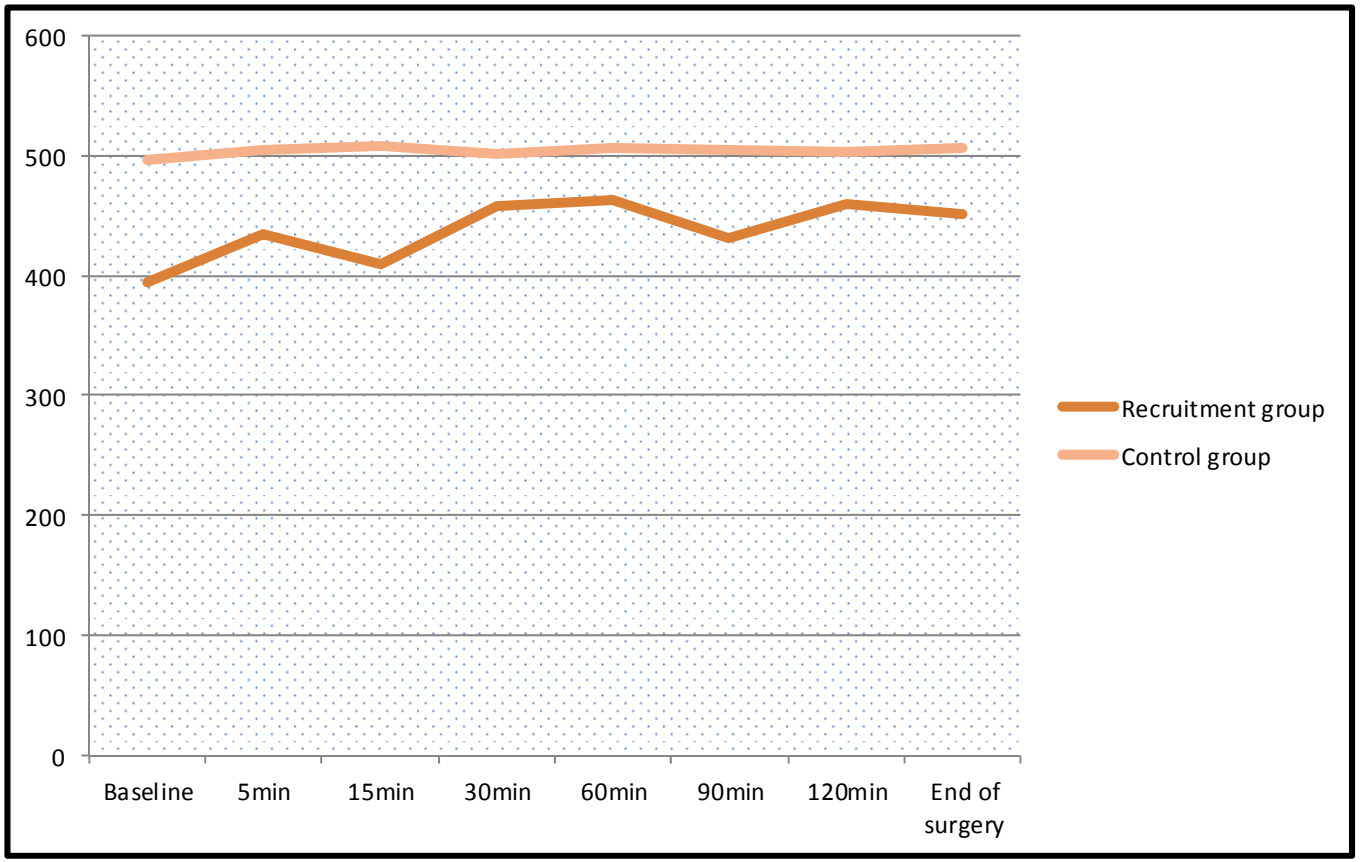

TABLE 8: Variation of minute ventilation over time during surgery (in litres/min)

	Baseline	5min	15min	30min	60min	90min	120min	End of surgery
Recruitment maneuver group	3.955 (0.321)	4.362 (0.437)	4.085 (0.412)	4.575 (0.519)	4.640 (0.503)	4.320 (0.451)	4.600 (0.583)	4.610 (0.560)
Control group	4.985 (0.514)	5.045 (0.594)	5.080 (0.545)	5.030 (0.495)	5.061 (0.589)	5.092 (0.512)	5.033 (0.408)	5.075 (0.572)
p value	<0.001	<0.001	<0.001	0.007	0.023	0.001	0.201	0.013

A significant increase in the tidal volumes and minute ventilation was observed in the recruitment maneuver group after every recruitment maneuver.

Variation of minute ventilation over time during surgery

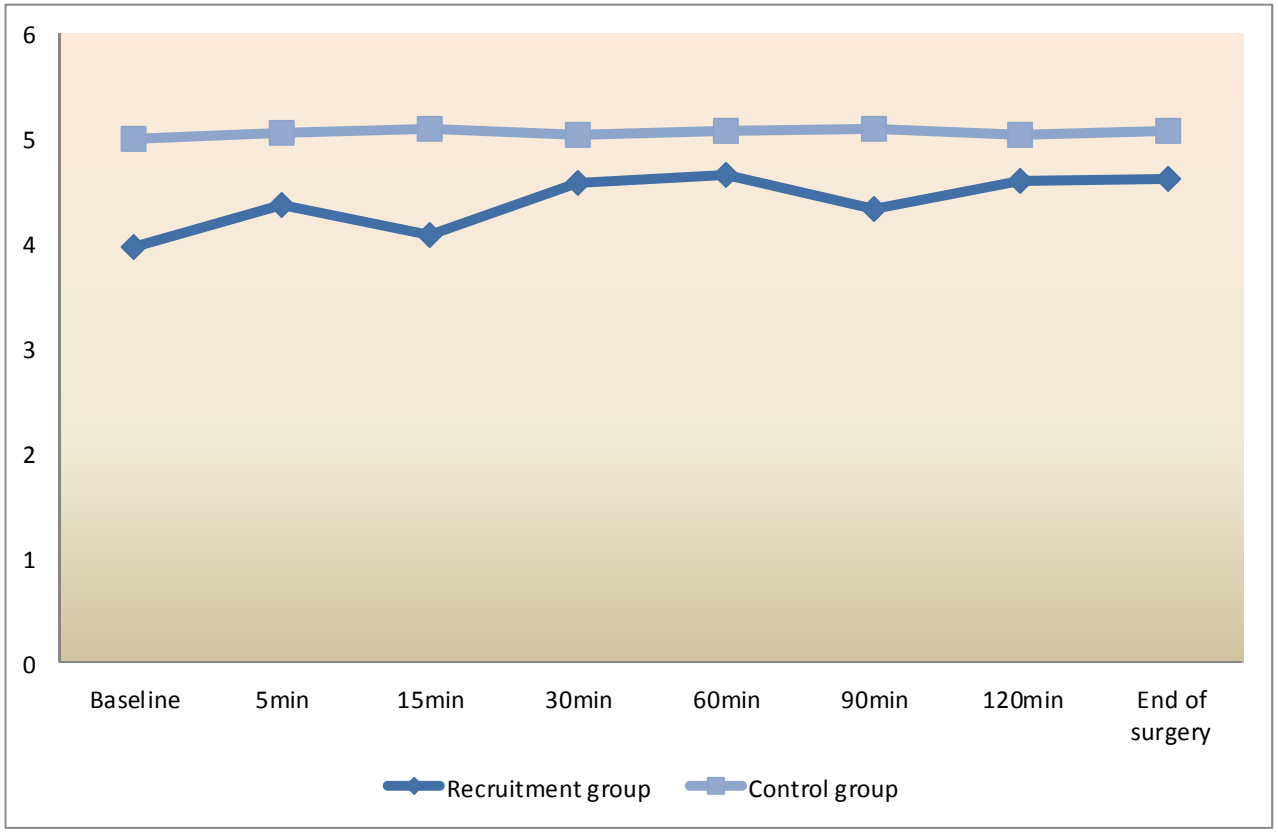

TABLE 9: Variation of dynamic compliance over time during surgery(in ml/cm of H_2O)

	Baseline	**5min**	**15min**	**30min**	**60min**	**90min**	**120min**	**End of surgery**
Recruitment maneuver group	42.455 (4.164)	58.924 (16.854)	44.956 (6.297)	48.784 (8.472)	47.859 (7.119)	45.076 (5.709)	43.832 (2.147)	45.167 (6.124)
Control group	34.022 (4.337)	36.173 (4.854)	37.045 (4.431)	37.189 (4.276)	38.843 (5.174)	38.587 (5.572)	37.305 (3.017)	38.307 (5.093)
p value	**<0.001**	**<0.001**	**<0.001**	**<0.001**	**<0.001**	**0.014**	**0.006**	**<0.001**

Dynamic compliance significantly increased from baseline values in the study group.

Variation of dynamic compliance over time during surgery

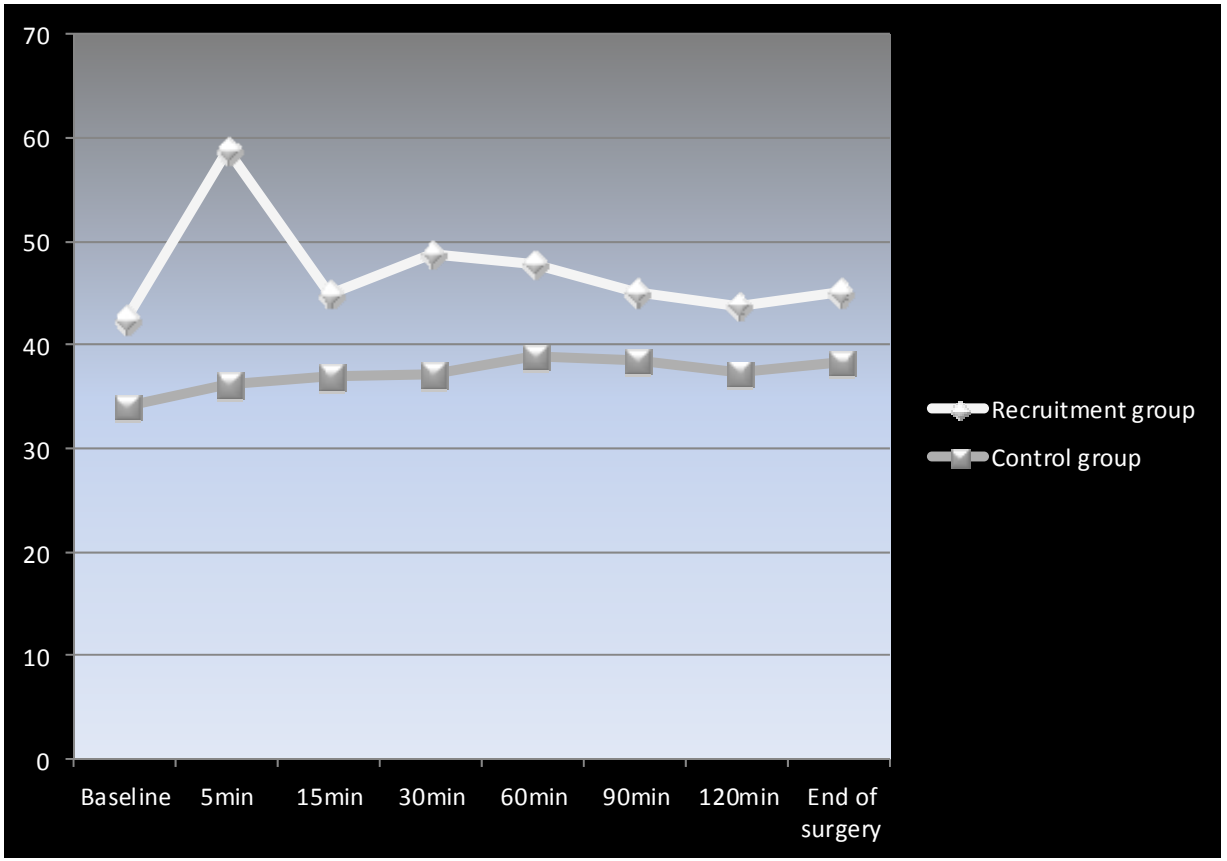

TABLE 10: Intra and post-operative oxygenation (in mm of Hg)

	PaO_2/FiO_2 Baseline	**PaO_2/FiO_2 At 60min**	**PaO_2/FiO_2 Recovery room**
Recruitment maneuver group	478.700 (31.241)	581.050 (15.476)	459.050 (28.570)
Control group	468.950 (26.466)	511.900 (13.494)	450.200 (27.331)
p value	**0.294** not significant	**<0.001 significant**	**0.323** not significant

A significant increase in the arterial oxygenation was observed at 60 min that drops to insignificant values in the recovery room, 30 min after extubation.

INTRA AND POST-OPERATIVE OXYGENATION

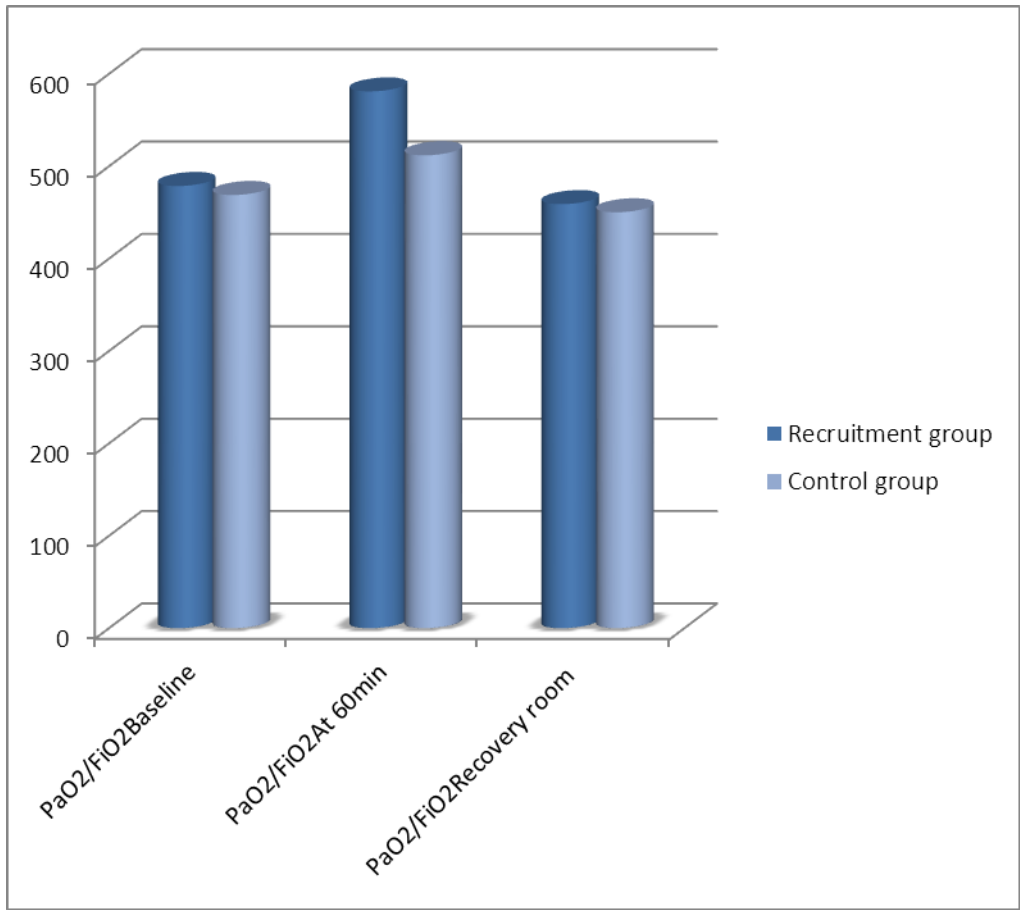

TABLE 11: Variation of End tidal carbon dioxide over time during surgery(in mm of Hg)

	Baseline	**5min**	**15min**	**30min**	**60min**	**90min**	**120min**	**End of surgery**
Recruitment maneuver group	36.100 (1.586)	37.650 (1.308)	38.250 (1.517)	39.500 (1.504)	41.200 (2.567)	41.800 (2.616)	42.250 (3.304)	42.050 (2.855)
Control group	35.550 (2.665)	36.350 (1.755)	36.400 (1.759)	36.150 (2.345)	36.611 (1.539)	36.250 (2.005)	37.000 (1.673)	36.450 (1.731)
p value	0.433	0.012	0.001	<0.001	<0.001	<0.001	0.010	<0.001

A significant increase in End tidal carbon dioxide over time during surgery was observed in recruitment maneuver group when compared with the control group.

VARIATION OF END TIDAL CARBON DIOXIDE OVER TIME DURING SURGERY

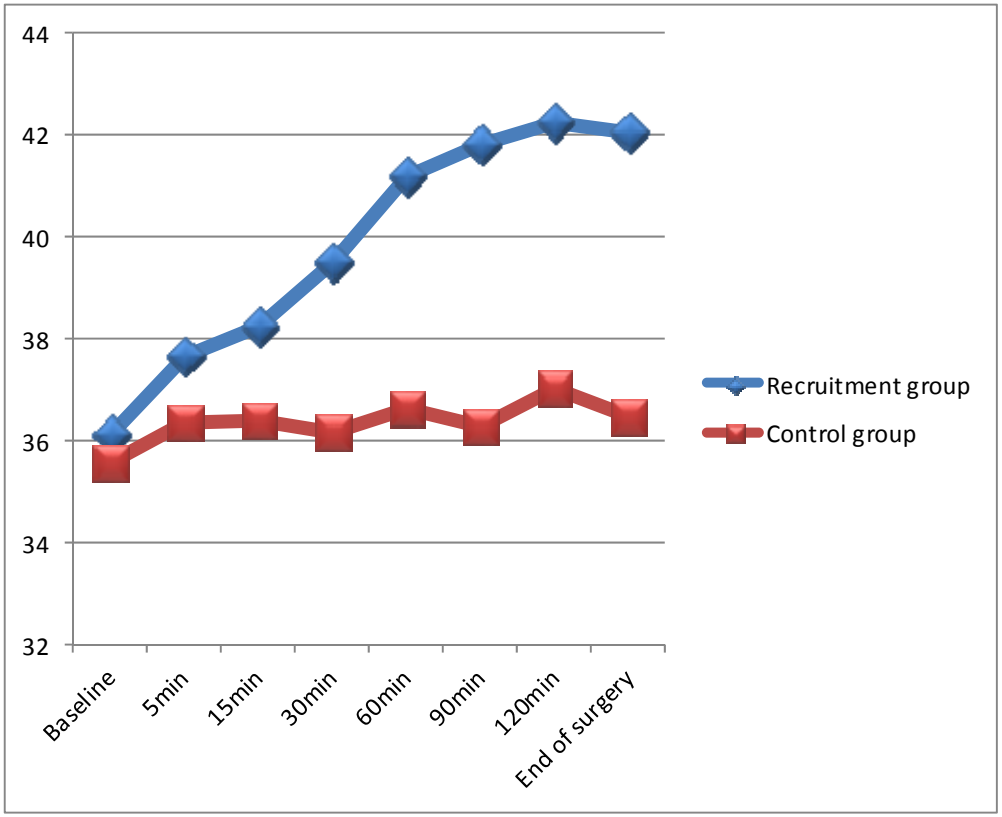

TABLE 12: Variation of pulse rate over time during surgery (in beats/min.)

	Baseline	**5min**	**15min**	**30min**	**60min**	**90min**	**120min**	**End of surgery**
Recruitment maneuver group	79.800 (12.580)	81.450 (10.575)	86.250 (10.025)	86.800 (8.062)	88.500 910.096)	89.500 (10.926)	96.000 (8.286)	90.85 (10.413)
Control group	78.900 (14.238)	83.550 (14.442)	83.600 (12.650)	87.111 (12.625)	90.083 (13.324)	96.000 (8.286)	94.333 (10.073)	87.65 (12.770)
p value	0.833	0.823	0.743	0.345	0.709	0.913	0.791	0.391

Changes in the pulse rate in both the groups were comparable.

TABLE 13: Variation of mean arterial pressure over time during surgery (in mm of Hg)

	Baseline	**5min**	**15min**	**30min**	**60min**	**90min**	**120min**	**End of surgery**
Recruitment maneuver group	84.450 (9.417)	90.900 (8.0320	93.350 (8.916	95.850 (8.797)	98.100 (6.479)	98.300 (4.137)	101.500 (4.725)	99.650 (6.547)
Control group	84.250 (11.087)	85.950 (11.771)	85.700 (12.397)	85.100 (10.391)	88.500 (11.073)	93.500 (10.689)	97.000 (7.771)	90.500 (10.520)
p value	0.951	0.129	0.031	0.001	0.002	0.197	0.335	0.002

Random variations in the mean arterial pressure were observed with no specific relation to changes in end tidal carbon dioxide or recruitment maneuver.

TABLE 14: Blood loss during surgery (in ml)

Blood loss	Recruitment maneuver group	Control group
Mean	236	329.75
Standard deviation	142.66	214.23
p value	0.112 not significant	

TABLE 15: Use of intravenous fluids during surgery (in ml)

	Recruitment maneuver group	Control group	p value
crystalloids	2085 (383.23)	2120 (437.21)	0.789 not significant
colloids/blood	350 (0.00)	525 (191.70)	0.07 not significant

DISCUSSION

Atelectasis is a common pulmonary complication of general anesthesia. It is known that atelectasis decreases the available lung units for ventilation thereby decreases lung compliance and increases the shunt. This results in decrease in the arterial oxygen content.

In this study, the effect of intraoperative alveolar recruitment maneuvers was studied wherein the collapsed alveoli were opened with high airway pressures and kept open with the help of positive end expiratory pressure.

Patients in the age group 18-55yrs were randomly selected for this study. Most of the patients in the recruitment maneuver group were of 42.7 ± 8.316years whereas in the control group, most of the patients were of 40 ± 9.021 years. In both cases and control, the patients were comparable with respect to duration of surgery (86.5 +/- 22.94 min in the cases and 89.5+/-26.15minin the control group).As body habitus also affects atelectasis formation, weight of the individual was also taken into consideration. Both the groups were comparable regarding their body weight.

Positive end expiratory pressures as set in the recruitment group was significantly greater than the zero end expiratory pressure in the control group. As a result, significantly higher proximal airway pressures were noted in the recruitment group compared to the control group.

Expiratory tidal volumes and minute ventilation over time were recorded in the study. An increase in the tidal volume and a corresponding increase in the minute ventilation were observed after every recruitment maneuver. In this study, from a baseline mean of394.75ml the expiratory tidal volume increased to a mean of 434 ml after the first recruitment maneuver at 5 minutes, 457.5ml at 30 minutes, 463.75ml at 60 minutes, 460ml at 120minutes when subsequent recruitment maneuvers were done. However, it remained constant around 500ml in the control group.

A significant difference between the dynamic compliance of the cases and control was noticed. (p value< 0.02)Dynamic compliance improves over time resulting in a significant increase in the intra operative arterial oxygenation in the case group as indicated by the PaO_2/FiO_2ratio. ($p<0.001$)But it was noticed that in the recovery room the PaO_2/FiO_2 ratio in both the groups were

similar with no significant difference when compared to the baseline ratios. Hence, recruitment maneuvers used during intraoperative mechanical ventilation improved the intraoperative arterial oxygen content that can be attributed to increased dynamic compliance. A similar observation was made by T.N. Weingarten et al in his study. (35)

A gradual fall in the dynamic compliance over time was observed in the study group but these values remained above the baseline. Tidal volumes at 15min and 90 min were comparatively lower than those at 5 min, 30min, 60 min and 120 min. The probable cause for this could be slow reappearance of atelectasis. (35)

Even in the absence of recruitment strategies, arterial oxygen saturation is maintained within normal limits. But these maneuvers significantly increase the tissue oxygen content which may reduce surgical site infections. (25) (35) By reducing areas of atelectasis, recruitment maneuvers reduce the mechanical stress to the lung units. This decrease in the intraoperative lung injury may in turn reduce the post-operative pulmonary complications.

Capnography was employed to record end tidal carbon dioxide. As shown by these recordings, carbon dioxide accumulation over time was more in the recruitment group than in the control group and this finding was statistically significant. End tidal carbon dioxide values were maintained less than 45mm of Hg throughout the surgery. In case of any increase beyond 45mm of Hg, it was brought well within limits by increasing the respiratory rate by increments of 2breaths/minute.

Intraoperative features like blood loss, use of intravenous fluids, variations in pulse rate were similar in both the groups. Mean arterial pressures varied randomly with no specific relation to recruitment maneuver or rise in end tidal carbon dioxide.

Vasopressors were not required in either of the groups. In the study by T.N. Weingarten et al, cardiac index was measured and there was no evidence suggestive of hemodynamic instability during the recruitment maneuver. (35)

Limitations of the study: post-operative pulmonary complications were not assessed. The possible benefits of increasing intra operative oxygenation such as reduced incidence of surgical site infections were not assessed in the post-operative period.

SUMMARY

The study was conducted to evaluate the effect of alveolar recruitment maneuvers during intra operative mechanical ventilation. Forty patients of ASA I and II, posted for elective laparotomies were randomly allocated into two groups.

In the study group, high positive end expiratory pressures for specified number of breaths were used to increase the peak inspiratory pressures. This increased airway pressure recruited the atelectatic alveoli .The recruited alveoli were prevented from collapsing by a baseline positive end expiratory pressure and repeated recruitment at regular intervals. No such recruitment strategies were employed in the control group.

Proximal airway pressures and dynamic compliance significantly increased in the study group. ($p<0.05$)A significant increase in the arterial oxygen content was observed at 60 min in the study group. (PaO_2/FiO_2 ratio 581.05).But in the recovery room, the PaO_2/FiO_2 ratios in both the groups were comparable to the baseline.

Hence, recruitment maneuvers increased the intra operative arterial oxygen content with not much effect in the post-operative period.

CONCLUSION

General anesthesia comes hand in hand with atelectasis. Prevention of atelectasis not only increases arterial oxygenation but also reduces associated complications. This study concluded that alveolar recruitment maneuver significantly increased intra operative arterial oxygenation, which might be beneficial in preventing pulmonary complications and surgical site infections in the post-operative period.

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PROFORMA

Name: study group:

Age/sex: IP No.:

Diagnosis: Procedure:

Weight:

EXCLUDE COPD, active asthma, previous lung surgery

ASA RISK:

	baseline	5min	15min	30min	60min	90min	120min	End of surgery
EtCO2								
plateau pressure								
Peak insp. Pressure								
PEEP								
Expiratory tidal volume								
minute ventilation								
Dynamic compliance (tidalvol./Pip-PEEP)								
Pulse rate								
BP								
SPO2								

Duration of surgery: Blood loss: Ephedrine:

Crystalloids: colloid/blood:

PaO2/FiO2 Obtained by 3 arterial blood gas samples:

1. Baseline
2. 1hour after intubation
3. 30 minutes after extubation.

MASTER CHART

Sl. No.	name	case/contol	age	sex	weight	duration	PEEP-base	PEEP5min	PEEP15min	PEEP30min	PEEP60min	PEEP90min	PEEP120min	PEEP-EOS	PIP-base	PIP5min	PIP15min	PIP30min	PIP60min	PIP90min	PIP120min	PIP-EOS	pl.P- base	pl. P5min	pl. P15min	pl. P30min	pl. P60min	pl. p90min	pl. P120min	pl. P-EOS	Vt-base	Vt5min	Vt15min
1	sudha	1	39	f	45	75	4	12	12	12	12			12	13	20	21	21	21			22	12	18	20	20	20			20	360	400	380
2	thangakani	1	25	f	45	60	4	12	12	12	12			12	13	20	22	23	21			21	12	19	21	21	20			20	360	375	360
3	senthil	1	30	m	50	90	4	12	12	12	12	12		12	14	21	22	22	23	22		24	12	20	22	22	23	23		23	400	420	410
4	kumar	1	44	m	45	100	4	12	12	12	12	12		12	12	20	22	22	22	22		24	10	18	21	21	21	22		23	360	385	360
5	jayalakshmi	1	39	f	41	120	4	12	12	12	12	12	12	12	13	19	20	21	21	21	21	21	12	19	20	20	20	19	19	20	330	345	360
6	subbaiah	1	49	m	50	120	4	12	12	12	12	12	12	12	14	20	22	23	23	22	24	24	12	18	20	22	22	21	22	22	400	500	475
7	vaijayanthimala	1	30	f	46	60	4	12	12	12	12			12	14	16	19	21	21			21	13	15	17	20	20			20	370	400	380
8	ramayee	1	50	f	55	60	4	12	12	12	12			12	13	18	20	20	21			21	11	16	19	20	20			20	440	450	430
9	rakku	1	43	f	54	90	4	12	12	12	12	12		12	13	22	22	22	22	23		23	12	20	20	20	21	21		21	435	440	430
10	irulandi	1	53	m	50	80	4	12	12	12	12			12	13	17	20	20	20			22	11	15	18	19	20			20	400	500	480
11	jagan	1	28	m	53	100	4	12	12	12	12	12		12	14	23	23	22	24	21		23	12	22	22	21	22	22		22	425	500	480
12	rajan	1	48	m	53	60	4	12	12	12	12			12	13	22	22	22	22			22	12	21	21	22	21			21	430	410	430
13	muthu	1	45	m	48	75	4	12	12	12	12			12	13	20	20	21	22			22	12	18	20	20	20			20	380	400	390
14	subramani	1	50	m	51	90	4	12	12	12	12	12		12	14	20	22	22	22	22		22	12	18	20	20	20	21		21	410	475	445
15	fathima	1	47	f	52	120	4	12	12	12	12	12	12	12	15	21	22	22	23	21	23	23	13	20	21	22	22	22	22	22	420	450	410
16	thevar	1	40	m	47	60	4	12	12	12	12			12	13	18	21	21	21			21	12	17	20	20	20			20	375	440	370
17	kannan	1	46	m	45	120	4	12	12	12	12	12	12	12	13	20	21	22	22	21	22	22	12	19	20	21	21	20	21	21	360	400	360
18	sundariammal	1	53	f	50	60	4	12	12	12	12			12	12	20	21	21	22			22	10	19	20	20	21			21	400	440	375
19	chandra	1	39	f	50	80	4	12	12	12	12			12	13	20	22	23	22			22	11	20	21	21	21			21	400	470	420
20	ganesan	1	42	m	55	110	4	12	12	12	12	12		12	14	20	21	20	22	21		21	12	18	18	20	20	20		20	440	480	430

Sl. No.	name	case/contol	Vt30min	Vt60min	Vt90min	Vt120min	Vt-EOS	MV-base	MV5min	MV15min	MV30min	MV60min	MV90min	MV120min	MV-EOS	dync base	dync5 min	dync15min	dync30min	dync60min	dync90min	dync120min	dync-EOS	P/F-base	P/F60min	P/F-recovery	etcd base	etcd5min	etcd15min	etcd30min	etcd60min	etcd90min	etcd120min
1	sudha	1	400	410			430	3.6	4	3.8	4	4.1			4.3	40	64	52	56	58			50	485	563	452	36	37	37	40	42		
2	thangakani	1	380	400			400	3.6	3.8	3.6	3.8	4			4	40	47	36	35	40	43		44	472	580	467	40	40	42	42	45		
3	senthil	1	440	460	430		430	4	4.2	4.1	4.4	4.6	4.3		4.3	40	47	41	44	42	37		36	466	564	440	34	36	38	40	42	42	
4	kumar	1	400	400	375		380	3.6	3.9	3.6	4	4	3.8		3.8	45	47	36	40	40	42		32	480	600	453	38	40	40	42	43	43	
5	jayalakshmi	1	380	400	380	410	410	3.3	3.5	3.6	3.8	4	3.8	4.1	4.1	41	49	45	42	44	48	46	46	386	558	375	36	37	37	40	42	44	43
6	subbaiah	1	510	510	480	520	520	4	5	4.8	5.1	5.1	4.8	5.2	5.2	40	63	48	46	46		43	43	490	568	480	37	38	38	38	38	40	42
7	vaijayanthimala	1	400	410			410	3.7	4	3.8	4	4.1			4.1	37	100	54	40	46			46	476	576	453	35	37	38	40	46		
8	ramayee	1	490	510			510	4.4	4.5	4.3	4.9	5.1			5.1	49	75	54	61	57	40		57	530	602	500	34	38	38	39	43		
9	rakku	1	480	470	440		440	4.4	4.4	4.3	4.8	4.7	4.4		4.4	48	44	43	48	47			40	513	590	496	36	37	39	40	40	42	
10	irulandi	1	560	550			550	4	5	4.8	5.6	5.5			5.5	44	100	60	70	69	57		55	495	587	473	34	36	36	40	42		
11	jagan	1	540	550	510		500	4.3	5	4.8	5.4	5.5	5.1		5	43	45	44	54	48			45	527	598	498	36	38	38	39	40	40	
12	rajan	1	450	460			460	4.3	4.4	4.1	4.5	4.6			4.6	48	41	40	45	46			46	480	578	470	37	38	38	40	39		
13	muthu	1	440	430			400	3.8	4	3.9	4.4	4.3			6	42	50	49	49	43	45		40	496	600	473	36	38	38	38	38		
14	subramani	1	480	500	450		450	4.1	4.8	4.5	4.8	5	4.5		4.5	41	59	45	48	50	50		45	458	566	440	36	37	38	38	42	45	
15	fathima	1	480	500	450	500	500	4.2	4.5	4.1	4.8	5	4.5	5	5	38	50	41	48	45		45	45	438	563	420	35	37	38	38	38	36	38
16	thevar	1	480	490			490	3.8	4.4	3.7	4.8	4.9			4.9	34	73	41	53	54	41		55	475	593	460	38	40	40	42	44		
17	kannan	1	400	385	370	410	410	3.6	4	3.6	4	3.9	3.7	4.1	4.1	40	50	40	40	39		41	41	490	570	470	36	38	40	40	43	42	46
18	sundariammal	1	460	470			470	4	4.4	3.8	4.6	4.7			4.7	50	55	42	51	47			47	456	568	440	34	35	35	37	37		
19	chandra	1	500	480			420	4	4.7	4.2	5	4.8			4.2	44	59	42	45	48	48		42	473	603	458	36	38	38	37	38		
20	ganesan	1	480	490	430		440	4.4	4.8	4.3	4.8	4.9	4.3		4.4	44	60	48	60	49			49	488	594	463	38	38	39	40	42	44	

Sl. No.	name	case/contol	etcd-EOS	PR-base	PR5min	PR15min	PR30min	PR60min	PR90min	PR120min	PR-EOS	MAP-base	MAP5min	MAP15min	MAP30min	MAP60min	MAP90min	MAP120min	MAP-EOS	satn-base	satn5min	satn15min	satn30min	satn60min	satn90min	satn120min	satn-EOS	blood loss	crystalloids	colloid /blood	ephedrine
1	sudha	1	45	110	90	84	98	96			96	80	86	98	110	108			108	99	100	100	100	100			100	125	1500		
2	thangakani	1	45	74	78	83	88	85			85	76	87	82	84	90			84	100	100	100	100	100			100	200	1750		
3	senthil	1	42	94	100	97	83	74	80		80	86	88	88	106	104	96		96	99	100	100	100	100	100		100	300	2500		
4	kumar	1	42	90	94	94	90	95	100		104	80	76	84	84	97	100		100	100	100	100	100	100	100		100	150	2250		
5	jayalakshmi	1	43	93	82	72	86	84	83	105	105	76	87	85	85	95	96	98	98	100	100	100	100	100	100	100	100	600	2000	350	
6	subbaiah	1	42	68	78	76	90	96	94	98	98	96	104	106	103	100	104	108	108	100	100	100	100	100	100	100	100	75	2800		
7	vaijayanthimala	1	46	73	78	86	84	96			96	76	84	89	93	95			95	100	100	100	100	100			100	175	1800		
8	ramayee	1	43	70	76	77	88	94			94	96	104	107	110	106			106	100	100	100	100	100			100	350	1500	350	
9	rakku	1	42	80	82	74	85	86	76		76	85	90	86	90	94	99		99	100	100	100	100	100	100		100	250	2300		
10	irulandi	1	42	64	67	78	75	70			78	104	100	106	107	108			110	100	100	100	100	100			100	120	2000		
11	jagan	1	40	78	88	86	90	96	94		94	70	86	83	88	90	95		96	100	100	100	100	100	100		100	500	2000	350	
12	rajan	1	39	85	80	76	88	90			90	86	90	96	98	98			98	100	100	100	100	100			100	125	1800		
13	muthu	1	38	88	83	80	86	84			84	84	86	96	98	105			105	99	99	100	100	100			100	275	2000		
14	subramani	1	45	68	64	70	78	76	86		86	80	84	89	95	93	95		95	99	100	100	100	100	100		100	100	2000		
15	fathima	1	38	76	84	88	92	96	98	96	96	80	96	100	100	100	104	102	102	100	100	100	100	100	100	100	100	300	2500		
16	thevar	1	44	84	80	92	95	98			98	76	90	85	97	100			100	100	100	100	100	100			100	50	1500		
17	kannan	1	46	67	69	70	78	86	76	85	85	90	96	98	85	88	92	98	98	100	100	100	100	100	100	100	100	400	2500	350	
18	sundariammal	1	37	58	64	66	68	68			68	78	86	87	88	88			88	100	100	100	100	100			100	250	2000		
19	chandra	1	38	86	92	94	90	96			96	104	108	110	104	105			105	100	100	100	100	100			100	175	2500		
20	ganesan	1	44	90	100	102	104	104	108		108	86	90	92	92	98	102		102	100	100	100	100	100	100		100	200	2500		

Sl. No.	name	case/contol	age	sex	weight	duration	PEEP-base	PEEP5min	PEEP15min	PEEP30min	PEEP60min	PEEP90min	PEEP120min	PEEP-EOS	PIP-base	PIP5min	PIP15min	PIP30min	PIP60min	PIP90min	PIP120min	PIP-EOS	pl.P- base	pl. P5min	pl. P15min	pl. P30min	pl. P60min	pl. p90min	pl. P120min	pl. P-EOS	Vt-base	Vt5min	Vt15min
21	balasundaram	2	55	m	45	60	0	1	2	1	2			2	16	16	16	15	15			15	15	15	15	14	14			14	450	440	450
22	deivarani	2	40	f	46	90	0	0	1	2	1	2		2	16	15	16	16	16	16		16	15	14	14	14	15	14		15	460	480	470
23	murugan	2	44	m	50	45	0	0	0	1				1	13	13	14	14				14	12	12	13	13				13	500	510	490
24	jaya	2	39	f	48	120	0	0	1	1	1	1	1	1	14	13	13	13	14	14	14	14	12	12	12	12	13	12	12	12	480	500	500
25	rajan	2	48	m	55	120	0	1	2	2	2	2	2	2	15	16	16	16	16	15	16	16	14	14	14	14	14	14	14	14	550	540	560
26	latha	2	26	f	60	60	0	1	1	2	1			1	14	14	15	15	15			15	13	13	14	14	14			14	600	630	620
27	mani	2	50	m	57	100	0	1	1	1	2	2		2	15	16	14	15	15	15		15	12	12	12	13	13	13		13	570	580	580
28	sekaran	2	32	m	43	90	0	0	1	1	0	1		1	16	16	15	16	14	15		16	15	15	14	14	14	14		14	430	450	460
29	balan	2	48	m	49	120	0	0	1	1	2	1	2	2	15	15	15	16	16	15	16	16	14	14	14	15	15	14	15	15	490	500	480
30	jeyapandi	2	36	m	40	80	1	1	1	2	2			2	14	13	13	14	14			14	13	12	12	12	12			12	400	380	420
31	ganthi	2	34	f	52	120	1	1	1	2	2	2	2	2	14	15	15	15	14	14	15	15	13	13	13	14	13	13	13	13	520	525	530
32	joseph	2	50	m	47	45	0	1	0	0				0	13	14	14	14				14	12	12	12	12				12	470	450	480
33	bose	2	43	m	45	120	0	1	2	1	2	2	2	2	16	16	16	16	15	15	15	15	15	15	14	13	13	14	14	14	450	440	450
34	chinnammal	2	34	f	50	75	0	1	1	1	2			2	14	14	15	15	15			15	13	13	13	14	14			14	500	500	530
35	sudha	2	24	f	47	60	0	1	1	2	1			1	15	14	15	14	14			14	14	12	12	12	12			12	470	480	450
36	sivan	2	30	m	55	90	0	1	1	1	2	2		2	16	15	15	14	14	15		14	14	14	13	13	13	13		13	550	570	540
37	rajesh	2	53	m	57	100	0	1	1	1	2	2		2	15	15	14	14	14	14		14	13	13	12	12	12	12		12	570	580	600
38	palani	2	38	m	48	120	0	1	1	1	1	1	1	1	15	15	15	14	14	15	15	15	13	14	13	13	13	13	13	13	480	500	510
39	pandi	2	40	m	53	75	0	1	1	1	2			2	15	15	16	16	16			16	14	14	14	15	15			15	530	550	550
40	selvakumar	2	50	m	48	100	0	0	0	1	1	1		1	15	14	13	14	13	14		14	14	13	12	13	12	12		12	480	480	490

Sl. No.	name	case/contol	Vt30min	Vt60min	Vt90min	Vt120min	Vt-EOS	MV-base	MV5min	MV15min	MV30min	MV60min	MV90min	MV120min	MV-EOS	dync base	dync5 min	dync15min	dync30min	dync60min	dync90min	dync120min	dync-EOS	P/F-base	P/F60min	P/F-recovery	etcd base	etcd5min	etcd15min	etcd30min	etcd60min	etcd90min	etcd120min
21	balasundaram	2	460	460			460	4.5	4.4	4.5	4.6	4.6			4.6	28	29	32	33	35			35	480	523	460	35	38	34	32	34		
22	deivarani	2	450	460	450		450	4.6	4.8	4.7	4.5	4.6	4.5		4.5	29	32	31	32	31	32		32	468	510	454	30	32	32	30	34	32	
23	murugan	2	510				510	5	5.1	4.9	5.1				5.1	38	39	35	39				39	490	520	484	35	37	36	36			
24	jaya	2	470	490	490	510	510	4.8	5	5	4.7	4.9	4.9	5.1	5.1	34	38	42	39	38	38	39	39	484	524	470	38	36	36	37	36	38	36
25	rajan	2	550	560	540	560	560	5.5	5.4	5.6	5.5	5.6	5.4	5.6	5.6	37	36	40	39	40	42	40	40	498	530	478	35	34	34	32	34	33	34
26	latha	2	590	630			630	6	6.3	6.2	5.9	6.3			6.3	43	48	44	45	45			45	470	500	450	38	39	37	37	38		
27	mani	2	575	550	570		570	5.7	5.8	5.8	5.8	5.5	5.7		5.7	38	39	45	41	42	44		44	464	500	450	40	38	39	38	38	38	
28	sekaran	2	460	420	430		430	4.3	4.5	4.6	4.6	4.2	4.3		4.3	27	28	33	31	30	31		29	484	510	470	34	35	36	36	36	36	
29	balan	2	510	520	500	490	490	4.9	5	4.8	5.1	5.2	5	4.9	4.9	33	33	34	34	37	36	35	35	478	504	464	36	36	38	38	36	38	38
30	jeyapandi	2	410	420			420	4	3.8	4.2	4.1	4.2			4.2	31	32	35	34	35			35	450	502	430	34	34	35	35	36		
31	ganthi	2	520	510	520	530	530	5.2	5.3	5.3	5.2	5.1	5.2	5.3	5.3	40	38	38	40	43	43	41	41	433	490	420	40	38	39	38	38	37	38
32	joseph	2	475				490	4.7	4.5	4.8	4.8				4.9	36	35	34	34				35	388	490	360	35	36	37	37			
33	bose	2	450	440	460	440	440	4.5	4.4	4.5	4.5	4.4	4.6	4.4	4.4	28	29	32	30	34	35	34	34	510	533	450	40	38	38	38	38	38	38
34	chinnammal	2	520	520			520	5	5	5.3	5.2	5.2			5.2	36	38	38	37	40			40	476	530	454	37	38	38	39	39		
35	sudha	2	475	460			460	4.7	4.8	4.5	4.8	4.6			4.6	31	37	32	40	35			35	448	498	430	32	36	37	37	37		
36	sivan	2	530	550	560		560	5.5	5.7	5.4	5.3	5.5	5.6		5.6	34	41	39	41	46	43		47	485	525	463	35	37	37	37	37	37	
37	rajesh	2	580	600	590		590	5.7	5.8	6	5.8	6	5.9		5.9	38	41	46	45	50	49		49	456	500	444	34	36	37	38	38	35	
38	palani	2	490	500	470	490	490	5	5	5.1	4.9	5	4.7	4.9	4.9	32	36	36	38	38	34	35	35	486	514	474	36	38	37	37	38	37	38
39	pandi	2	540	560			560	5.3	5.5	5.5	5.5	5.4	5.6		5.6	35	39	37	36	40			40	476	522	466	34	36	36	35	36		
40	selvakumar	2	470	480	480		480	4.8	4.8	4.9	4.7	4.8	4.8		4.8	32	34	38	36	40	37		37	455	513	433	33	35	35	36	36	36	

Sl. No.	name	case/contol	etcd-EOS	PR-base	PR5min	PR15min	PR30min	PR60min	PR90min	PR120min	PR-EOS	MAP-base	MAP5min	MAP15min	MAP30min	MAP60min	MAP90min	MAP120min	MAP-EOS	satn-base	satn5min	satn15min	satn30min	satn60min	satn90min	satn120min	satn-EOS	blood loss	crystalloids	colloid /blood	ephedrine
21	balasundaram	2	34	110	115	110	100	100			100	96	90	80	84	88			88	99	99	99	99	99			99	300	1500		
22	deivarani	2	32	83	86	90	84	92	88		88	80	76	78	84	80	84		84	99	99	99	99	99	99		99	350	2000		
23	murugan	2	36	68	77	66	68				68	80	84	88	86				86	99	99	99	99				99	75	1250		
24	jaya	2	36	76	80	87	78	84	80	88	88	80	78	84	88	85	90	86	86	100	100	100	100	100	100	100	100	100	2800		
25	rajan	2	34	90	98	100	93	100	100	100	100	90	100	104	100	102	100	102	102	99	100	100	100	100	100	100	100	400	2500	350	
26	latha	2	38	64	68	68	74	75			78	70	80	84	86	90			90	100	100	100	100	100			100	250	2000		
27	mani	2	38	88	96	98	100	104	107		107	80	85	68	70	78	86		86	99	100	100	100	100	100		100	700	2000	700	
28	sekaran	2	36	70	67	74	76	72	80		80	70	74	66	60	74	72		72	100	100	100	100	100	100		100	200	2250		
29	balan	2	38	83	80	98	90	94	96	94	94	85	70	64	74	78	84	88	88	100	100	100	100	100	100	100	100	500	2500	350	
30	jeyapandi	2	36	70	74	77	88	90			90	74	80	84	80	76			76	99	100	100	100	100			100	125	1750		
31	ganthi	2	38	100	102	100	93	100	100	106	106	100	110	105	102	102	104	102	102	99	99	100	100	100	100	100	100	150	2500		
32	joseph	2	37	72	75	84	86				86	80	96	100	94				94	100	100	100	100				100	100	1300		
33	bose	2	38	90	96	100	105	103	104	100	100	86	84	90	92	100	102	102	102	100	100	100	100	100	100	100	100	650	2000	700	
34	chinnammal	2	39	58	63	68	66	68			68	66	60	70	74	68			68	99	100	100	100	100			100	250	1800		
35	sudha	2	37	72	76	84	73	88			88	96	90	86	84	90			90	99	99	100	100	100			100	800	2500	700	
36	sivan	2	37	63	77	70	78	75	78		78	104	100	96	96	102	108		108	100	100	100	100	100	100		100	320	2500		
37	rajesh	2	35	57	60	62	60	63	64		64	78	86	85	88	94	94		94	100	100	100	100	100	100		100	600	2500	350	
38	palani	2	38	86	80	70	74	78	82	78	78	104	100	88	96	102	104	102	102	99	99	100	100	100	100	100	100	300	2500		
39	pandi	2	36	84	78	72	86	90			90	80	86	90	84	98			98	100	100	100	100	100			100	225	2000		
40	selvakumar	2	36	94	99	93	100	92	102		102	86	90	104	80	86	94		94	100	100	100	100	100			100	200	2250		

ABBREVIATIONS USED IN THE MASTER CHART

- base – baseline values
- EOS – end of surgery
- PEEP – positive end expiratory pressure in cm of H_2O
- PIP – peak inspiratory pressure in cm of H_2O
- pl.P – plateau pressure in cm of H_2O
- Vt – exhaled tidal volume in ml
- MV – minute ventilation in litres/min
- dync – dynamic compliance in ml/cm of H_2O
- P/F base - preoperative PaO_2/FiO_2 in mm of Hg
- P/F 60 - PaO_2/FiO_2 in mm of Hg at 60min after intubation
- P/F recovery - PaO_2/FiO_2 in mm of Hg in the recovery room 30 minutes after extubation
- etcd – end tidal carbon dioxide in mm of Hg
- PR – pulse rate in beats/minute
- MAP – mean arterial pressure in mm of Hg
- satn - peripheral arterial oxygen saturation expressed as percentage

Depart of Anaesth

Ref. No. 5336 /E4/3/2012

Govt. Rajaji Hospital,
Madurai.20. Dated: .08.2012

Institutional Review Board / Independent Ethics Committee.
Dr. N. Mohan, M.S., F.I.C.S., F.A.I.S.,
Dean, Madurai Medical College & 2521021 (Secy)
Govt Rajaji Hospital, Madurai 625020.
Convenor
grhethicssecy @gmail.com.

Sub: Establishment-Govt. Rajaji Hospital, aMadurai-20-
Ethics committee-Meeting Agenda-communicated-regarding.

The Ethics Committee meeting of the Govt. Rajaji Hospital, Madurai was held at 11.00 Am to 1.00Pm on 28.06.2012 at the Dean Chamber, Govt. Rajaji Hospital, Madurai. The following members of the committee have been attended the meeting.

---

1. Dr.N.Vijayasankaran,M.ch(Uro.) 094-430-58793 0452-2584397	Sr.Consultant Urologist Madurai Kidney Centre, Sivagangai Road,Madurai	Chairman
2. Dr.P.K. Muthu Kumarasamy, M.D., 9843050911	Professor & H.O.D of Medical, Oncology(Retired)	Member Secretary
3. Dr.T.Meena,MD 094-437-74875	Professor of Physiology, Madurai Medical College	Member
4. Dr. S. Thamilarasi, M.D (Pharmacol)	Professor of pharmacology	
5.Dr.Moses K.Daniel MD(Gen.Medicine) 098-421-56066	Professor of Medicine Madurai Medical College	Member
6.Dr.M.Gobinath,MS(Gen.Surgery)	Professor of Surgery Madurai Medical College	Member
7.Dr.S. Dilshadh, MD(O&G) 9894053516	Professor of OP&Gyn Madurai Medical College	Member
8.Dr.S.Vadivel Murugan., M.D, 097-871-50040	Professor of Medicine Madurai Medical College	Member
9.Shri.M.Sridher,B.sc.B.L. 099-949-07400	Advocate, 2, Deputy collectors colony 4th street KK Nagar, Madurai-20.	Member
10.Shri.O.B.D.Bharat,B.sc., 094-437-14162	Businessman Plot No.588, K.K.Nagar,Madurai.20.	Member
11.Shri. S.sivakumar,M.A(Social) Mphil 093-444-84990	Sociologist, Plot No.51 F.F, K.K Nagar, Madurai.	Member

Following Projects were approved by the committee

for info to Dr. Ramesh

21/8/12

Sl. No	Name of P.G.	Course	Name of the Project	Remarks
1.	Dr. Ramakani. K	M.D Anaesth	Does alveolar recruitment by PEEP during intra-operative mechanical ventilation improve arterial oxygenation?.	Approved

Please note that the investigator should adhere the following: She/He should get a detailed informed consent from the patients/participants and maintain Confidentially.

1. She/He should carry out the work without detrimental to regular activities as well as without extra expenditure to the institution to Government.
2. She/He should inform the institution Ethical Committee in case of any change of study procedure site and investigation or guide.
3. She/He should not deviate for the area of the work for which applied for Ethical clearance.

She/He should inform the IEC immediately, in case of any adverse events pr Serious adverse reactions.

4. She/he should abide to the rules and regulations of the institution.
5. She/He should complete the work within the specific period and apply for if any Extension of time is required She should apply for permission again and do the work.
6. She/He should submit the summary of the work to the Ethical Committee on Completion of the work.
7. She/He should not claim any funds from the institution while doing the word or on completion.
8. She/He should understand that the members of IEC have the right to monitor the work with prior intimation.

17.8.12

DEAN

To
All the above members and Head of the Departments concerned.
All the Applicants.

DIRECTOR
INSTITUTE OF ANAESTHESIOLOGY
Madurai Medical College &
Govt. Rajaji Hospital
MADURAI-625020.

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E-mail	dr_rrk@yahoo.in
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EVALUATION OF EFFECT OF ALVEOLAR RECRUITMENT MANEUVER DURING INTRAOPERATIVE MECHANICAL VENTILATION DISSERTATION SUBMITTED FOR DOCTOR OF MEDICINE BRANCH X (ANAESTHESIOLOGY) APRIL 2013 THE TAMILNADU DR.M.G.R.MEDICAL UNIVERSITY CHENNAI BONAFIDE CERTIFICATE This is to certify that this dissertation titled “EVALUATION OF EFFECT OF ALVEOLAR RECRUITMENT MANEUVER DURING INTRAOPERATIVE MECHANICAL VENTILATION” is a bonafide record work done by Dr. RAMA RANI K. under my direct supervision and guidance, submitted to the Tamil Nadu Dr. M.G.R. Medical University in partial fulfillment of University regulation for MD, Branch X - Anaesthesiology. PROF. Dr. S .C. GANESH PRABHU, M.D. D.A. Director, Institute...

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