Anaesthesia for Laparoscopic Abdominal Procedures. Clinical pearls for the safe conduct.

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Laparoscopic surgeries are often described as minimally invasive procedures by surgeons and to a certain extent by the lay public too. This description unfortunately places the anaesthesiologist in a sticky wicket. For any practicing anaesthesiologist, the terminology exactly is a misnomer! To add to the problems, recently laparoscopic procedures are being carried out in neonates, older and sicker patients. The following text is to highlight the challenges and special considerations to be adopted during laparoscopic intra abdominal procedures.

Physiological consequences of pneumoperitoneum are often highlighted as a major concern during laparoscopic procedure. But the anaesthesiologist needs to be vigilant from the time of incision onwards! Veress needle technique using a blind method to first pass a needle followed by a trochar has been reported in literature to cause devastating injuries to aorta or bowel. Also a large volume of insufflating gas inadvertently placed in a wrong plane can cause subcutaneous emphysema. Extensive emphysema involving the entire torso, neck or anterior abdominal wall up to the inguinal ligaments has been reported in literature. Rarely pneumomediastinum, pneumopericardium or pneumothorax is all reported. This happens through congenital or potential communications between the two body cavities or by accidental puncturing of pleura, pericardium or dome of diaphragm. Anaesthesiologist need to have high degree of suspicion regarding these potential problems. Clinical observation of reduced breath sounds, increasing airway pressure, increase in PETCO2 in spite of adequate controlled ventilation, increased PaCO2-PETCO2 gradient or radiological findings complement the diagnosis. The Anaesthesiologist need to be extra vigilant if the surgical colleague is relatively new or in the learning curve for laparoscopy. Fortunately a much more common technique used today is microlaparotomy and placement of trochar under direct vision. With this method, trochar should go fairly smooth without multiple attempts.

After induction, decompress the stomach and bladder. This is to avoid the problem of accidental bladder or Stomach puncture and for improving visualisation.

Points to Ponder: Be vigilant at the time of trochar insertion!! Nasogastric tube, urinary catheter after induction.

Often focussed part of anaesthesia concern is the physiological consequences of pneumoperitoneum. This is akin to methylmethacrylate injection by orthopaedic surgery in which the surgeons administer a substance with wide ranging physiologic sequelae. The gas for inflation is almost always CO2. (As a precautionary measure, the flow gets cut off when
the intra abdominal pressure (IAP) exceeds the limit set by physician usually 12-15 mm of Hg).

The perceived advantages with CO2 are that it is relatively inert, readily absorbed into the blood, don’t support combustion so as to permit use of electrocautery. CO2 is also easily buffered by the principle physiological buffer (bicarbonate) and eliminated via the respiratory system. The consequences of CO2 pneumoperitoneum are twofold. 1) Mechanical 2) Chemical. In addition to the pressure effects of raised intra abdominal pressure, high solubility of CO2 allows rapid changes in the blood gas chemistry.

The effects can cause insult on multiple systems.

**Cardiovascular effects:**

The potential cardiovascular effects are the ones most pronounced at the time of and just after insufflations. In a patient without major cardiopulmonary involvement, it is of multifactorial aetiology. That is due to 1) effects of pneumoperitoneum and raised intra abdominal pressure secondary to it (mechanical effects) 2) position of patient 3) amount of CO2 absorbed and 4) autonomic response of patient. Of course the anaesthetic agents also play its part.

Normal intra-abdominal pressure (IAP) is 0-5mm of Hg. Increase in IAP above 10 mm of Hg is clinically significant, and above 15 mm of Hg can result in abdominal compartment syndrome. The cardiovascular manifestations of pneumoperitoneum are best explained by following equation.

$\text{Mean Arterial Pressure (MAP)} = \text{Cardiac output (CO)} \times \text{Systemic vascular resistance (SVR)}$

Pneumoperitoneum causes an increase in SVR while decreasing CO. The mechanical effect of pneumoperitoneum is compression of the inferior vena cava which causes reduction in venous return to heart (after a transient increase in venous return due compression of splanchnic bed and vena cava). This can cause a decrease in cardiac output. However, increase in SVR more than compensates for the reduction in CO and there is an overall increase in IAP. Compression of major vessels and abdominal organs results in an increase in SVR. In addition to mechanical factors, neurohumoral factors (release of catecholamines and vasopressin, activation of rennin angiotensin) also contribute to the resistance to flow through the arterial beds. The decrease in CO are due to decreased venous return by compression of the inferior vena cava, increased resistance in the venous circulation and from hypovolemia due to preoperative bowel preparation. CO typically decreases from 10 to 30%. Even with reduction in intra cardiac blood volume, intra cardiac
filling pressures are actually elevated due to increased pressure transmitted across the diaphragm to the heart. There is also an increase in pulmonary vascular resistance (PVR) and decrease in CO to the lungs.

Points to ponder: **Increase in right and left filling pressures. Drop in cardiac index as much as 50% in steep Trendelenburg positions!!**

All these negative effects of pneumoperitoneum are tolerated in a healthy adult (with less than 15 mm of Hg) and organ perfusion is maintained. But patients with cardiac disease and volume depleted patients (patients in shock) appear to tolerate least all these changes.

Points to ponder: **To minimize negative effects of pneumoperitoneum-**
1. **Use lowest inflation pressure to achieve adequate surgical exposure.**
2. **Increase in SVR treated with vasodilating agents, centrally acting alpha-2 agonists, opioids.**
3. **Decrease in venous return and Co may be attenuated by appropriate IV fluid loading prior to the induction of pneumoperitoneum**
4. **IAP of less than 15 mm of Hg is an absolute requisite.**

Dysrhythmias and even asystole can happen during insertion of laparoscopic ports or during insufflation of the abdomen. Sudden stretching of peritoneum can precipitate a brady arrest especially in healthy adults with high vagal tone. Unexplained tachycardia, hypertension, dysarrythmias are early warning sign of hypercapnia.

Points to ponder: **Because of the potential for reflex increase of vagal tone, atropine should be readily available.**

Effect of hypercarbia on cardiovascular system in fact is much more complex. Directly it dilates arterioles and depresses myocardium. But on the other hand it enhances sympathetic activity by release of catacholamines.

Points to ponder: **Beware of and guard against bradyarrhythmias:**
1) Slow inflation
2) Release of pneumoperitoneum
3) Anticholinergic medications

Pulmonary effects:

Insufflation of abdomen is generally considered among the stronger indications of endotracheal intubation because of pulmonary changes. The upward displacement of the
diaphragm can predispose to active or passive regurgitations. Upper gastrointestinal tract must be decompressed after intubation.

Pneumoperitoneum leads to elevation of diaphragm and consequently collapse of basal portions of the lung (atelectasis) and ultimately causing a decrease in functional residual capacity (FRC). Atelectasis and reduction in FRC alters the normal relationship between ventilation perfusion ratio (V/Q mismatch), and increase in intrapulmonary shunting of blood (The atelectasis areas of lung are underventilated relative to their perfusion). The older patients are particularly at risk for atelectasis, because the minimum end expiratory volume that is required to prevent atelectasis (closing capacity) increases with age. Consequence of all these is hypoxemia and increased alveolar-arterial oxygen gradient.

Old age patients are more at risk for developing V/Q mismatch and basal atelectasis.

The other effect of pneumoperitoneum is the reduction in thoraco-pulmonary compliance. This means controlled mechanical ventilation is more difficult. Consequently in order to achieve the given tidal volume, more airway pressure is required. Conversely, a mechanically delivered tidal volume will result in higher airway pressure.

Points to ponder: 1) The adverse effects on the respiratory system is proportional to IAP. 2) There may be further exacerbation of these negative effects in restrictive lung diseases and by Trendelenburg position. 3) Chance of Endo tracheal tube slipping endobronchial especially with head down tilt.

Pneumoperitoneum also causes hypercapnea and respiratory acidosis from systemic absorption of CO2. Clinically, moderate to severe hypercarbia can result in premature ventricular contraction, ventricular tachycardia and ventricular fibrillation. In uncomplicated cases arterial CO2 (PaCO2) rises on induction of pneumoperitoneum and will be in equilibrium in 15 to 30 minutes. The degree of hypercapnea depends on CO2 insufflation pressure.

Patients with chronic obstructive pulmonary diseases (COPD) or morbid obesity are at high risk for pulmonary decompensation and for a significant and catastrophic increase in PCO2!

Points to ponder: 1) In routine cases, under general anesthesia and controlled mechanical ventilation, hypercapnea is easily managed by increasing alveolar ventilation by 10-25% 2) In case unmanageable with hyperventilation alone, temporarily release pneumoperitoneum and allow CO2 elimination. 3) Respiratory embarrassment worsened head down tilt and in respiratory compromised patients
4. Allow adequate expiratory time in COPD patients.

All these negative effects of pneumoperitoneum on the respiratory system can be managed by increased frequency of mechanical ventilation and mild positive end expiratory pressure (PEEP), increasing the fraction of inspired oxygen (FIO2). In fact, it is now accepted that a PEEP of 5 cm H2O should be considered essential during laparoscopic surgery to decrease intraoperative atelectasis caused by pneumoperitoneum. The increase in FRC can facilitate gas exchange and oxygenation. Higher PCO2 levels as the case progresses can potentially cause resumption of spontaneous ventilation. To offset this effect, neuromuscular block should be optimal. With minimal increase in minute ventilation (15-20%), PETCO2 can be maintained around 35-40 mm of Hg.

Another crucial point to monitor is an increase in airway pressure. This could be due to pneumothorax! Especially when the procedures are done close to diaphragm (eg: gastric fundoplication)

Whether raised intra abdominal pressure leads to increased risk of regurgitation and aspiration is unclear. It’s logical to conclude so. But it’s also argued that the changes in pressure are transmitted to the lower oesophageal sphincter, preventing regurgitation.

**Points to Ponder:** Aspiration risk!, decompress stomach, Increased Frequency of mechanical ventilation & high FIO2, PEEP of 5cm H2O. Adequate muscle relaxation, Airway pressure monitoring.

CO2 embolism is a rare but devastating complication of Laparoscopic procedure. An increase in ETCO2 is warning sign! This is followed by an acute decrease in ETCO2 in the event of severe hypotension. Embolism happens due to accidental intravascular entry of needle or trochar, excessive intra abdominal insufflations leading to puncture of vessel. But CO2 being more soluble than air; the chance of severe consequence like complete blockade of preload is fortunately less common.

**Intracranial effects:**

Intracranial effects are the easiest to overlook, as it is extremely rare to have direct measurement of intracranial pressure (ICP) during laparoscopy. Trendelenburg positions, decrease in venous return from the head, elevated systemic vascular resistance, hypercapnea can all result in increased intracranial pressure and reduced cerebral perfusion pressures. Intraocular pressure is also increased.

**Points to Ponder:** Extreme Trendelenbug position be cautious about cerebral edema, retinal detachment, cyanosis and edema in the face and head & neck
Neuroendocrine effects:

There are increased levels of stress hormones with initiation of pneumoperitoneum. Increase in levels of ADH and aldosterone were well documented. Though the implications are not clear or minimal, there is immunological and cytokine response to laparoscopy. Cortisol, C-reactive proteins, tumor necrosis factor are all increased.

Hepato renal and splanchnic effects:

In parallel with reduction in cardiac output, there is decrease in glomerular filtration rate and renal blood flow. Raised intra abdominal pressure also contributes to the increased renal vascular resistance resulting in reduction in effective renal blood circulation. Clinically this is manifested as reduction in urine output. Normally in patients without any pre-existing renal dysfunction all these changes are reversible and there is no long term renal sequelae. The urine output improves once intra abdominal pressure comes down.

A matter of concern is fluid management during laparoscopic donor nephrectomy. Despite the theoretical risk of reduction in GFR and effective renal blood flow, animal models showed no adverse histological changes. But as donors are generally healthy and free of cardiopulmonary complications, volume expansion and slight fluid over load is always tolerated by the donor. Alternate is to maintain a low intra abdominal pressure for visualization of intra abdominal contents. However there should be greater concern to avoid over resuscitation, fluid over load, pulmonary oedema and exacerbation of congestive heart failure in an attempt to improve urine output.

Points to Ponder: **Patients with pre-existing renal dysfunction are at risk of further deterioration**

Effects on the hepatic and splanchnic flows are unclear. Vasoconstrictive effect of raised intra abdominal pressure is opposed by the vasodilatory effect of hypercapnia. Elevated hepatic enzyme and bilirubin levels have been reported but return to normal levels in healthy patients. This may not be the case with hepatically compromised patients.

Post operative nausea and vomiting is a predicted complication of laparoscopic procedures and prophylactic use of antiemetic is strongly advocated.

Soft tissue effects:

Patients can occasionally develop subcutaneous emphysema especially during long procedures. Because of high solubility of CO2, considerable quantities can accumulate within the body tissues during long procedures. This need to consider before extubating patients who has undergone extended procedures. Severe subcutaneous emphysema of
head and neck may require extended postoperative intubation to prevent airway obstructions when allowing tissue stores of CO2 is removed. Any increase in ETCO2 more than 25% or occurring later than 30 minutes after beginning of pneumoperitoneum suggest CO2 sub cutaneous emphysema. Elderly patients or patients with pre-existing pulmonary diseases may not be able to sustain the PCO2 induced high minute ventilation and are at high risk of reintubation.

Points to Ponder: **Extubation to be carried out delayed in patients who develop subcutaneous emphysema following long laparoscopic procedure.**

**Taking care of positional injuries:**

Trendelenburg, reverse trendelenburg and lithotomy positions are commonly employed during laparoscopic procedures. Utmost care should be employed to protect and pad pressure points, eyes and prevent over extension of arm. Over extended arm can result in brachial plexus injuries (use shoulder braces!). Improperly padded lithotomy position can result in common perineal nerve injury.

After positioning the patient and tilting the operating table (gradually 15-20 degree at a time) it should be made a habit to check the haemodynamic status, reconfirmation of tube position and bilateral air entry.

Patients are at high risk for development of deep vein thrombosis (DVT) especially with venous stasis in the lower limbs and increased IAP. Head up tilt and lithotomy position also facilitates femoro-popliteal venous stasis. For DVT prophylaxis, physical measure (calf pump) is commonly employed.

Points to Ponder: **Care of positional injuries, DVT prophylaxis, check vitals and air entry after each change of position of patient!**

**Role of Nitrous Oxide:**

Major concerns with use of nitrous oxide are 1) it diffuses into abdominal cavity in concentrations sufficient to support combustion of intestinal gas while surgeon using cautery 2) It will diffuse into CO2 or air bubbles increasing their size in the event of accidental embolism leading to obstruction of pulmonary circulation 3) diffusing into the bowel and causing distension during laparoscopic procedure.

Points to Ponder: **Serious concern with regard to use of nitrous oxide. Use Oxygen, Air, inhalational agent**
Choosing appropriate patient:

In normal healthy patient’s pneumoperitoneum up to IAP 15 mm of Hg is well tolerated with proper ventilation and monitoring. But in patients with pre-existing cardio-respiratory involvement, it can cause significant increase in PaCO2 levels and can cause increased morbidity and mortality. So in high risk cases it’s better to limit pneumoperitoneum time and pressure (IAP) to the point of adequate visualization. So also the change in positions should be cut short to limit haemodynamic fluctuations (Trendelenburg and reverse Trendelenburg).

During laparoscopy there is frequent and abrupt changes in preload and afterload conditions of heart due to raised IAP. These changes are poorly tolerated by patients with severe coronary artery diseases, valvular heart diseases or in fixed output states! So also any fall in coronary perfusion pressure in the event of increased left ventricular work load can lead to myocardial ischaemia or ventricular dysfunction. Patients with retinal detachment, raised intracranial pressure are not good candidates for laparoscopic procedures.

The positive effect of laparoscopy counterbalances the risk of pneumothorax and inadequate gas exchange in pulmonary compromised patients! So that it is preferred over laparotomy even in those circumstances.

**Points to Ponder:** *Laparoscopy appears preferable to laparotomy in patients with respiratory diseases because of reduced post operative dysfunction.*

**Points to Ponder:** *Whether the post operative benefits far exceeds the stress of intraoperative pathophysiological changes?*

**Monitoring:**

All the anticipated problems can be detected and managed early with adequate close monitoring of patient. Of course, one should be well versed with the physiological and biochemical impact of laparoscopic procedure on the body physiology to interpret the warning signals on the monitor! What is considered essential during procedure is electrocardiogram, non invasive blood pressure, airway pressure, pulse oximeter (SpO2), EtCO2 and temperature monitoring. Check the cuff pressure after intubation and keeping the pressure below 30 cm of H2O of will help to prevent post operative sore throat especially after prolonged trendelenburg position. Neuromuscular monitor helps to judiciously dose muscle relaxants especially when surgeon is working near the diaphragm. This will blunt any hypercarbia induced respiratory effort.

Anaesthesiologist should keep a close watch on the intra abdominal pressure. Typically an IAP of around 10-15 mm of Hg is sufficient for adequate visualization of most
procedures. Sometimes higher pressures may be required for visualization transiently; but an IAP more than 20 mm of Hg is really unsafe. Invasive haemodynamic monitoring in high risk cases and prolonged surgery helps not only a close watch on the haemodynamic fluctuations but also for frequent blood gas analysis. Trans esophageal echocardiography may be more helpful in patients with severe cardiac involvement (Hypertrophic obstructive cardiomyopathy)

**Ponits to ponder:** *Use invasive haemodynamic monitoring for prolonged pneumoperitoneum, cardio-pulmonary, renal compromised patients*

**Summary:**
Safe conduct of anaesthesia for intra abdominal laparoscopic procedure involves thorough understanding of the pathophysiological and blood chemistry changes expected to happen during pneumoperitoneum. Thorough screening of patient’s cardio-pulmonary status is essential before procedure. Anaesthesiologist must have the knowledge and foresight to see the complications and deal with any that is likely to happen. Proper monitoring of haemodynamics and respiratory parameters are very critical for detection of any warning signs. As with any surgical procedure, it’s a team work and anaesthesiologist must be able to share his/her concerns with the surgical team in high risk patients.

**Further reading:**