PHYSIOLOGICAL CHANGES DURING LAPROSCOPIC SURGERY

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The advantages of laparoscopy in comparison to open abdominal surgery include reduced surgical trauma, less pain, fewer post-operative pulmonary complications, more cosmetic and shorter recovery times.

The most commonly used gas for creating pneumoperitoneum is carbon dioxide. Carbon dioxide gas is rapidly absorbed in blood and peritoneum because of its high solubility, colorless, does not support combustion, and generally has few systemic side effects in healthy patients. For these reasons, Co2 is widely accepted for the establishment and maintenance of pneumoperitoneum during laparoscopic surgery. CO2 is a convenient choice because it is buffered by the principle physiologic buffer (bicarbonate), and is easily eliminated via the respiratory system.

Other agents have been tested for pneumoperitoneum, but to date, appear to be inferior. Nitrogen gas is not very soluble in blood and can cause gas embolism. Nitrous oxide is combustible and has been associated with sudden cardiac arrest. Helium is inert, but very poorly absorbed from the abdominal cavity. Helium, because of its low solubility in blood, has a high risk of embolism. Argon is also poorly soluble in blood and has a high risk of embolism. Argon is slowly absorbed and can remain in a closed space such as the peritoneal cavity for weeks. Oxygen and air while inexpensive, colorless, and easily absorbed, eagerly support combustion and preclude the use of many energy devices during laparoscopic interventions.

Although CarboPeritonium seems a safe altered physical state, it is a complex, pathophysiologic condition with significant physiologic effects. The physiologic effects of CarboPeritonium are principally due to raised intra abdominal pressure (IAP) and, possibly, hypercarbia. These changes are typically insignificant in healthy patients; however, they may have severe consequences in those with at-risk or major pre-existing medical conditions.

PHYSIOLOGIC EFFECTS OF LAPAROSCOPIC SURGERY

Cardiovascular System

A. Hemodynamic Effects of Pneumoperitoneum

Hemodynamic disturbances during laparoscopy are primarily due to pneumoperitoneum. The pneumoperitoneum is established by insufflating the abdomen with pressures of 15 to 20 mm Hg. Normal intra-abdominal pressure (IAP) is
0 to 5 mm Hg. Increases in IAP above 10 mm Hg are clinically significant, and above 15 mm Hg can result in an abdominal compartment syndrome, which affects multiple organ systems.

The cardiovascular manifestations can be understood via the following simple relationship, which expresses the determinants of blood pressure:

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\text{Mean Arterial Pressure (MAP)} = \text{Cardiac Output (CO)} \times \text{Systemic Vascular Resistance (SVR)}
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Pneumoperitoneum causes an increase in SVR while causing a decrease in CO. However, MAP is increased overall because increases in SVR exceed decreases in CO. These effects are proportional to the increase in IAP. The mechanism for increased SVR is compression of the abdominal organs and vessels. Resistance to flow through arterial beds is increased due to both mechanical and neurohumoral factors (e.g., release of catecholamines and vasopressin, and activation of the renin-angiotensin system).

The cardiac output changes during laparoscopy appears to be phasic. It begins with early transient phase where the venous return and cardiac filling pressures increase because of the compression of splanchnic bed. The decreases in Cardiac Output occurs during the next steady state, due to decreased venous return (i.e., decreased cardiac preload) from compression of the inferior vena cava, from increased resistance in the venous circulation. Cardiac Output typically decreases from 10 to 30%. However, despite a decrease in intracardiac blood volume, intracardiac filling pressures may be elevated due to pressure transmitted across the diaphragm to the heart. There are analogous effects in the pulmonary circulation that manifest as an increase in pulmonary vascular resistance (PVR) and decrease in CO to the lungs.

Although hypertension is typical with Pneumoperitonium, hypotension can also occur, particularly with IAP ≥20 mm Hg. In this instance, hypotension may be due to compression of the inferior vena cava impeding venous return that causes a reduction in cardiac output and blood pressure. Intermittent positive pressure ventilation, by causing increased intrathoracic pressure, can also impair venous return and cardiac output, particularly if positive end-expiratory pressures are applied.

Healthy patients appear to tolerate these hemodynamic effects well. Several studies demonstrate that end organ perfusion is maintained in these patients despite a decrease in CO. However, patients with cardiac disease may be at increased risk for further cardiac compromise. Patients with intravascular volume depletion appear to tolerate these effects the least well.

To minimize these effects, the lowest insufflation pressure required to achieve adequate surgical exposure should be used. Ideally, insufflation pressure should be less than 15 mm Hg. Increases in SVR may be treated with vasodilating agents, centrally acting alpha-2 agonists, or opioids. Decreases in venous return and CO may
be attenuated by appropriate intravenous fluid loading prior to the induction of pneumoperitoneum.

B. Hemodynamic Effects of Positioning

In supine position with the Trendelenburg position generally increases venous return and Cardiac output. However, in the presence of a pneumoperitoneum, venous return and CO are decreased overall. If the patient is placed in extreme Trendelenburg, a decrease in venous return from the head may result, thus leading to increased intracranial and intraocular pressures. If this position is maintained for an extended duration, cerebral edema and retinal detachment may occur. Because of venous stagnation, cyanosis and edema in the face and neck may be expected. The head up (reverse Trendelendburg) position reduces venous return, which may lead to a fall in cardiac output and arterial pressure. The lithotomy position will induce autotransfusion by redistributing blood from vessels of the lower extremities into the central body compartment, which thus will increase the preload of the heart.

C. Cardiovascular Complications

Bradyarrhythmias, dysrhythmias, and even asystole can occur during insertion of laparoscopic ports or during insufflation of the abdomen. Sudden stretching of the peritoneum can precipitate a sudden, reflexive, and sometimes profound increase in vagal tone. Slow insufflation of CO$_2$ can decrease the risk of arrhythmias. Administration of anticholinergic medications may be appropriate in bradyarrhythmias. If the arrhythmia persists or results in hemodynamic compromise, prompt interruption of the surgery and release of the pneumoperitoneum is indicated.

Pulmonary and Respiratory System:

A. Pulmonary and Respiratory Effects of Pneumoperitoneum

Pneumoperitoneum transmits pressure to the thorax. The upward pressure elevates the diaphragm, compresses the lungs, and impedes expansion of the lungs and chest cavity (i.e., decreases thoracopulmonary compliance). The pulmonary implications of this mechanical effect are two-fold. First, compression of the lungs leads to decreased functional residual capacity (FRC), i.e., the volume of gas remaining in the lungs after a normal exhalation. Laproscopy exacerbates the decrease in FRC that normally occurs under general anesthesia. The decreased end-expiratory lung volume is insufficient to maintain patent alveoli, and thus atelectasis results.

Atelectasis alters the normal relationship between ventilation and perfusion of the lungs. The atelectatic areas of lung are underventilated relative to their perfusion and therefore cause hypoxemia.

Older patients are particularly at risk for atelectasis, because the minimum end-expiratory lung volume that is required to prevent atelectasis - known as the closing capacity - increases with age. Institution of positive end-expiratory pressure (PEEP) can mitigate the decreases in FRC by stenting alveoli open at end expiration.
The second mechanical effect of pneumoperitoneum is that controlled mechanical ventilation is more difficult due to the decrease in thoracopulmonary compliance. Greater airway pressure is required to generate a given tidal volume. Conversely, a mechanically delivered tidal volume will result in higher airway pressures. As with hemodynamic effects, these effects are proportional to the increase in IAP. Thoracopulmonary compliance may be decreased by up to 50% during pneumoperitoneum.

Pneumoperitoneum furthermore causes hypercapnea from systemic absorption of CO₂. In uncomplicated laparoscopy, the partial pressure of arterial CO₂ (PaCO₂) rises on induction of pneumoperitoneum and plateaus from 15 to 30 minutes later, thus signifying CO₂ equilibrium. PaCO₂ can be reliably monitored via analysis of end-tidal gasses. The degree of hypercapnea depends on CO₂ insufflation pressure, but in routine cases under general anesthesia and controlled mechanical ventilation, hypercapnea is easily managed by increasing alveolar ventilation by 10% to 25%. In cases where the degree of hypercapnea becomes unmanageable with hyperventilation alone, the pneumoperitoneum can be temporarily released to allow for CO₂ elimination.

**Pulmonary and Respiratory Complications**

i. **CO₂ Subcutaneous Emphysema.**

Subcutaneous emphysema is the most common respiratory complication during laparoscopy. It is suggested by an increase in end-tidal CO₂ (etCO₂) greater than 25% or an increase that occurs > 30 minutes after abdominal CO₂ insufflation. Subcutaneous emphysema can often be palpated. The cause is extraperitoneal insufflation of CO₂. Although in some cases this is unintentional, in other cases it is required to operate on extraperitoneal structures. The hypercapnea is managed by increasing mechanical ventilation. CO₂ subcutaneous emphysema itself is not a contraindication to extubation at the end of surgery provided that other extubation criteria are satisfied.

ii. **Pneumothorax**

Movement of gas from the peritoneum into the thorax can occur under pressure through weak areas and defects in the diaphragm. The resulting pneumothorax may be asymptomatic, or it may manifest as increased peak airway pressure, decreased O₂ saturation, and hypotension. In severe cases, there can be profound hypotension and cardiac arrest.

Early diagnosis and treatment can be life saving. Surgery should be stopped and the pneumoperitoneum released. Supportive measures should be continued while confirming the diagnosis, either clinically or with chest radiography. Depending on the degree of cardiopulmonary compromise, the pneumothorax may be observed or treated with an intercostal cannula or a thoracostomy tube. After stabilization of the patient, conversion to an open procedure may be indicated.
iii. Endobronchial intubation

Elevation of the diaphragm by the pneumoperitoneum can alter the position of the endotracheal (ET) tube within the trachea. In some cases, the lungs are pushed cephalad such that the ET tube is advanced past beyond the carina and into a mainstem bronchus. When this occurs, only one lung is ventilated. The non-ventilated lung still remains perfused, and as such becomes a large source of intrapulmonary shunt. Endobronchial intubation is suspected when there is a decrease in O$_2$ saturation and pulmonary compliance. The decrease in compliance occurs because a given volume is being delivered into one lung rather than two. The diagnosis is often confirmed by the finding of unequal breath sounds when the lungs are auscultated. The ET tube should then be slightly withdrawn as needed to reestablish two-lung ventilation.

iv. Gas (CO$_2$) Embolism

Gas embolism, although rare, has a mortality rate of nearly 30%. Profound hypotension, arrhythmias, or asystole can occur as a result of a “gas lock” in the vena cava or right ventricle (RV) that interrupts circulation. The major cause is intravascular insufflation of gas from misplacement of the Veress needle or trocar either directly into a vessel or into a parenchymal organ. Risk factors include hysteroscopy, hypovolemia, and a history of prior abdominal surgeries. Gas embolism most frequently occurs on induction of pneumoperitoneum but can occur at any point during surgery.

Initial steps include immediate deflation of the pneumoperitoneum, institution of 100% FiO$_2$, placement of the patient in the left lateral head-down position to remove air from the RV outflow track, and hyperventilation to eliminate the increased PaCO$_2$ caused by the sudden increase in pulmonary dead space. A central line may be required to aspirate gas from the RV. CPR may be required. Hyperbaric O$_2$ treatment should be considered if there is suspicion of cerebral gas embolism.

Renal System

The pneumoperitonum causes decrease in renal blood flow, urinary output, and renal function. It is likely the decrease in renal blood flow relates to a regional effect, increased IAP; The increased intra abdominal pressure also exerts pressure on renal outflow tract, this pressure reflected back to glomeruli and reduces the filtration gradient. Oliguria is often seen and should be described as normal physiological response during laproscopy. Effects are transient and reversible upon desufflation.

however, a systemic cause, decreased cardiac output, stroke volume, or ejection fraction, is also possible.

It is tempting to assume that the reduction in urinary output and renal function are due to the reduction in renal blood flow. However, the precise pathophysiology is unclear and multiple factors are likely present. Increases in antidiuretic hormone (vasopressin), aldosterone, and rennin occur during pneumoperitonium.
Gastrointestinal System

The literature review indicates that gastric and mesenteric insufficiency and ischemia can occur during Pneumoperitonium. Documented decreases in blood flow during Pneumoperitonium include aortic, inferior vena cava, gastric, and mesenteric vessels. However, a systemic cause, such as decreased cardiac output, stroke volume, or ejection fraction, is also feasible. Mechanical compression of the splanchnic capillary beds likely causes an increase in visceral vascular resistance. Increased vascular resistance can lead to vasoconstriction potentially by neural, hormonal, or intrinsic mechanisms.

Liver

The literature shows that hepatic dysfunction can occur with Pneumoperitonium. There is also evidence that hepatic and portal venous pressures increase and flow decreases. It seems likely that hepatic functional alterations relate to these changes in blood pressure and flow. However, a systemic cause, such as decreased cardiac output, stroke volume, or ejection fraction, may also be contributory.

Endocrine System

Pneumoperitonium elicits substantial stress hormonal response, similar to laparotomy. The precise pathophysiology is uncertain, but likely stems from surgical stress and regional pressure and flow alterations due to increases in abdominal pressure. One should also consider that systemic hormonal stress responses might also be a result of decreases in cardiac output, stroke volume, or ejection fraction.

Central Nervous System

Pneumoperitonium can increase cerebral blood flow and intracranial pressure. Right atrial pressure typically increases as IAP and intrathoracic pressures increase during Pneumoperitonium, thus expanding intracranial blood volume, with risk for increasing intracranial pressure. A decrease in cerebrospinal fluid absorption during pneumoperitoneum may also increase intracranial CSF and raise intracranial pressure. Hypercarbia also causes cerebral vasodilatation and increases ICP. Prolonged head down tilt may cause intracranial haemorrhages and retinal detachment.