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Review Article

Peri-Operative Atelectasis and Alveolar Recruitment Manoeuvres

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ABSTRACT

Respiratory complications are a significant cause of postoperative morbidity and mortality. Perioperative atelectasis, in particular, affects 90% of surgical patients and its effects can be prolonged due to changes in respiratory mechanics, pulmonary circulation and hypoxaemia. Alveolar collapse is caused by certain predisposing factors, mainly due to compression and absorption mechanisms. Several therapeutic strategies have been proposed to prevent or treat this atelectasis, such as alveolar recruitment manoeuvres, which has become widely used in the last few years. Its application in patients with alveolar collapse, but without a previous significant acute lung lesion has some unusual features. Its use is, therefore, not free of uncertainties and complications. This review describes the frequency, pathophysiology, importance and treatment of perioperative atelectasis. Special attention is paid to treatment with recruitment manoeuvres, so as to provide a basis for their rational and appropriate use.

Atelectasias perioperatorias y maniobras de reclutamiento alveolar

Palabras clave:
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Período postoperatorio
Complicaciones postoperatorias

RESUMEN

Las complicaciones respiratorias postoperatorias constituyen una causa importante de morbimortalidad. Las atelectasias perioperatorias, en concreto, afectan hasta al 90% de los pacientes quirúrgicos y su repercusión puede prolongarse en el tiempo en forma de alteraciones de la mecánica respiratoria, de la circulación pulmonar y de hipoxemia. El colapso alveolar se produce en presencia de ciertos factores predisponentes, fundamentalmente por mecanismos de compresión y absorción. Para prevenir o tratar estas atelectasias, se han propuesto diversas estrategias terapéuticas, como las maniobras de reclutamiento alveolar, cuyo uso se ha popularizado en los últimos años. Su aplicación en pacientes con colapso alveolar, pero sin lesión pulmonar aguda previa relevante, presenta ciertas particularidades, por lo que su empleo no está exento de incertidumbres y complicaciones. Esta revisión describe la frecuencia, la fisiopatología, la relevancia y el tratamiento de las atelectasias perioperatorias, y hace especial incidencia en el tratamiento con maniobras de reclutamiento con el objetivo de proporcionar las bases para un empleo racional y adecuado de éstas.

Introduction

About 2-4% of elective thoracic or abdominal operations are associated with post-operative pulmonary complications and the percentage increases to 20% when surgery is urgent.[1] The development of atelectasis is one of the most common respiratory complications during the peri-operative period,[2,3] and it affects nearly 90% of patients.[4-6] In general, they are of little relevance, as the vast majority disappear within the first 24 h after an intervention,[7,8] but in certain circumstances these atelectases persist for days after surgery.[5,9-11] The impression of clinicians is that they may contribute to the development of other respiratory complications[3] and increase the morbidity of surgical patients, especially those who are obese or have undergone cardiothoracic and abdominal surgery[12] although this progression has not been proven conclusively.[9,13] Furthermore, atelectasis and pulmonary oedema underlie 15% of patient readmissions to critical care units.[14]

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In the 1960s it was observed that anaesthetised patients showed reduced respiratory system compliance, which was accompanied by a deterioration in oxygenation. It was proposed that the formation of atelectasis could be the cause of this phenomenon.[15] However, conventional radiology was unable to demonstrate this hypothesis.[2] Studies conducted in the 1980s using computerised tomography showed an increase in the density of the dorsal and caudal lung regions, which developed very rapidly following anaesthetic induction.[16-18] These areas accounted for 5% of the pulmonary surface, but, owing to the greater volume of the lung when it is aerated[18-20] they could correspond to 15-20% of pulmonary volume.[1,16,18]

The administration of an intravenous contrast agent revealed an increase in uptake in these hyperdense regions, indicating that they were perfused but not ventilated.[17] When the patient changed from a supine decubitus to a lateral position, the densities persisted in the dorsal region of the lower lung.[21] The explanation for this phenomenon is the development of atelectasis[17] which are distributed from a lesser to a higher degree from the upper to the lower lung regions.[22,23] Furthermore, a pulmonary portion similar to the part affected by atelectasis, right on top of the latter, appears to be poorly aerated.[16] Thus, during anaesthesia, ventilation predominates in the ventral lung regions as opposed to the dorsal regions.[16]

Compared to conscious patients, in patients who were anaesthetised the analysis of successive radiological slices close to the base of the lung showed an increase in the visualisation of the most cranial portion of the diaphragm and indicated a cephalic displacement of the muscle of the diaphragm, which was indicative of a reduction in lung volume.[24] Finally, using the multiple inert gas elimination technique, it was possible to observe the existence of a left-right *shunt* fraction in anaesthesised patients which represented 1-17% of cardiac output and this proportion correlated linearly with the radiological areas of increased density.[21,22,24]

Physiopathology of Alveolar Collapse during Anaesthesia

The causes of atelectasis in the peri-operative period have not been fully clarified, but the most plausible mechanisms are 3 in number[2,23,25,26] and they interact simultaneously in vivo:

a. *Compression:* as a result of the strain exerted on the alveolus, which increases pleural pressure (Ppl) and exceeds the transpulmonary pressure (TPP) which keeps it open (*TPP = alveolar pressure [Palv]-Ppl*).[6,27]
   During general anaesthesia the properties of the wall of the thorax and the lung undergo changes and this leads to a reduction in lung compliance and residual functional capacity (RFC). The decubitus position reduces RFC by about 1l with respect to a standing position and about 0.4l more when anaesthesia is induced.[28] With these low lung volumes approaching residual volume, a collapse of the small airways is produced during expiration in the lower parts of the lung,[29] as these airways which are less than 1mm in diameter have no cartilage to keep them firm.[30] During mechanical ventilation it is difficult to reopen these airways.[31]
   An artificially induced reduction in RFC does not lead to the formation of atelectasis if the diaphragm remains active.[32] Consequently, the loss of the muscle tone of the diaphragm appears to be a crucial factor in the formation of atelectasis. In the patient who is awake, the dorsal portion of the diaphragm is the part which is most effective during its contraction, while during anaesthesia it is the ventral portion.[2,22] Muscular block permits the transmission of abdominal pressure[2,33] which in a supine position is greater than intrathoracic pressure[34,35] especially in the lower regions.[34] This results in a cephalic displacement of the most dorsal portion of the diaphragm[28] and an increase in Ppl in the lower lung portions[34] which are the ones with the greatest surface area affected by atelectasis.[36]
   Other factors which contribute to the compression mechanism are the loss of intercostal muscle tone, the increase in abdominal blood caused by positive thoracic pressure as a result of mechanical ventilation, the weight of the heart, the increase in the vertical Ppl gradient and the reduction in the transversal diameter of the thorax.[13,17,37]
b. *Absorption of alveolar gas:* in turn, this can occur in two ways:
   - Complete occlusion of the small airways, which leaves a pocket of trapped distal gas that gradually collapses because the mixed venous blood passing through the lung capillaries continues to imbibe oxygen as a result of the diffusion gradient.
   - Atelectasis in areas with a low ventilation-perfusion ratio (V/Q), for example, in the case of very narrow airways. When the V/Q coefficient is reduced, a point is reached in which the gas which enters the alveolus and the gas that is absorbed from capillaries reach a state of balance. Below this critical V/Q ratio the alveolus tends to collapse.[19] It is typical of patients who have a very low mixed venous saturation or patients in whom a highly soluble mixture of gases is administered into the bloodstream (high inspired oxygen fraction [FiO_2], anaesthesia with nitrous oxygen). Mathematical models indicate that when we breathe a gas mixture with an FiO_2 of 0.3 it takes hours for atelectasis to develop as a result of absorption and that this period is reduced to 8 min when we breathe a FiO_2 of 1 or even less if the mixture is rich in nitrous oxide.[38-40]
c. *Alteration of the surface-active agent*: the surface-active agent acts by reducing the surface tension of the alveolus and stabilising it. Once an atelectasis has formed, production of the surface-active agent tends to be reduced, which facilitates the tendency for the alveolus to collapse and, even if it reopens, it will be more unstable. The repeated opening and closing of alveoli and general anaesthesia with mechanical ventilation lead to a deactivation of the surface-active agent, owing to the compression of the film which forms and its elimination from the alveolus to the small airways.[20,41] The reduction in the surface-active agent produces an increase in surface tension at the local level and an overall reduction in RFC,[13] as well as an increase in the permeability of the alveoloendothelial barrier. However, as the surface-active agent has a long half-life and it is replaced in 14 h, this mechanism for producing atelectasis is believed to be less important during anaesthesia but that it may have a greater role in the case of patients who receive prolonged mechanical ventilation.[42]

Factors which favour the Development of Atelectasis

a. *High FiO_2:* the use of a high FiO_2, both during anaesthetic induction and during surgery or prior to extubation favours the development of atelectasis and *shunt*, and can impair gaseous exchange.[23,43-45]
b. *Selection of tidal volume and ventilation parameters:* in patients with acute respiratory distress syndrome (ARDS) the use of low tidal volumes is recommended, but, in the absence of pulmonary lesions, this strategy could encourage an increase in atelectasis, especially if PEEP (positive end expiratory pressure) is not employed.[37]
c. *Obesity:* although it has only been possible to establish a weak linear correlation between body mass index and the development of atelectasis,[46] RFC is lower in obese patients and abdominal pressure is greater.[7] Both factors promote alveolar collapse. A predominance of atelectasis has been demonstrated in obese surgical populations in comparison with patients who are not overweight[7] and their impact is greater, owing to the presence of fewer reserves for hypoxaemia, so obese people constitute a group of patients in which collapse prevention is very important.[2]

d. *Type of anaesthesia:* atelectases are produced following the induction of anaesthesia and not in the patient who is awake, in the case of both inhalatory and intravenous anaesthetics, except ketamine.[47] In general, at high doses all anaesthetics attenuate the activity of the respiratory muscles,[22] but, even at low doses, they can cause respiratory depression and impair the neurological regulation of the respiratory muscles.

e. *Chronic obstructive pulmonary disease:* this patient group develops few atelectases and little *shunt*[24] but a greater deterioration of the V/Q ratio.[2,48] The mechanism for preventing atelectasis in these patients has not been entirely elucidated.[20] Hyperinsufflation tends to avoid compressive alveolar collapse,[47,48] which minimises the fall in RFC; however, it can encourage the development of absorption atelectasis.[47] Another possible explanation is a change in the balance between the wall of the thorax and the lung (resulting in little elastic recoil), which counteracts the reduction in lung volumes.[20,48]

f. *Age:* it does not appear to be a key factor,[49] although in children atelectases are very common because they present a lower RFC, greater compliance of the thoracic cage and paradoxical movements of the ribcage in response to the contraction of the diaphragm, as well as having a greater closure volume.[50] The application of 5cm H_2O PEEP is capable of recruiting alveolar units in children and of promoting the disappearance of atelectatic areas. As age increases, there are more cases of premature closure of the small airways and an increase in areas with a low V/Q ratio,[34] which impedes the formation of compression atelectasis, but could predispose patients to atelectasis formed by absorption.[5]

g. *Type of surgery:* atelectasis seem to reach a maximum level during the first minutes of anaesthesia, irrespective of the type of surgery.[29] However, surgical trauma stimulates reflexes which, through the mediation of the somatic and visceral nerves, elicit inhibition of the phrenic and other nerves that inervate the respiratory muscles. Furthermore, the muscular disruption caused by surgery impairs the efficacy of respiratory movements and pain produces a voluntary limitation on their use. All these factors result in hypoventilation and lead to a reduction in RFC of up to 20% after abdominal surgery, as well as facilitating the development of atelectasis.[9,51]

During laparoscopy the insufflation of the pneumoperitoneum with CO_2 at pressures of 11-13mmHg increases the development of atelectasis.[52,53] Other interventions which predispose patients to developing atelectasis are thoracic surgery, given that during thoracic interventions there is compression of the dependent lung, an increased tendency for secretions to be produced and greater airway reactivity,[54] and cardiac surgery with a cardiopulmonary *by-pass,*[31,55] in which *shunt* and hypoxaemia are usually produced in association with atelectasis and facilitated by the increase in capillary permeability and alveolar oedema, which increases extravascular pulmonary water and the weight of the lung.[56] Pleural distension, mediastinic drainage, gastric distension and transitory or permanent damage to the phrenic nerve[11,55,57,58] also play a role. Patients who have operations without extracorporeal circulation appear to show smaller increases in the *shunt* fraction[31] and deterioration in compliance or oxygenation.[59]

Consequences of Atelectasis

a. *Reduction in compliance:* as a result of lung volume reduction, which leads to an impairment of pulmonary mechanics.[60] The respiratory cycles begin with a lower RFC, the respiratory system functions in a less efficient sector of the pressure-volume curve and energy consumption is greater.[2,60]

b. *Deterioration of oxygenation:* during anaesthesia the presence of *shunt* is constant and it correlates with the amount of atelectasis.[22] There is also a very good correlation between the lung surface affected by atelectasis, *shunt* and the development of hypoxaemia.[22] Although atelectasis and small airway closure are the main contributers to hypoxaemia in surgical patients,[1,29,61] they also contribute to hypoventilation, hypovolaemia, low output, anaemia and changes in the V/Q ratio.[2]

c. *Increase in pulmonary vascular resistance (PVR):* the relationship between lung volume and PVR follows a "U" curve pattern, in which resistances are lower when lung volume is equal to RFC, especially when the latter is normal.[62] Regional hypoxia which develops in atelectatic areas produces an increase in local PVR,[13] facilitated by hypoxic pulmonary vasoconstriction, which is activated when there is a reduction in alveolar PaO_2 and in venous mixed blood, and this contributes to an increase in pulmonary vascular pressure, right ventricular failure and the extravasation of fluid at the microvascular level, even in previously healthy patients. Experimental studies show an increase in capillary permeability in non-recruited lungs, as well as a higher degree of right ventricular dysfunction.[62]

d. *Exacerbation of lung damage:* when there is repeated opening and closure of the alveolus, pulmonary damage occurs, not only in the atelectic area but throughout the entire lung. The greater the amount of tissue affected by atelectasis, the smaller the portion of the lung which must adapt to the tidal volume that is administered, which encourages the development of hyperinsufflation in healthy areas of the lung,[37] together with activation of the inflammatory response and a reduction in the functionality of the surface-active agent.[41,63] The lung injury mechanism elicited by atelectasis has been given the name "atelectrauma".[13] This pulmonary lesion seems to elicit greater structural damage and inflammation than lesions which are not accompanied by atelectasis.[64]

e. *Post-operative infection:* in clinical terms it has not been possible to demonstrate a direct link between the development of peri-operative atelectasis and respiratory infection, although at the experimental level lowering atelectasis reduces the development of pneumonia and translocation towards the blood following the instillation of intratracheal bacterial colonies.[65] In addition, the presence of atelectasis reduces the penetration of antibiotics into the lung, which makes it difficult to obtain the right drug concentrations to fight pathogens.[66]

Prevention and treatment

The prevention of atelectasis in the peri-operative period increases oxygen reserves.[2] As we have explained, the development of peri-operative atelectasis begins during anaesthetic induction so we should adopt a series of measures aimed at reducing their formation or reversing them if they have already been produced:

- Maintenance of *spontaneous ventilation* can contribute to lung tissue recruitment, as it eliminates the negative effects of the loss of diaphragmatic tone[29] and reduces the arterial alveolar oxygen gradient ($\Delta P_{A?a}O_2$)[22] although this strategy is not possible in many procedures.
- *Preoxygenation with FiO_2 lower than 1.* Preoxygenation prior to anaesthetic induction is normally achieved by making the patient inhale fresh gas with a FiO_2 of 1. These high oxygen concentrations and denitrogenisation foster the development of atelectasis.[23,45,67] The mechanism is potentiated in anaesthetised patients as a result of the usual fall in RFC. Moreover, pure oxygen may abolish hypoxic pulmonary vasoconstriction, resulting in the maintenance of or even an increase in blood flow to areas with atelectasis. The employment of lower oxygen concentrations may reduce the formation of atelectasis, but it also reduces the safety margin for the development of hypoxaemia, so preoxygenation with an FiO_2

lower than 1 cannot be recommended in patients suspected of having difficult airways or with a reduced RFC and limited oxygen reserves, for example pregnant women who are due to give birth, or patients who are obese or have abdominal distension.[67] For other patients some authors propose a reduction of FiO_2 during preoxygenation[23] to levels of 0.6-0.8.[19,23,68]

During operations the lowering of FiO_2 to 0.3-0.4, which increases if arterial oxygenation is impaired[19,34] may reduce atelectasis formed by absorption[44,45] although the risk of the formation of atelectasis must be weighed against the positive effects of a high FiO_2 on post-operative nausea and vomiting, and surgical wound infection.[37,69] If a high FiO_2 is used, the addition of PEEP is recommended.

For extubation the criterion for employing the oxygen mixture should be similar to the criterion which is followed for preoxygenation. Unfortunately, the patients who are most predisposed to developing atelectasis in response to a high FiO2 are the same ones who present lower oxygenation reserves.19 During the post-operative phase there is one study that shows no differences in the production of atelectasis when oxygen is administered with a FiO_2 of 0.3 or 0.8.[69]

- *Administration of continuous positive airway pressure (CPAP) or PEEP during induction*. The use of continuous positive airway pressure at 5-6cm H_2O during anaesthetic induction prior to orotracheal intubation reduces intraoperative atelectasis and improves oxygenation by increasing lung volume and oxygen reserves, without increasing dead space.[70] In obese patients the required pressures are about 10cm of H_2O.[71] Furthermore, CPAP or non-invasive ventilation appear to improve the efficacy of preoxygenation[72,73] and increase the safety margin in cases of apnoea.[71,74,75] The risk of insufflation of the digestive tract and regurgitation occur at pressures above 20mmHg,[70] although, in the case of patients with delayed gastric emptying, we should use this resource with precaution, owing to the risk of bronchoaspiration.[71] Overdistension of the lungs may occur at PEEP H_2O levels over 10-15cm but not at lower levels.[70,75]

  During the cardiopulmonary by-pass period of cardiac surgery, the application of a 5 cm H_2O CPAP does not reduce the appearance of atelectasis and is even associated with a deterioration in cardiac output after the by-pass period,[76] however, the use of 10cm H_2O CPAP can reduce shunt and improve the gaseous exchange evaluated after the by-pass and even in the post-operative phase.[77] However, sometimes these pressure levels cannot be applied because they may interfere with surgical manoeuvres.
- Use of *optimal ventilatory parameters*. The association of low tidal volume+moderate/high PEEP seems more favourable in terms of preventing collapse than a high volume associated with a low PEEP.[63] A 6ml/kg tidal volume does not produce an increase in peri-operative atelectasis or gasometric deterioration with respect to a more conventional ventilatory strategy,[6] but volumes greater than or equivalent to 10ml/kg body weight are associated with hyperinsufflation.[78] A good option appears to be the association of a tidal volume of around 8-10ml/kg body weight and PEEP.[79] During one-lung ventilation, a tidal volume below or equivalent to 8ml/kg together with a 4-10cm H_2O PEEP, with a limitation of plateau pressure to 35cm H_2O and supported by regular recruitments, appears to be associated with less development of atelectasis following surgery.[79]

  The use of PEEP increases RFC, redistributes extravascular lung water, improves the V/Q ratio and contributes to avoiding atelectasis formation. Its use during invasive ventilation has recently been linked to a reduction in the incidence of pneumonia associated with mechanical ventilation.[80] However, PEEP does not appear to promote the release of surface-active agent on its own merits[20] and it does not usually produce a full lung re-expansion,[58,81,82] so its isolated effect would be useful for keeping small airways open, but alveolar distension requires greater pressure, which can be achieved by means of recruitment manoeuvres.[83] Moreover, the effect of PEEP disappears rapidly after its withdrawal,[17,20] which facilitates alveolar collapse,[35] on a par with the level prior to its application.[29]

  In addition, the PEEP which is administered may not be distributed homogeneously so it may predominate in the alveoli which are already ventilated and this can cause their overdistension. This phenomenon would redistribute the blood flow from ventral regions towards dorsal regions and would maintain *shunt* in poorly ventilated areas, as well as impairing venous return and cardiac output.[22]
- *Recruitment manoeuvres.* Once atelectasis have been produced, we can employ these techniques, the aim of which is to elicit an increase in sustained TPP in order to re-expand collapsed alveoli.[82] Recruitments increase the release of surface-active agent and can restore alveolar stability and reduce injury induced by mechanical ventilation.[84] However, despite their efficacy and the odd study indicating that they have a lasting effect,[85] their effectiveness seems to be partial[59] or transitory[31,56] and it has not been proved that they reduce post-operative pulmonary complications nor that they improve the prognosis of patients[12] or hospital stay.[55] Some authors indicate that recruitments only manage to convert collapsed areas in regions with a low V/Q ratio.[38,86]

  Manoeuvre efficacy depends on a range of factors: ventilator adjustments (pressure, time and FiO_2),[37,38] the pulmonary situation of the patient[87-89] (which makes the amount of recruitable tissue variable)[90] and his tolerance to increased thoracic pressures. Therefore, manoeuvres must be tailored to the needs of individual patients.[91,92]

  Pressures: recruitment occurs along the inspiratory branch of the pressure-volume curve[25,93] up until its highest inflection point.[63] It is estimated that it reaches its peak at a TPP of 20-25cm H_2O and it seems that the more tissue is recruited during inspiration, the more will remain recruited at the end of expiration.[94] The TPP required for distension depends on Ppl (*TPP = Palv–Ppl*) and, therefore, it must be greater in cases of reduction of thoracic cage compliance.[63] A significant distension of the collapsed lung surface in a healthy lung begins at an inspiratory pressure (IP) of about 30cm H_2O; it is not uniform for the entire lung, a greater pressure being required in lower or dependent regions,[25] and it is not complete until 40cm H_2O.[34,46] In patients with ARDS higher pressures may be needed[95] (up to 60-70cm H_2O), given that in these patients there is greater surface-active agent dysfunction[41] and alveolar oedema.[58]

  Once opened, the pressure required to avoid alveolar collapse is less than that required to open the alveolus.[12] The application of PEEP after recruitment stabilises the pulmonary units and prevents reocclusion,[58] while recruitments without subsequent PEEP application have a transitory effect.[27] As a result, after re-expansion, PEEP must be used to help to prevent the reappearance of atelectasis.[87,93,96]

  Optimal PEEP would be that which manages to keep the alveoli open without overdistending ventilated areas. It has been commented that the lowest inflection point on the pressure-volume curve only indicates the beginning of recruitment[93] and that PEEP must be adapted to each patient and each lung condition.[81] An alternative way of determining optimal PEEP is by lowering it step by step following recruitment[96,97] and evaluating various respiratory parameters (RFC, compliance, PaO_2, $PaCO_2$ and alveolar dead space).[97,98] Based on these premises, it has been indicated that the appropriate PEEP after recruitment in a non-obese population of patients, who have undergone surgery under a general anaesthetic, is 10cm H_2O[97] and that in obese patients a PEEP of 10cm H_2O is more effective than 5cm H_2O to maintain oxygenation.[99] The need to repeat a manoeuvre may be a sign that the PEEP applied after the manoeuvre is insufficient.[37]

Indications and frequency of repetition: the use of recruitments is indicated in patients who experience a deterioration in gaseous exchange during general anaesthesia, especially if a high FiO_2 is being used[27] or in the case of a low previous PEEP.[87] It would also seem advisable to perform recruitment, in accordance with the physiopathology of the atelectasis, after the induction of general anaesthesia, after any manoeuvre involving the disconnection of ventilation or tracheal suction,[41] before extubation and on admittance to the post-operative care unit if the patient is mechanically ventilated when he is transferred to the unit,[41] although, owing to a lack of studies, the level of current evidence is classed as "recommendations by experts".[25] Despite the fact that the repetition of manoeuvres every 6 h does not appear to produce histological pulmonary injury,[36] the repetition of recruitments without the application of an appropriate PEEP can contribute to pulmonary injury,[84] as it promotes damage to the alveolocapillary barrier.

Determination of efficacy: various parameters have been used for this purpose: the increase in the PaO_2/FiO_2 ratio,[58,83,100] the decrease in $\Delta P_{A-a}O_2$,[85] the effect on lung compliance,[58,83,100] the effect on end-expiratory lung volume[58,83] or the improvement in parameters derived from volumetric capnograms.[83,101] The combination of an increase in RFC and a reduction in dead space enables maximum efficacy, in terms of alveolar opening without overdistension, to be determined,[97] although the need for access to specific monitors limits the application of these measurements in daily clinical practice. At the bedside, the most widely used parameters tend to be an improvement in the PaO_2/FiO_2 ratio, $\Delta P_{A-a}O_2$ and compliance, without deterioration in $PaCO_2$.[85]

Target populations: the effectiveness of recruitment manoeuvres has been proved to reduce *shunt* or atelectasis in a range of diseases and surgical procedures, for example during one-lung ventilation,[54,101-103] following cardiopulmonary *by-pass* as part of cardiac surgery,[56,86] and in obese populations during bariatric[100] or general abdominal surgery.[99]

Complications: although the safety of these manoeuvres is endorsed by the large number of patients to whom they have been applied with no evidence of complications,[12,36,100] they have been questioned, owing to the possible haemodynamic damage they may cause, given that they have been linked with transitory reductions in venous return, preload,[104] blood pressure,[89,104] heart rate[104] and cardiac output,[81,103-105] especially in patients with right ventricular dysfunction and severe pulmonary hypertension, and in patients whose Palv is readily transmitted to the mediastinum.[25] The haemodynamic impact seems to be smaller in patients who respond to a manoeuvre by showing an improvement in oxygenation than in those who fail to respond.[106]

One or two studies indicate an absence of relevant haemodynamic effects when patients are properly hydrated[54,107], although central venous pressure is not a reliable reflection of preload during recruitment and it may increase simply as a result of the transmission of intrathoracic pressure.[81] Often a transitory reduction in oxygenation is observed after a recruitment is performed. This is due to the return of desaturated "stored" blood from peripheral circulation, in addition to haemodynamic deterioration and the shunting of bloodflow to hyperinsufflated areas.[12,108]

Experimental models indicate that recruitments have more deleterious effects in situations of sepsis[89] and lower efficacy in pneumonia models.[87] In addition, manoeuvres are not advised in patients with intracranial hypertension,[37] as they can cause barotrauma[25] and, in general, they must be administered with prudence because high TPPs, such as those which may be elicited in some forms of intensive recruitment, have been linked with disruption of the alveolocapillary barrier, the release of alveolar cytokines into the blood and interstitial pulmonary oedema.[96]

Forms of recruitment: during anaesthesia and surgery, in patients with no pulmonary disease, the most widely used forms are modifications of the following 2 manoeuvres:

- The vital capacity manoeuvre (administration of a continuous 40cm H_2O IP for 15 s) facilitates virtually complete distension of the non-pathological lung and thus corrects any atelectasis which are produced during induction or after the cardiopulmonary *by-pass* period.[12,57,59,86,109] Although most studies indicate that there are few haemodynamic consequences as a result of this manoeuvre,[12] continuous sustained pressure appears to provoke greater haemodynamic instability than other recruitment alternatives.[55,104] This is why some authors reduce it to 7-8 s to minimise adverse effects and its efficacy is similar, as it is in the initial phase of recruitment that the greatest distension of atelectasis is produced.[110] In healthy lungs the prolongation of the manoeuvre would not guarantee complete alveolar distension.[29,111] When conducted during pressure-controlled ventilation, the manoeuvre appears to be more effective than if it is performed manually[111] and it lasts longer if it is performed with a reduced FiO_2.[25] In ARDS patients the manoeuvre is usually applied for longer[106] or using higher pressures,[112] as it is more difficult for atelectasis to re-expend themselves and there is less transmission of Palv to the mediastinum as a result of the reduction in pulmonary compliance.
- An alternative, during pressure-controlled ventilation, consists of a step-by-step increase in IP and PEEP every 2-3 respiratory cycles, maintaining a constant differential pressure (IP-PEEP) of 20-25cm H_2O until a peak IP of 40 and a PEEP of 20cm H_2O is reached. This is maintained for about 1 min, followed by a reduction, which is also progressive, of the pressures until finally the optimal PEEP is attained.[54,82,83,95,99-102] This manoeuvre appears to be associated with less haemodynamic deterioration than its vital capacity counterpart.[92] It is usually performed with inspiration-expiration ratios from 1:1 to 1:1.5 and respiratory frequencies of 10-12 cycles per min.

In children the lungs are less developed. The diameter of the airways and the alveoli is smaller, the ribcage is highly distensible and there is little gravitational effect on the dependent part of the lung. However, atelectases during anaesthesia are distributed in a similar way in adults and children.[113] The general principles of recruitment can be applied to this population, although the IP which is needed seems to be somewhat lower than in adults (about 25-30cm of H_2O).[114] Higher pressures (40cm of H_2O) have been employed without notable adverse effects.[113]

- In some studies the *efficacy of non-invasive mechanical ventilation during the post-operative period*,[57] as well as intensive physiotherapy[31] and early mobilisation,[7] have been demonstrated, but this is not the case for incentivated spirometry.[115] CPAP at about 10cm H_2O can re-establish RFC and reduce atelectasis and the incidence of hypoxaemia by increasing intrathoracic pressure and reducing respiratory effort.[57] The application of CPAP in patients who develop hypoxaemia following abdominal surgery reduces the incidence of atelectasis,[116] the need for reintubation and the incidence of respiratory infections.[117] In another study nasal CPAP at 10cm H_2O reduces respiratory complications (including atelectasis) following aortic aneurysm surgery.[118] Finally, the use of non-invasive ventilation with pressure support appears to reduce the incidence of atelectasis with respect to the use of continuous positive pressure.[119]
- *Appropriate analgesia* contributes to the reduction of atelectasis,[120] as it can increase lung volumes and vital capacity and improve indexes which have been interpreted as a reflection of diaphragmatic activity.[9,51] Regional epidural techniques combined with local anaesthetics might be beneficial, although the studies on this are inconclusive,[51,121] given that, on the one hand, the results are variable, depending on how pulmonary complications are

defined, and, on the other, this type of analgesia could block abdominal and intercostal muscles, which also contribute to ventilation. Systemic opiates can increase the pressure generated by the abdominal muscles, whose activity has been linked to a reduction in lung volume,[122] while epidural analgesia can improve this situation.[123]

Conclusions

Most patients who undergo a surgical intervention under a general anaesthetic develop atelectasis in the lower areas of the lung, the consequences of which have been demonstrated. Although these are usually limited, it is important to understand the mechanism underlying the formation of these atelectases in order to implement the right therapeutic strategies, which will enable their formation to be minimised and thus reduce their contribution to the development of peri-operative complications as far as possible.

Correctly performed recruitment manoeuvres can contribute to improving respiratory mechanics and gaseous exchange in many patients who present atelectasis during general anaesthesia, but, before recommending their generalised and systematic use with total confidence, conclusive studies are needed to demonstrate the moment, the frequency and the optimal mode of recruitment and to show whether the improvement in intermediate parameters of oxygenation and lung mechanics translates into a reduction of post-operative complications and a final prognostic benefit to patients.

Conflict of Interest

The author declares that he has no conflict of interest.

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