



International Journal of Medical Anesthesiology

E-ISSN: 2664-3774
P-ISSN: 2664-3766
www.anesthesiologypaper.com
IJMA 2024; 7(2): 51-59
Received: 18-03-2024
Accepted: 23-04-2024

Mina Morkos Nasr
Department of Management,
Faculty of Medicine,
Anesthesiology & Surgical
Intensive Care and Pain,
Tanta University, Tanta,
Egypt

Tarek Abdel Hay Mostafa
Department of Management,
Faculty of Medicine,
Anesthesiology & Surgical
Intensive Care and Pain,
Tanta University, Tanta,
Egypt

Nagat Sayed Elshmaa
Department of Management,
Faculty of Medicine,
Anesthesiology & Surgical
Intensive Care and Pain,
Tanta University, Tanta,
Egypt

Hoda Alsaid Ezz
Department of Management,
Faculty of Medicine,
Anesthesiology & Surgical
Intensive Care and Pain,
Tanta University, Tanta,
Egypt

Corresponding Author:
Mina Morkos Nasr
Department of Management,
Faculty of Medicine,
Anesthesiology & Surgical
Intensive Care and Pain,
Tanta University, Tanta,
Egypt

Ultrasound guided lung recruitment maneuvers and prevention of postoperative atelectasis after pediatric laparoscopic abdominal surgery

Mina Morkos Nasr, Tarek Abdel Hay Mostafa, Nagat Sayed Elshmaa and Hoda Alsaid Ezz

DOI: <https://doi.org/10.33545/26643766.2024.v7.i2a.476>

Abstract

One of the most prevalent consequences of mechanical ventilation that might lead to pulmonary issues after surgery is atelectasis. The use of lung ultrasound (LUS) to diagnose a variety of pulmonary disorders in both children and adults has been steadily increasing due to its lack of radiation and lack of invasiveness. There is growing evidence that perioperative treatment, in addition to emergency and critical care settings, may benefit from LUS. Pediatric patients who have had anesthesia-induced atelectasis have been diagnosed using LUS. By increasing the number of alveoli in the dependent portion of the lungs that participate in gas exchange, the lung recruitment procedure improves gas exchange in pulmonary capillaries, enhances lung compliance, and decreases intrapulmonary shunt. In order to increase lung aeration and decrease the incidence of post-operative atelectasis, ultrasound-guided lung recruitment techniques are a safe and effective option.

Keywords: Ultrasound guided lung, maneuvers, postoperative, postoperative atelectasis

Introduction

In children who are under anesthesia, there is a significant occurrence of lung collapse during phases of low oxygen levels. General anesthesia may cause diaphragmatic dysfunction, which is a significant determinant in the development of regional lung aeration deficits. The weight of the abdominal organs exerts upward pressure on the diaphragm, causing compression of the lungs in the lower regions. The regional collapse of the lung may vary from a little reduction in air supply to a full collapse known as atelectasis^[1-3].

Because it causes an additional rise in intra-abdominal pressure, capnoperitoneum created during laparoscopy might worsen lung collapse. The pleural pressure may be affected by variations in thoracic compliance brought about by capnoperitoneum since the chest wall and abdomen are connected^[4,5].

Various strategies for attracting new members have been detailed. High pressure-controlled breathing, incremental positive end-expiratory pressure (PEEP), intermittent sighs, and persistent inflation procedures are the most important. Unfortunately, the optimal recruitment maneuver strategy is still a mystery and might change depending on the details^[6].

Ultrasound of the lungs is becoming more widely accepted as a safe and effective method for identifying a wide range of pulmonary disorders in both children and adults. There is mounting evidence that perioperative care, in addition to emergency and critical care settings, is an appropriate environment for lung ultrasounds^[7-9].

Risk factors for atelectasis

Atelectasis risk factors may be grouped into three main areas: patient, anesthetic, and operation. Through imaging methods and their associations with global assessments of gas exchange and respiratory mechanical dysfunction, several risk variables have been discovered^[10].

Patient-related Risk Factors

Obesity: There are more atelectatic regions in obese people compared to thin ones^[11]. The diaphragm protrudes more cephalad due to the increasing weight of abdominal and thoracic

adipose tissue, which in turn increases the compressive pressures conveyed to the lung from the chest wall [12].

Age: At the age of 20 the vulnerability to airway closure, as measured by the disparity between FRC and closing capacity, is small, but grows at both the early and elderly stages of life [13].

Diaphragmatic Dysfunction: An increased incidence of atelectasis is reported after upper abdominal and cardiothoracic surgeries, which are characterized by perioperative diaphragmatic dysfunction. In the case of heart surgery, for example, the ultrasound-measured preoperative diaphragmatic thickening fraction is a reliable predictor of pulmonary atelectasis, pneumonia, and extended mechanical ventilation [14]. Furthermore, a diaphragmatic excursion measuring less than 10 mm on ultrasound examination 24 hours after surgery is linked to a greater likelihood of atelectasis occurring after thoracic surgery [15].

Increased intra-abdominal pressure: Large pulmonary atelectasis in the supine position, especially after diaphragmatic tone loss, may be precipitated by intra-abdominal hypertension (ileus, ascites, tumor, hematoma), which raises pleural pressure and lowers transpulmonary pressures [16].

Anesthesia-related Risk Factors

General Anesthesia Drugs

- **Sedative-hypnotics:** Intraoperative pulmonary atelectasis has been linked to both inhaled and intravenous anesthetics. Pulmonary problems after noncardiac surgeries are equally likely to occur with inhalational and complete intravenous anesthesia.⁽¹⁷⁾ The administration of propofol results in a dose-dependent decrease in the fraction of respiratory capacity (FRC) and an increase in ventilation inhomogeneity in preschoolers who breathe on their own.⁽¹⁸⁾ Premedication with midazolam in preschoolers may result in a little decrease in FRC and respiratory compliance as well as a slight increase in ventilation inhomogeneities [19].
- **Opioids:** Opioids are respiratory depressants that

reduce the sensitivity to carbon dioxide and the central neuronal drive to the respiratory muscles, causing cough inhibition and respiratory depression. As a result, postoperative atelectasis has been dose-dependently linked to intraoperative systemic opioids [20].

- **Neuromuscular Blocking Agents and Antagonists:** Most children under the age of 3, especially when employing neuromuscular blockade, have atelectasis soon after the start of general anesthesia [21]. After surgery, the presence of remaining neuromuscular blockade leads to impaired functioning of the respiratory muscles, collapse of the lungs, and low levels of oxygen in the blood [22, 23]. This emphasizes the need of reversing the neuromuscular blockade [24].
- **Fraction of Inspired Oxygen:** The early development of atelectasis may be prevented by using a gas combination that contains a gas that is poorly absorbed, such nitrogen, during the induction of anesthesia. If the lungs are ventilated with a low proportion of oxygen in nitrogen after a recruitment procedure, pulmonary collapse will progressively resurface under continued anesthesia. However, atelectasis quickly returns once pure oxygen is blown into the lungs. Consequently, it is recommended to use a moderate fraction of inspired oxygen (FIO₂, e.g. 0.3-0.4) while ventilating a patient under anesthesia, if at all feasible. Another option is to think about using PEEP if the lungs are ventilated with a high inspiratory proportion of oxygen [11].
- **Blood Transfusion:** Postoperative pulmonary problems, such as atelectasis, have been linked to perioperative blood transfusion based on systematic chest computed tomography after orthopedic surgery [25, 26].

Surgery-related Risk Factors

Body Position

Operating Table Angle: The supine posture causes a 27% reduction in FRC compared to the sitting position (at a 90-degree angle). This reduction occurs because the supine position allows the diaphragm to move upwards due to the pressure from the organs in the abdomen. In sedated youngsters, the Trendelenburg position causes even more compression of the dorso-caudal lung, resulting in a further decrease in FRC of around 12% [27].

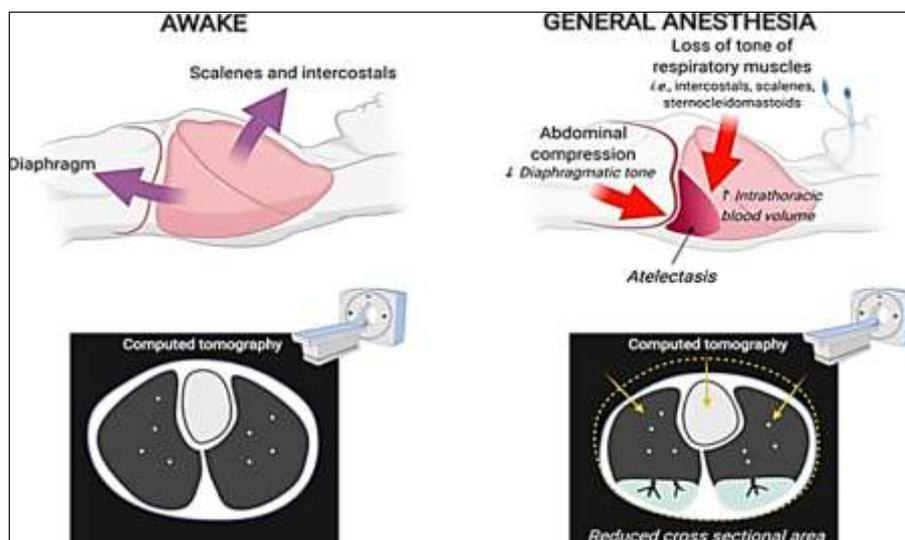


Fig 1: The impact of being flat on one's back.

As a result of being under general anesthesia, a supine patient's chest wall may change form. The expansion of the lungs is sustained during awake spontaneous breathing by contracting the diaphragm and the accessory muscles of respiration. Muscle atrophy due to anesthesia causes the dependent diaphragm to move cephalad, the chest's cross-sectional area to decrease, and nongravitational compressive pressures to be generated, which are known as cephalocaudal gradients. Lung volume loss and collapse, especially in the dorsal and basal lung areas, is caused by these causes as well as by gravity forces and a possible rise in intrathoracic blood volume [28].

- **Prone Positioning:** When lying flat on one's back, it might be difficult to move the front of the chest and the belly, and the diaphragm and the dorsal wall of the chest are the primary determinants of lung expansion. By lowering the dependent lung's mass to the ground, the prone posture adjusts the chest wall's and lungs' form matching to a more favorable degree and lessens the pressure on the lungs from the heart and abdominal tissues. Because of both gravitational (dorsal larger than ventral expansion) and nongravitational (caudal greater than cranial expansion) causes, they lead to reduced degradation of lung inflation and regional strain over time [29] and spatial homogeneity of lung aeration [30].
- **Lateral Decubitus:** Because the nondependent lung, mediastinum, and abdominal organs all bear down on the dependent lung, it is compressed. Consequently, computed tomography nearly always finds atelectasis in the dependent lung in individuals who are anesthetized [31].
- **Pneumoperitoneum:** By raising intra-abdominal pressure, pneumoperitoneum compresses the juxta-diaphragmatic lung areas, which in turn encourages the diaphragm to move cephalad [32]. The end-expiratory lung volume, respiratory system compliance, and end-expiratory transpulmonary pressure are all independently reduced by peritoneal insufflation (about 35% in nonobese people and around 15% in obese people) [33, 34].
- **Surgery Duration**
General anesthesia rapidly causes upward movement of the diaphragm in individuals who are lying down or in a semi-sitting position [35, 36]. Pulmonary atelectasis occurs shortly after losing consciousness. [37] Studies conducted on sheep showed that lung collapse can happen in healthy lungs with similar air distribution as humans for up to 16 hours after starting general anesthesia with low tidal volume ventilation and without PEEP [38]. Progressive collapse of the body might worsen the negative impact of the length of anesthesia on difficulties in the lungs after surgery [39].

Laparoscopic surgery

Anesthesia administration during laparoscopy has become increasingly challenging due to the increasing focus on performing advanced laparoscopic surgery on elderly and medically compromised patients. While laparoscopy is considered a safe and straightforward treatment that can be done on an outpatient basis, it is important to exercise extra care when it comes to the anesthetic approach due to its potential to affect the circulatory and pulmonary function of patients. Moreover, laparoscopy is also being used with safety and efficacy in youngsters [40].

For optimal vision and manipulation of the abdominal organs, it is recommended to create a pneumoperitoneum using 2.5 to 5.0 liters of insufflated carbon dioxide. The presence of pneumoperitoneum inevitably increases the pressure inside the abdomen, known as intra-abdominal pressure (IAP), which may have notable impacts on the cardiovascular, respiratory, and neurological systems [41].

A. Cardiovascular effects

Significant hemodynamic changes include modifications in arterial blood pressure, such as hypotension and hypertension, as well as arrhythmias and cardiac arrest. The magnitude of the circulatory alterations linked to the establishment of pneumoperitoneum may vary based on factors such as the level of intra-abdominal pressure achieved, the amount of carbon dioxide absorbed, the patient's intravascular volume, the method of ventilation, the surgical circumstances, and the anesthetic medications used. The key factors that significantly affect cardiovascular function during laparoscopy are the IAP and the posture of the patient [41].

Assuming a head-up posture leads to a reduction in the amount of blood returning to the heart and the overall pumping ability of the heart, resulting in a drop in the average pressure inside the arteries and the overall efficiency of blood circulation. Additionally, it causes an increase in the resistance to blood flow in the peripheral blood vessels and the blood vessels in the lungs [42].

Pediatric laparoscopic operations are expected to result in elevated pulmonary and systemic vascular resistance, as well as abrupt bradycardia due to increased IAP during pneumoperitoneum. These effects are more likely to occur in children than in adults. Children possess a heightened amount of vagal tone, and they may experience bradycardia or asystole as a result of peritoneal stimulation caused by a sudden influx of gas or the insertion of trocars and laparoscopes [43].

Individuals who have good cardiovascular function are able to handle changes in preload and afterload without any issues. However, patients with cardiovascular diseases, anemia, or hypovolemia need to be carefully monitored and managed in terms of volume loading, posture, and insufflation pressures. It has been advised to limit the intra-abdominal pressure (IAP) to 6 mmHg in babies and 12 mmHg in older children. The impact of these pressures on cardiac index is modest [44].

B. Respiratory Effects

Pulmonary function is altered during laparoscopy because to decreased lung volumes, elevated peak airway pressures, and reduced pulmonary compliance resulting from increased IAP and patient posture [45].

Pulmonary pathophysiological alterations result in hypercapnia and hypoxemia when ventilation is ineffective, leading to pulmonary vasoconstriction [42]. Increased intra-abdominal pressure (IAP) has a greater effect on reducing thoracic compliance and may lead to pneumothorax and pneumomediastinum due to elevated alveolar pressures. This is especially true for patients with significant pulmonary illness who are having laparoscopic upper abdominal procedures [45].

C. Neurological Effects

The combination of hypercapnia, elevated systemic vascular

resistance, and a head-down posture results in an increase in intracranial pressure [44].

The cerebral blood flow rises during pneumoperitoneum in the head-down position as a result of the increase in arterial CO₂ pressure [46]. The augmentation of blood flow in the brain, which is often well-tolerated

, may provide a risk for patients with cerebral pathology, reduced intracranial compliance, or compromised cerebral physiology. The selection of anesthetic drugs also contributes to reducing the cerebrovascular reactions during laparoscopy [47].

Lung recruitment maneuvers

Positive pressure mechanical ventilation is an artificial method of breathing support that may be lifesaving, but it is not without significant adverse consequences. It consistently leads to varying levels of collapse in either the tiny airways or the whole acinus. The ensuing decrease in lung aeration might vary from limited ventilation to total collapse of the lung (atelectasis) [48].

The implications include not only the degradation of gas exchange and impairment of lung mechanics, but also an increased risk of eliciting an inflammatory response in the lungs [49].

As a result of a short and controlled rise in transpulmonary pressure, alveolar recruitment causes the previously compressed lung sections to re-expand. In order to increase the end-expiration volume and enhance gas exchange, the goal of AR is to establish and sustain a collapse-free environment [50].

Multiple methods have been described

- 1. A sustained inflation:** is the most often used recruiting tactic. Conventional wisdom is that the patient should be closely observed for symptoms of side effects, such as hemodynamic compromise, while the ventilator is adjusted to CPAP mode and the pressure is increased to 30–40 cm H₂O for 30–40 seconds. When the PEEP is increased, more aggressive treatments include pressure-controlled breaths (e.g., 10 breaths/min with a 1:1 inspiration-expiration ratio and a pressure control of 10–20 cm H₂O) [51].
- 2. Sigh:** The aim is to achieve a specified plateau pressure by making modifications to either increase tidal volume or apply PEEP during one or more breaths [52].
- 3. Extended sigh (ES):** The initiation of a degassed lung unit is determined by both the applied pressure and the duration of maintained pressure, known as the inflating pressure-time product. They developed a novel kind of sigh called "extended sigh" (ES) to effectively apply pressure and prevent excessive airway pressure in the lungs of patients with ARDS. The approach was to replicate the mechanistic framework and physiological impact seen in patients with ARDS. It may enhance recruitment of new employees and reduce the time required by progressively increasing the pressure near the end of expiration. This phenomenon takes into account the relationship between pressure and time, and is distinguished by a gradual rise in PEEP (positive end-expiratory pressure), accompanied by a reduction in tidal volume over an extended duration [53].
- 4. Titrating PEEP Recruitment:** The PEEP is gradually raised by 2–5 cmH₂O while maintaining a set tidal volume (VT) of 6 ml per kilogram of ideal body

weight. This is achieved using volume control ventilation. At each stage, the monitoring includes the measurement of driving pressure (the difference between P_{plat} and PEEP, compliance, SPO₂, and blood pressure. PEEP is elevated when there are indications of recruitment, such as reduced driving pressure, plateau pressure (P_{plat}) below 30 cmH₂O, or elevated peripheral oxygen saturation (SpO₂). If there are signs of overdistension, such as an increased driving pressure, a plateau pressure over 30 cmH₂O, hypotension, or a drop in SpO₂, the positive end-expiratory pressure (PEEP) is reduced to the prior level. The duration of each step is typically 3–5 minutes, unless there are any negative effects like as hypotension or desaturation, which would need a reduction in PEEP to the prior level. An alternate method involves maintaining a constant degree of pressure control while increasing the PEEP. However, there is little data to establish the superiority of one technique over the other, and the decision is influenced by individual bias [54].

- 5. High-frequency ventilation:** High-frequency ventilation, in comparison to standard mechanical ventilation modes, results in elevated mean airway pressures. This helps prevent cyclic alveolar closure and promotes an increase in the volume of the lungs at the end of expiration. The number is 55. High-frequency oscillatory ventilation administers small tidal volumes (equivalent to or less than anatomical dead space) with frequencies ranging from 3 to 15 Hz. Additionally, it sustains an elevated airway pressure to facilitate recruitment. The relationship between ventilation and respiratory frequency is inverse, whereas the relationship between ventilation and pressure amplitude of oscillation is direct. Optimally, this approach allows for a more uniform spread of air circulation by preserving the average pressure in the airways, while preventing excessive inflation and reducing the risk of lung damage caused by the ventilator via limiting fluctuations in the amount of air breathed in and out [56–58].
- 6. Ventilation in prone decubitus:** Placing the patient in a prone decubitus posture changes the way the transpulmonary pressure gradient is distributed, resulting in a more even filling of the alveoli. While it does not significantly alter blood flow, it does enhance the ratio of ventilation to perfusion. Prone decubitus might be considered a kind of recruiting on its own. Furthermore, using some standard ventilation strategies will lead to a more even distribution of the applied pressures [52, 59]. Absolute and relative reasons why a person shouldn't be lying on their back include spine instability, high intracranial pressure, heart or blood flow problems, heavy bleeding, thoracic or abdominal surgery, anterior chest tubes with leaks, and deep vein thrombosis that has been treated for less than two days [60].

Lung ultrasound

Lung ultrasonography surpasses the combined diagnostic accuracy of physical examination and chest radiography. It improves safety by eliminating ionizing radiation and the necessity for potentially risky transfers inside the hospital. Additionally, it may be used for fluid management,

weaning, and therapeutic operations like thoracocentesis. LUS is a language that can be learned reasonably quickly, although it has a high learning curve [61, 62].

The appearances of LUS are determined by the proportions of air and fluid present in the lung, which is influenced by the acoustic impedance (Z) phenomena. This is a quantification of the ability of particles in a substance to withstand mechanical vibrations. The resistance is directly proportional to both the density of the medium and the propagation velocity of ultrasound in the medium. When a sound wave encounters a significant and level interface between two media with varying impedances, a portion of the sound is transmitted across the boundary while the rest is reflected, resulting in an echo. As the disparity in Z increases, so does the intensity of the reflection. The fluid's constant Z value eliminates echoes, causing it to seem black. Soft tissues have a high degree of similarity in their Z values, leading to negligible reflection. Approximately 40% of the energy in the United States is reflected at the interfaces between bone and soft tissue. The presence of soft tissue and air results in a reflection of 99.9%, making it very difficult for ultrasound waves to penetrate this contact [62].

In an air-filled lung, it is not possible to see objects below the pleura. Only artifacts will be visible. LUS depends on the analysis of artifacts in situations when the lung is mostly filled with air. The characteristics of these artifacts will vary based on the proportion of air and fluid. Direct visualization of the lung is possible when it is significantly filled with fluid [61].

Typically, US machines in the US are equipped with either a linear probe for vascular access, a curvilinear probe for abdominal imaging, a phased array probe for echocardiography, or a combination of these probes. One significant benefit of LUS is that it allows for the acquisition of valuable photos for each of these [62].

Linear probe (8–12 MHz)

These probes with high frequencies provide excellent resolution of surface-level features. Due to its superficial position, the anterior pleura allows for high-quality imaging of the pleura and lung slide. Due to the limited ability of high-frequency ultrasound to penetrate deeply and the short range it can cover, imaging of deeper tissues is inadequate [62].

Curvilinear probe (3–5 MHz)

This probe is the most versatile and effective for LUS. The visualization of lung slippage is straightforward. Effusions, consolidated lung, and the diaphragm are clearly seen due to the excellent depth penetration and wide sector width. To prevent interference with the ribs during postero-lateral scanning, considerable angulation is required due to the probe's significant footprint [62].

Phased array (3–4.5 MHz)

For the purpose of going in between the ribs, these probes have a footprint that is helpful. Although they are capable of demonstrating all of the symptoms of LUS, the quality of

the pictures is not as high as it may be [62].

LUS signs

All indications in LUS originate from the pleural line, except for subcutaneous emphysema, which eliminates it due to the presence of air above it. The presence of rib shadows will be observable due to the reflection of sound back to the probe. In the intervening spaces, a distinct brilliant white pleural line may be seen, positioned about 0.5 cm below the rib line. In a regularly aerated lung, the pleura appears as a distinct and bright horizontal line when seen. Align the 'bat symbol' (Fig.2) near the center of your picture, with the ribs serving as the wings. The presence of air below the pleural line causes most ultrasound waves to be reflected back to the transducer. This serves as a reflector, causing some waves from the US to rebound repeatedly between the pleura and transducer, resulting in the creation of artifacts known as A lines. There are horizontal lines located underneath the pleura that have equal spacing to the distance between the probe and the pleural line. They are found in both normal lungs and in pneumothorax because they indicate the presence of air underneath the pleura. Rotating the probe horizontally will eliminate the rib shadows, allowing for better visualization of the pleural line. An untrained user may mistakenly identify missing lung slide by falsely interpreting a rib as the pleural line, posing a potential threat [62].

Fig.2. The ultrasound image of a typical aerated lung shows the presence of horizontal reverberation artifacts, often known as A-lines, along the pleural line (shown by red arrows). The distinctive visual representation of two ribs with the pleural line in between is often known as the "bat sign" [62].

The visceral and parietal pleura are often in close proximity, with a little quantity of fluid between them, allowing for smooth sliding during breathing. The phenomenon seen is known as lung sliding, characterized by the reciprocal motion of the pleura, typically accompanied by the presence of small blebs moving along the pleural line [62]. The M-mode picture utilizes a single scan line to display echoes plotted against time. In this image, the subcutaneous tissue located above the pleural line appears as horizontal straight lines. On the other hand, below the pleural line, the movement of lung slides creates a sandy appearance. This particular phenomenon is referred to as the 'seashore sign'. [62] (Fig.3)

The presence of lung sliding will decrease when using low tidal volumes or in cases of lung hyperinflation. Absence of the pleural space may occur in conditions when the pleura are not directly facing each other (such as pneumothorax or effusion), when they are adhered together (as in pneumonia, ARDS, or pleurodesis), or in cases of missing breathing (such as pneumonectomy or one lung intubation [62]. The 'stratosphere sign' (also known as the barcode sign) is seen on M-mode ultrasound imaging. It is characterized by unbroken smooth horizontal lines that represent the fixed chest wall, due to the absence of lung movement [63]. (Fig.4).

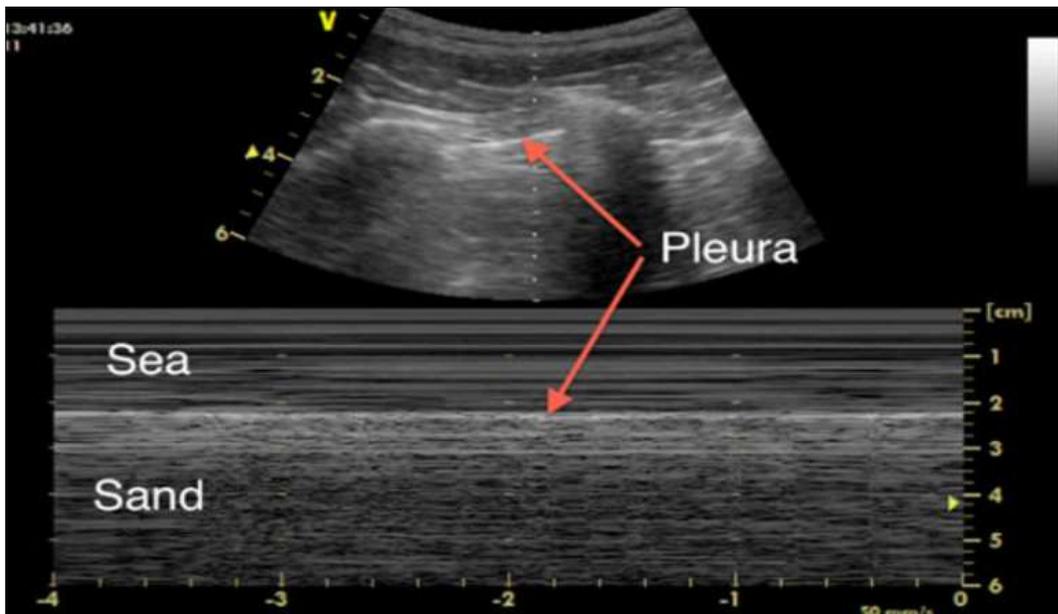


Fig 3: M-mode image of lung sliding (the 'seashore sign') [62].

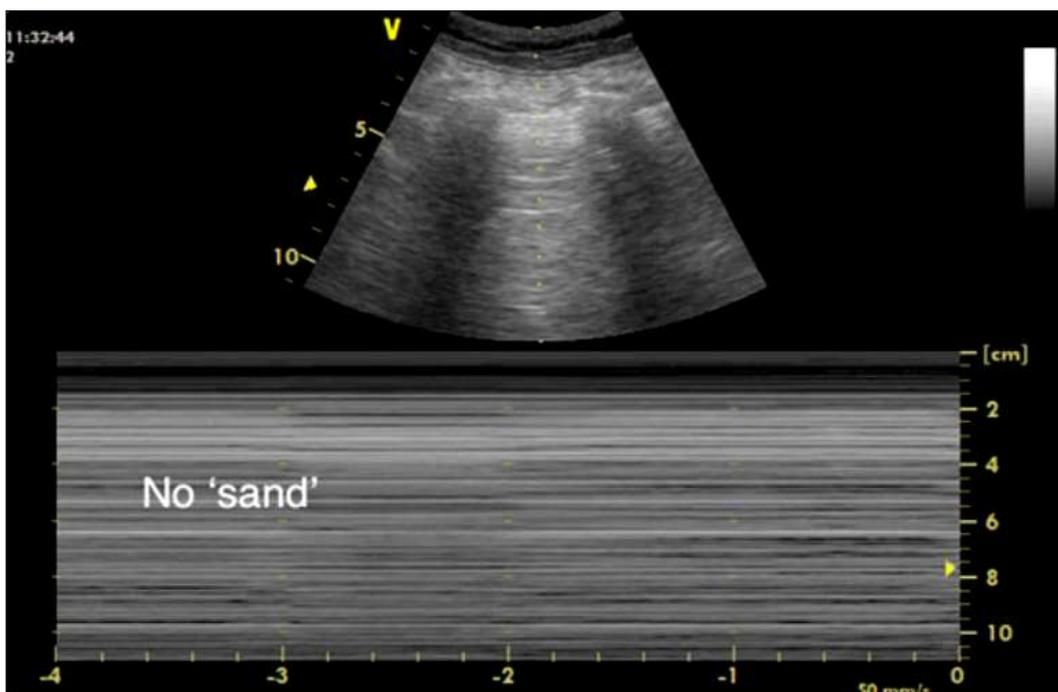


Fig 4: M-mode image of absent sliding (the 'stratosphere sign') [62].

Interstitial syndrome [64].

Is a sonographic entity caused by:

Pulmonary edema may be caused by either fluid overload and heart failure, or by increased permeability due to ALI or ARDS. It can also be associated with interstitial pneumonia or pneumonitis, as well as lung fibrosis.

The distinguishing characteristic of the US image is the presence of B lines, as seen in Figure 5. These artifacts are produced when alveolar air and septal thickness (caused by fluid or fibrosis) are placed side by side. Their characteristics are:

- The pleural line gives rise to lengthy, vertical hyperechoic lines that extend deep into the picture. These lines resemble comet tails and are also known as such. They eliminate A lines and exhibit movement along with lung sliding.

In normal lungs, some B lines may be seen, particularly at the bases. A maximum of two ribs between two neighboring ribs may be regarded within the typical range. Pathological conditions are indicated when three or more gaps between the ribs are present, or when they are closely grouped together in a transverse view. Depending on the pathophysiology, they might exhibit localization, dissemination, homogeneity, or non-homogeneity. They are found in all diseases that impact the interstitium. The most frequent cause is pulmonary edema, characterized by the presence of Kerley B lines. As oedema worsens, indicated by a ground-glass look on CT scans, the number and spacing of B lines increase. Severe swelling leads the ribs to merge together with a dense and continuous pattern that fills the space between them, resulting in a condition known as "white lung".

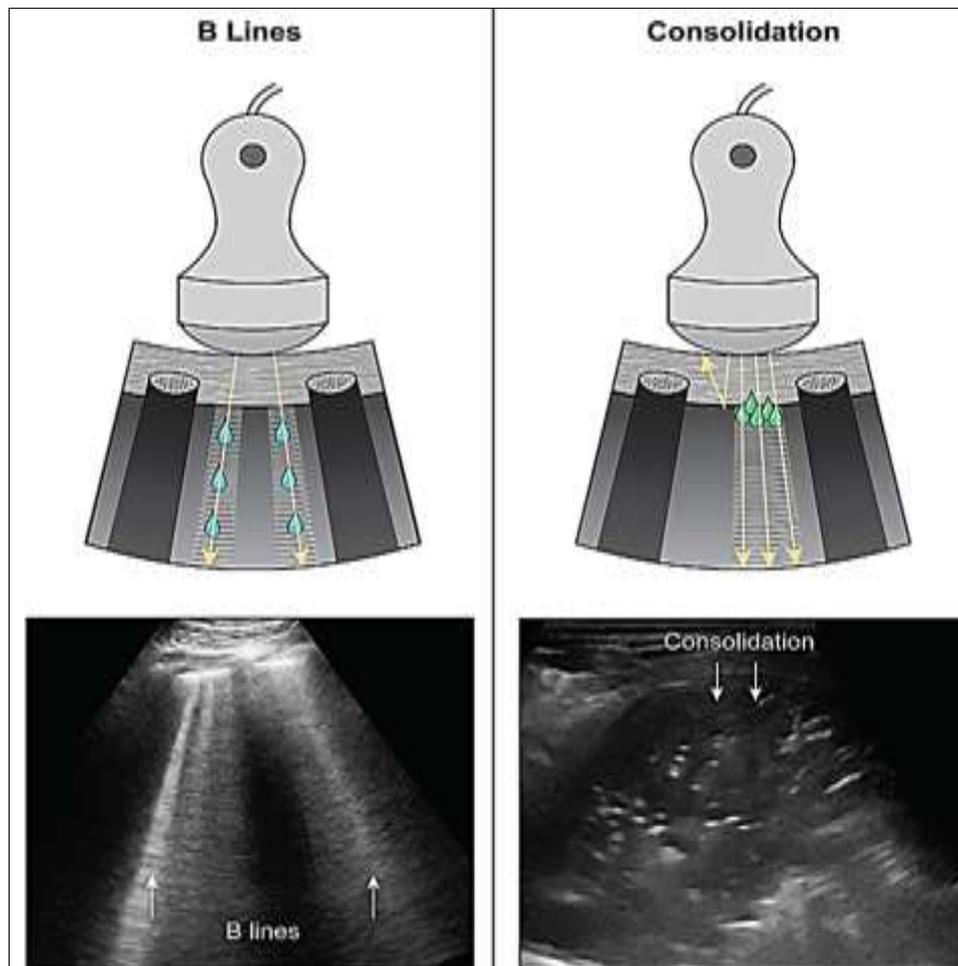


Fig 5: Interstitial syndrome.

(Left) As the interstitium becomes thicker, the pattern of artifacts alters, with B-line artifacts completely obscuring A-lines. B-lines are vertical artifacts that appear as bright echoes that originate from the pleural line, reaching all the way to the bottom of the field of vision. Correct The US directly penetrates the consolidation, resulting in artifact-free imaging if the consolidation is in contact with the pleural line^[63].

Atelectasis: Distinguishing between atelectasis and consolidation is challenging with ultrasound. The presence of bronchograms is indicated as described above, however they have a limited level of specificity. Compression atelectasis is more likely to occur in the presence of a substantial pleural effusion. A little accumulation of fluid increases the probability of consolidation. If a substantial collapse occurs, it will be accompanied by indications such as an elevated hemidiaphragm. Almost all pleural effusions in critically sick patients are accompanied with underlying consolidated or atelectatic lung tissue. To definitively differentiate compression atelectasis from consolidation, it is necessary to remove the effusion and observe whether the lung undergoes re-aeration^[62].

References

1. Acosta CM, Maidana GA, Jacovitti D, Belaunzarán A, Cereceda S, Rae E, *et al.* Accuracy of transthoracic lung ultrasound for diagnosing anesthesia-induced atelectasis in children. *Anesthesiology*. 2014;120(6):1370-1379.
2. de Graaff JC, Bijker JB, Kappen T, van Wolfswinkel L, Zuithoff NP, Kalkman CJ. Incidence of intraoperative hypoxemia in children in relation to age. *Anesthesia & Analgesia*. 2013;117(1):169-175.
3. Song IK, Kim EH, Lee JH, Ro S, Kim HS, Kim JT. Effects of an alveolar recruitment manoeuvre guided by lung ultrasound on anaesthesia-induced atelectasis in infants: a randomised, controlled trial. *Anaesthesia*. 2017;72(2):214-222.
4. Malbrain ML, De Laet I, De Waele JJ, Sugrue M, Schachtrupp A, Duchesne J, *et al.* The role of abdominal compliance, the neglected parameter in critically ill patients-a consensus review of 16. Part 2: measurement techniques and management recommendations. *Anaesthesiology intensive therapy*. 2014;46(5):406-432.
5. Manner T, Aantaa R, Alanen M. Lung compliance during laparoscopic surgery in paediatric patients. *Pediatric Anesthesia*. 1998;8(1):25-29.
6. Pelosi P, de Abreu MG, Rocco PR. New and conventional strategies for lung recruitment in acute respiratory distress syndrome. *Critical Care*. 2010;14:1-7.
7. Cattarossi L. Lung ultrasound: its role in neonatology and pediatrics. *Early Human Development*. 2013;89:S17-S19.
8. Ashton-Cleary D. Is thoracic ultrasound a viable alternative to conventional imaging in the critical care setting? *British Journal of Anaesthesia*. 2013;111(2):152-160.

9. Alsaddique A, Royse AG, Royse CF, Mobeirek A, El Shaer F, AlBackr H, *et al.* Repeated monitoring with transthoracic echocardiography and lung ultrasound after cardiac surgery: feasibility and impact on diagnosis. *Journal of cardiothoracic and vascular anesthesia.* 2016;30(2):406-412.
10. Lagier D, Zeng C, Fernandez-Bustamante A, Vidal Melo MF. Perioperative pulmonary atelectasis: Part ii. Clinical implications. *Anesthesiology.* 2022;136(1):206-236.
11. Hedenstierna G, Rothen HU. Atelectasis formation during anesthesia: causes and measures to prevent it. *Journal of clinical monitoring and computing.* 2000;16:329-335.
12. Boriek AM, Lopez MA, Velasco C, Bakir AA, Frolov A, Wynd S, *et al.* Obesity modulates diaphragm curvature in subjects with and without COPD. *Am J Physiol Regul Integr Comp Physiol.* 2017;313(5):R620-R629.
13. Mansell A, Bryan C, Levison H. Airway closure in children. *Journal of Applied Physiology.* 1972;33(6):711-4.
14. Cavayas YA, Eljaiek R, Rodrigue É, Lamarche Y, Girard M, Wang HT, *et al.* Preoperative diaphragm function is associated with postoperative pulmonary complications after cardiac surgery. *Crit Care Med.* 2019;47(12):e966-e74.
15. Spadaro S, Grasso S, Dres M, Fogagnolo A, Dalla Corte F, Tamburini N, *et al.* Point of care ultrasound to identify diaphragmatic dysfunction after thoracic surgery. *Anesthesiology.* 2019;131(2):266-278.
16. Pickhardt PJ, Shimony JS, Heiken JP, Buchman TG, Fisher AJ. The abdominal compartment syndrome: CT findings. *AJR American journal of roentgenology.* 1999;173(3):575-579.
17. Uhlig C, Bluth T, Schwarz K, Deckert S, Heinrich L, De Hert S, *et al.* Effects of volatile anesthetics on mortality and postoperative pulmonary and other complications in patients undergoing surgery: a systematic review and meta-analysis. *Anesthesiology.* 2016;124(6):1230-1245.
18. von Ungern-Sternberg B, Frei F, Hammer J, Schibler A, Doerig R, Erb T. Impact of depth of propofol anaesthesia on functional residual capacity and ventilation distribution in healthy preschool children. *British journal of anaesthesia.* 2007;98(4):503-508.
19. von Ungern-Sternberg BS, Erb TO, Habre W, Sly PD, Hantos Z. The impact of oral premedication with midazolam on respiratory function in children. *Anesthesia & Analgesia.* 2009;108(6):1771-1776.
20. Friedrich S, Raub D, Teja B, Neves S, Thevathasan T, Houle T, *et al.* Effects of low-dose intraoperative fentanyl on postoperative respiratory complication rate: a pre-specified, retrospective analysis. *British journal of anaesthesia.* 2019;122(6):e180-e188.
21. von Ungern-Sternberg BS, Hammer J, Schibler A, Frei FJ, Erb TO. Decrease of functional residual capacity and ventilation homogeneity after neuromuscular blockade in anesthetized young infants and preschool children. *The Journal of the American Society of Anesthesiologists.* 2006;105(4):670-675.
22. Martinez-Ubieto J, Ortega-Lucea S, Pascual-Bellosta A, Arazo-Iglesias I, Gil-Bona J, Jimenez-Bernardó T, *et al.* Prospective study of residual neuromuscular block and postoperative respiratory complications in patients reversed with neostigmine versus sugammadex. *Minerva anesthesiologica.* 2015;82(7):735-742.
23. McLean DJ, Diaz-Gil D, Farhan HN, Ladha KS, Kurth T, Eikermann M. Dose-dependent association between intermediate-acting neuromuscular-blocking agents and postoperative respiratory complications. *Anesthesiology.* 2015;122(6):1201-1213.
24. Blobner M, Hunter JM, Meistelman C, Hoefl A, Hollmann MW, Kirmeier E, *et al.* Use of a train-of-four ratio of 0.95 versus 0.9 for tracheal extubation: an exploratory analysis of POPULAR data. *British Journal of Anaesthesia.* 2020;124(1):63-72.
25. Song K, Rong Z, Yang X, Yao Y, Shen Y, Shi D, *et al.* Early pulmonary complications following total knee arthroplasty under general anesthesia: A prospective cohort study using CT scan. *BioMed Research International.* 2016;2016.
26. Yin S, Tao H, Du H, Feng C, Yang Y, Yang W, *et al.* Postoperative pulmonary complications following posterior spinal instrumentation and fusion for congenital scoliosis. *PLoS One.* 2018;13(11):e0207657.
27. Regli A, Habre W, Saudan S, Mamie C, Erb T, Von Ungern-Sternberg BS, *et al.* Impact of Trendelenburg positioning on functional residual capacity and ventilation homogeneity in anaesthetised children. *Anaesthesia.* 2007;62(5):451-455.
28. Zeng C, Lagier D, Lee J-W, Vidal Melo MF. Perioperative pulmonary atelectasis: part I. Biology and mechanisms. *Anesthesiology.* 2022;136(1):181-205.
29. Motta-Ribeiro GC, Hashimoto S, Winkler T, Baron RM, Grogg K, Paula LF, *et al.* Deterioration of regional lung strain and inflammation during early lung injury. *American Journal of Respiratory and Critical Care Medicine.* 2018;198(7):891-902.
30. Xin Y, Cereda M, Hamedani H, Martin KT, Tustison NJ, Pourfathi M, *et al.* Positional Therapy and Regional Pulmonary Ventilation High-resolution Alignment of Prone and Supine Computed Tomography Images in a Large Animal Model. *Anesthesiology.* 2020;133(5):1093-1105.
31. Lohser J, Slinger P. Lung injury after one-lung ventilation: a review of the pathophysiologic mechanisms affecting the ventilated and the collapsed lung. *Anesthesia & Analgesia.* 2015;121(2):302-318.
32. Andersson LE, Bååth M, Thörne A, Aspelin P, Odeberg-Wernerman S. Effect of carbon dioxide pneumoperitoneum on development of atelectasis during anesthesia, examined by spiral computed tomography. *The Journal of the American Society of Anesthesiologists.* 2005;102(2):293-299.
33. Futier E, Constantin J-M, Pelosi P, Chanques G, Kwiatkoski F, Jaber S, *et al.* Intraoperative recruitment maneuver reverses detrimental pneumoperitoneum-induced respiratory effects in healthy weight and obese patients undergoing laparoscopy. *The Journal of the American Society of Anesthesiologists.* 2010;113(6):1310-1319.
34. Tharp WG, Murphy S, Breidenstein MW, Love C, Booms A, Rafferty MN, *et al.* Body Habitus and Dynamic Surgical Conditions Independently Impair Pulmonary Mechanics during Robotic-assisted Laparoscopic Surgery A Cross-sectional Study. *Anesthesiology.* 2020;133(4):750-763.

35. Lagier D, Velly LJ, Guinard B, Bruder N, Guidon C, Vidal Melo MF, *et al.* Perioperative Open-lung Approach, Regional Ventilation, and Lung Injury in Cardiac Surgery A PROVECS Trial Substudy. *Anesthesiology*. 2020;133(5):1029-1045.
36. Ukere A, März A, Wodack K, Trepte C, Haese A, Waldmann A, *et al.* Perioperative assessment of regional ventilation during changing body positions and ventilation conditions by electrical impedance tomography. *BJA: British Journal of Anaesthesia*. 2016;117(2):228-235.
37. Hedenstierna G, Edmark L. The effects of anesthesia and muscle paralysis on the respiratory system. *Applied Physiology in Intensive Care Medicine 1: Physiological Notes-Technical Notes-Seminal Studies in Intensive Care*. 2012:299-307.
38. Tucci MR, Costa EL, Wellman TJ, Musch G, Winkler T, Harris RS, *et al.* Regional lung derecruitment and inflammation during 16 hours of mechanical ventilation in supine healthy sheep. *Anesthesiology*. 2013;119(1):156-65.
39. Fernandez-Bustamante A, Frenzl G, Sprung J, Kor DJ, Subramaniam B, Ruiz RM, *et al.* Postoperative pulmonary complications, early mortality, and hospital stay following noncardiothoracic surgery: A multicenter study by the perioperative research network investigators. *JAMA surgery*. 2017;152(2):157-166.
40. Mattioli G, Repetto P, Carlini C, Torre M, Prato PA, Mazzola C, *et al.* Laparoscopic vs open approach for the treatment of gastroesophageal reflux in children. *Surgical Endoscopy and Other Interventional Techniques*. 2002;16:750-752.
41. Gerges FJ, Kanazi GE, Jabbour-Khoury SI. Anesthesia for laparoscopy: a review. *Journal of clinical anaesthesia*. 2006;18(1):67-78.
42. Gutt C, Oniu T, Mehrabi A, Schemmer P, Kashfi A, Kraus T, *et al.* Circulatory and respiratory complications of carbon dioxide insufflation. *Digestive surgery*. 2004;21(2):95-105.
43. Saxena A. *A Practical Approach to Robotic Surgery*: JP Medical Ltd; c2017.
44. Pennant JH. Anesthesia for laparoscopy in the pediatric patient. *Anesthesiology Clinics of North America*. 2001;19(1):69-88.
45. Rauh R, Hemmerling TM, Rist M, Jacobi KE. Influence of pneumoperitoneum and patient positioning on respiratory system compliance. *Journal of clinical anaesthesia*. 2001;13(5):361-365.
46. Lee J, Lee P, Do S, Jeon Y, Lee J, Hwang J, *et al.* The effect of gynaecological laparoscopic surgery on cerebral oxygenation. *Journal of international medical research*. 2006;34(5):531-536.
47. Moka E. Cerebral oximetry and laparoscopic surgery. *Journal of minimal access surgery*. 2006;2(2):47.
48. Brismar B, Hedenstierna G, Lundquist H, Strandberg A, Svensson L, Tokics L. Pulmonary densities during anesthesia with muscular relaxation--a proposal of atelectasis. *Anesthesiology*. 1985;62(4):422-428.
49. Magnusson L, Spahn D. New concepts of atelectasis during general anaesthesia. *British journal of anaesthesia*. 2003;91(1):61-72.
50. Richard J-C, Maggiore SM, Mercat A. Clinical review: Bedside assessment of alveolar recruitment. *Critical Care*. 2003;8(3):1-7.
51. Hess DR, Bigatello LM. Lung recruitment: the role of recruitment maneuvers. *Respiratory care*. 2002;47(3):308-317; discussion 17.
52. Pelosi P, Bottino N, Chiumello D, Caironi P, Panigada M, Gamberoni C, *et al.* Sigh in supine and prone position during acute respiratory distress syndrome. *American journal of respiratory and critical care medicine*. 2003;167(4):521-527.
53. Lim C-M, Koh Y, Park W, Chin JY, Shim TS, Lee SD, *et al.* Mechanistic scheme and effect of "extended sigh" as a recruitment maneuver in patients with acute respiratory distress syndrome: a preliminary study. *Critical care medicine*. 2001;29(6):1255-1260.
54. Hess DR. Recruitment maneuvers and PEEP titration. *Respiratory care*. 2015;60(11):1688-1704.
55. Chan KP, Stewart TE, Mehta S. High-frequency oscillatory ventilation for adult patients with ARDS. *Chest*. 2007;131(6):1907-1916.
56. Bauer K, Brücker C. The role of ventilation frequency in airway reopening. *Journal of Biomechanics*. 2009;42(8):1108-1113.
57. Pillow JJ. Tidal volume, recruitment and compliance in HFOV: same principles, different frequency. *Eur Respiratory Soc*; 2012. p. 291-293.
58. van Genderingen HR, van Vught AJ, Jansen JR. Regional lung volume during high-frequency oscillatory ventilation by electrical impedance tomography. *Critical care medicine*. 2004;32(3):787-794.
59. Rival G, Patry C, Floret N, Navellou JC, Belle E, Capellier G. Prone position and recruitment manoeuvre: the combined effect improves oxygenation. *Critical Care*. 2011;15:1-9.
60. Modrykamien AM, Gupta P, editors. *The acute respiratory distress syndrome*. Baylor University Medical Center Proceedings; 2015: Taylor & Francis.
61. Lichtenstein DA, Meziere GA. Relevance of lung ultrasound in the diagnosis of acute respiratory failure*: the BLUE protocol. *Chest*. 2008;134(1):117-125.
62. Miller A. Practical approach to lung ultrasound. *Bja Education*. 2016;16(2):39-45.
63. Marini TJ, Rubens DJ, Zhao YT, Weis J, O'Connor TP, Novak WH, *et al.* Lung ultrasound: the essentials. *Radiology: Cardiothoracic Imaging*. 2021;3(2):e200564.
64. Copetti R, Soldati G, Copetti P. Chest sonography: a useful tool to differentiate acute cardiogenic pulmonary edema from acute respiratory distress syndrome. *Cardiovascular ultrasound*. 2008;6(1):1-10.

How to Cite This Article

Nasr MM, Mostafa TAH, Elshmaa NS, Ezz HA. Ultrasound guided lung recruitment maneuvers and prevention of postoperative atelectasis after pediatric laparoscopic abdominal surgery. *International Journal of Medical Anesthesiology*. 2024;7(2):51-59.

Creative Commons (CC) License

This is an open access journal, and articles are distributed under the terms of the Creative Commons Attribution-NonCommercial-ShareAlike 4.0 International (CC BY-NC-SA 4.0) License, which allows others to remix, tweak, and build upon the work non-commercially, as long as appropriate credit is given and the new creations are licensed under the identical terms.