REVIEW ARTICLE

Pneumoperitoneum : Sailing in tsunami!

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SUMMARY :

Laparoscopic surgeries are the theme of today's surgical practice, which requires pneumoperitoneum. The complex patho-physiological changes following pneumoperitoneum is well tolerated in healthy adults in contrast to cardio-pulmonary compromised patients. This comprehensive review article helps in understanding the basis for changes in cardiopulmonary system.

KEY WORDS :

Hypercarbia, Intra-abdominal pressure, Laparoscopy, Pneumoperitoneum

INTRODUCTION :

Minimal access surgery is rapidly replacing traditional open surgeries[1].The advantages[1,2,] include minimal post operative stay, less invasive procedure and early return to normal life, which may provide economical advantages as well to the patients. Although history dates back to a century, only in late 80s became a regular practice in surgery. Significant changes in the homeostasis after pneumoperitoneum and position used for laparoscopic surgeries are expected. Hemodynamic changes are due to combined effects of mechanical and neuro-humoral factors[1,2,3.] The patho-physiology of pneumoperitoneum has been discussed completely in this article.

Pneumoperitoneum: Pneumoperitoneum is a complex patho-physiologic state, normally well tolerated by young healthy subjects. Before going in-depth, we will briefly go through the procedure involved in creating pneumoperitoneum; Using small incision, verres needle is inserted at the umbilicus and peritoneum is insufflated with carbon dioxide. Depending on the type of procedure up to four ports are used. Depending on the surgery, anesthesia ranges from just an intravenous sedation (for tubal sterilization) to general anesthesia. For abdominal procedures intra-abdominal pressure is not allowed to raise more than 15mm Hg. Many factors govern in choosing the insufflant gas, among them the most important are, solubility in the blood, permeability

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in the tissues combustibility, other systemic side effects and of course the expense.

Why only CO_2? Carbon dioxide[4,5] is favored , as it is colorless, non combustible, highly soluble and permeable in tissues thus reducing the risk of gas embolism. Also less expensive and readily available. The Oswald's blood gas partition co-efficient is 0.48. It is rapidly buffered by bicarbonates in the blood and excreted via lungs. Due its high solubility the incidence of gas embolism is 0.0016 to 0.013 %. However it causes hypercarbia, respiratory acidosis and shoulder pain[6-13]Air is out dated because of incandescent light and increased risk of air embolism[13,14] .Nitrogen is not soluble in blood thus can be a hazardous cause for gas embolism[15]. Nitrous oxide is deleterious as it is slightly combustible and associated with sudden cardiac arrest[15,16]. Both helium and argon are good inert gases but do carries increased risk of embolism,[13-19] as the volume of gas required for embolic episode is very low when compared to carbondioxide[13]. Oxygen is not used, as we all know it is good supporter of combustion.

RESPIRATORY EFFECTS OF CAPNOPERITONEUM :

Effects due to hypercarbia: Carbon dioxide is insufflated at the rate of 1-2L/min. On an average, a total of 25-30L may be insufflated during the procedure. There is about 10mm of Hg raise in P_aCO_2, mixed venous blood PCO_2 and in P_ACO_2 within 5 minutes of insufflation. Hypercarbia may develop due to transperitoneal absorption of CO_2.[19-24] Normal carbon dioxide excretion of 100-200ml/min is increased by 14 to 48 ml after capnoperitoneum. Absorbed CO_2 is rapidly eliminated by lungs in healthy patients. If alveolar ventilation is impaired because of altered respiratory mechanics and cardiovascular system, the additional CO_2 is not eliminated resulting in hypercarbia and acidosis. End tidal CO_2 monitoring used routinely grossly underestimates P_ACO_2, if the $PaCO_2$ is more than 41mm Hg. Generally $PaCO_2$ returns to normal within one hour of peritoneal exsufflation. The rate of CO_2 elimination is dependent on alveolar and mixed venous CO_2 gradient which in turn depends on alveolar ventilation, cardiac output and respiratory quotient. The greater the use of peripheral CO_2 capacity, the longer the duration of elevated $PaCO_2$. Thus in prolonged laparoscopic procedures it may take several hours to achieve a steady state of CO_2 after exsufflation[25-38]. CO_2 pneumoperitoneum under local anesthesia stimulates hyperventilation due to reduced lung expansion especially in lithotomic position and hypercarbia stimulates respiratory center in medulla. However under general anesthesia minute ventilation has to be increased by 12-16% to maintain eucapnia in healthy patients[38]. This compensation might not help in patients with compromised pulmonary function. If hypotension complicates the pneumoperitoneum, there is only little increase in $PaCO_2$ than in normotensive, as hypotensive state reduces peritoneal blood flow there by reduces the transport of CO_2. However the CO_2 tension in the venous blood is more reflecting low lung perfusion and increased CO_2 production by ischemic tissues. Extra peritoneal CO_2 insufflations causes more rapid CO_2 absorption as well as more chance of leak into mediastinum and pleural space as it is not bound by peritoneal membrane.

Effects due to raised intra abdominal pressure : Peak and plateau airway pressures rise by 50 and 81 percent respectively. While pulmonary compliance decreases by 47 %. These changes increase the work of breathing[30-34]. An elevated peak airway pressure is required to maintain minute ventilation volume. Following exsufflation peak and plateau airway pressure remains elevated by 27% and 37% respectively[31-35]. Pulmonary compliance is 86% of pre-insufflations value. Ventilated patients with pulmonary compromise might require PEEP to compensate these changes. Trendelenberg position per se does not appear to exacerbate the rise in airway

pressures. Inspiratory tidal volume is reduced but no significant effects on dead space by capnoperitoneum[36,37]

Diaphragm movements : Diaphragmatic movements are reduced due to increase in intraabdominal pressure and trendelenberg position. These tend to return to normal on exsufflation .The associated increased use of intercostals muscles in addition to reduced diaphragmatic movement reduces FRC and tidal volume. Post operatively the neuronal afferents arising from gallbladder area or from the abdominal wall may exert inhibitory action on phrenic nerve discharge leading to diaphragmatic dysfunction. These changes may assume clinically significant when it is associated with conditions like pneumonia or atelectasis[23-36]

Oxygenation: Capnoperitoneum has minimum effect on oxygenation which is clinically insignificant. Baratz[29] and Karis showed minor decrease in PaO_2 presumably due to altered ventilation in lung bases. In contrast Puri[20] and Singh found no change in ventilated patients maintained in 15°-20° trendelenberg position

Spirometry: Post operatively spirometry is moderately affected. In patients undergoing laparoscopic cholecystectomy FVC and FEV_1 reduced 22% and 21% respectively[34,35]. FEV1 is reduced because of reduced TLC due to cephalad push of diaphragm. FRC decreases by 20% due to capnoperitoneum promoting collapse of the alveoli. Reverse trendelenberg position increase the FRC and presumably compliance[6,7,21]. Intraoperative pulmonary dysfunction is more with laparoscopic procedures when compared with open procedures[2,4,6]. However by minimizing the trauma to skin and abdominal muscles laparoscopic procedures causes less post operative pulmonary dysfunction. Relatively less pain with laparoscopic surgeries leads to less splinting of rib rage, reduction in FRC, tachypnea, shallow breathing and less suppression of cough reflex. All these factors help in reduced pulmonary atelectasis and respiratory infection.

CARDIOVASCULAR EFFECTS OF CAPNOPERITONEUM

Effects due to hypercarbia : Mild hypercarbia 45-50 mm of Hg produces no significant effects. But moderate to severe levels of 50-70 mm of Hg has direct myocardial depressant and vasodilator effect. A moderate decline in cardiac output, stroke volume, systolic BP, and PH due to retained CO2 and are dependent on the volume insufflated and intraabdominal pressure. There is catecholamine mediated increase in heart rate, blood pressure, central venous pressure, pulmonary artery pressure, cardiac output, stroke volume and a decrease in peripheral vascular resistance. These changes are independent of gases used[5, 6,30-35, 38].

Effects due to Intraabdominal pressure: Pneumoperitoneum increases intraabdominal volume and pressure. The hemodynamic effects depend on amount of gas insufflated, baseline hemodynamic status and intravascular volume. Intraabdominal pressure of approximate 14 mm Hg raises systemic vascular resistance, heart rate, caval pressure and mean arterial pressure. Whereas stroke volume falls. These effects are exaggerated by head up position[12,39]. Increased systemic vascular resistance is due to multiple factors which include increased venous resistance, compression of abdominal aorta and increased after load as a result of raised catecholamine, vasopressin and enhanced renin-angiotensin activity[12,43] However trendelenberg position correct the fall in cardiac output and stroke volume by increase in venous return. Initially cardiac output increases due to increase in venous return as a result of splanchnic compression. Within minutes the cardiac output reduces by 25 to 35% due to reduced venous return and myocardial contractility. This is further aggravated by increased SVR. SV reduces due to shift in ventricular function curve to

right perhaps due to increased after load[25-32,34,40]. Pneumoperitoneum reduces blood flow in superior mesenteric artery and portal vein . But the hepatic blood flow is maintained as a result of auto-regulation. However if intarabdominal pressure exceeds more than 20 mm of Hg, there is 60% decrease in portal venous circulation resulting in hepatic dysfunction which persists in the post operative period[25,34,35].Venous blood velocity is reduced due to pooling of blood in lower extremities, promptly returns back to normal with exsufflation[32,33]The combined effects of anesthesia, pneumoperitoneum and head up tilt can reduce the cardiac index up to 50% of the baseline as shown by Joris[43]

Patients with cardiac disease:

Laparoscopic procedures are potentially dangerous in cardiac patients because of the serious hemodynamic derangements induced by pneumoperitoneum and anesthesia. There is reduced venous return, increased SVR, MAP. Due to reduced preload heart rate increases to maintain the CO.

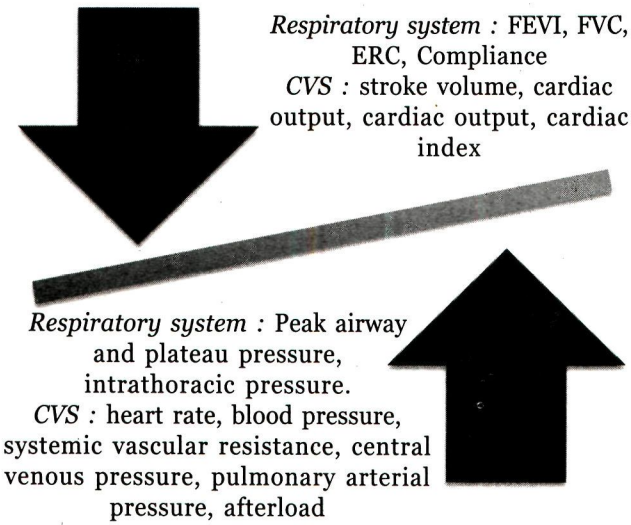

Figure 1: Cardiopulmonary changes after pneumoperitoneum

Concomitantly increased SVR increases ventricular wall tension thus reducing coronary blood flow leading to myocardial ischemia and left ventricular dysfunction. Intravascular volume expansion increases preload, SV and CO. But cardiac patients receiving optimum treatment for CCF have a low preload and after load. In these patients a rapid increase in intravascular volume is disastrous. In a relatively healthy patient undergoing laparoscopic surgery intraabdominal pressure of approximately 14 mm of Hg produces significant increase in HR, MAP, SVR, venous return. SV falls but CO is relatively maintained by increased HR. These changes might push for myocardial ischemia in cardiac patients[2,3,7,39]. A laparoscopic surgery hence not so smooth sailing as described; but has serious cardiopulmonary events as summarized above [figure-1]. Patients with ischemic heart disease thus lie on the threshold of tsunami tide, if not properly managed

Gastrointestinal system: Patients undergoing laparoscopy are usually considered at high risk for acid aspiration syndrome due to gastric regurgitation which might occur due to the rise in intragastric pressure consequent to increased intraabdominal pressure, however during pneumoperitoneum the lower oesophageal sphincter tone far exceeds the intragastric pressure and the raised barrier pressure limits the incidence of regurgitation[39,40,41]

Renal system: Increased intrabdominal pressure affects renal hemodynamics by alterations in CO and direct effect on renal vasculatre. Mechanical obstruction to renal venous flow, increased vasopressin levels , renin angiotensin activity leads to increase in the renal vascular resistance , which results in a reduced filtration pressure and urine output[39].

Effects on ICP and IOP: Intrabdominal pressure increases lumbar spinal pressure by reducing drainage from lumbar spinal plexus, thus increasing IOP and ICP. Hypercarbia itself produces cerebral vasodilatation leading to further increase in ICP.

Variations in temperature: During laparoscopy continuous flow of dry gases over the peritoneal surface can lead to loss of heat from the

body. There is 0.3° C decreases in core temperature per 50L volume flow of carbon dioxide during capnoperitoneum[5,6]

Neurohumoral stress response: Pneumoperitoneum stimulates stretch receptors of the peritoneal cavity along with hypercarbia enhance the sympathetic nervous system activity. Five minutes after the beginning of pneumoperitoneum, there is marked increase of vasopressin and neurophysin. Plasma concentration of vasopressin then decreased, where as plasma concentrations of neurophysin is plateaued. The profile of vasopressin release closely resembled the time course of changes in SVR. Plasma concentrations of epinephrine, norepinephrine and rennin also increased during laparoscopy, but these changes are more progressive than the increase in SVR. Plasma concentration of cortisol gradually increased during surgery and immediate post operative period. Plasma concentration of endothelin, TxB_2, 6-keto-$PGF_{1\alpha}$ and PGE_2 do not change significantly throught the procedure[2,3,8,43]

Thromboembolism: Intrabdominal pressure of 14mm of Hg, prolonged duration of surgery and reverse trendelenberg position causes venous stasis in the lower extremities. All these factors might increase the risk of thromboembolism.

Complications: Pneumoperitoneum can open up embryonic channels to pleura, mediastinum and pericardium resulting capnothorax, capnomediastinum and capnoperitoneum respectively. Sudden hypoxia, hypercarbia, altered hemodynamic status, and abnormal movement of hemidiaphragm in laparoscopy view should raise the suspicion of pneumothorax[42].

Subcutaneous emphysema[4] occurs if the verres needle do not penetrate peritoneal cavity properly before insufflation. The gases may accumulate in the subcutaneous tissue plane; there is increased CO_2 absorption than intraperitoneal insufflation which is reflected by increased $EtCO_2$ and increase in airway pressures. Venous gas embolism although very rare if occurs, is a potential life threatening complication. Gas may enter the circulation if the verres needle directly punctures the vessel. In majority of cases no such evidence is found. The prognosis depends on the rate of gas entry as well as size of the bubbles[4] Other complications include reflex increase in vagal tone due to excessive stretching of peritoneum and arrhythmias[4].

Position of the patient during the procedure : For peritoneal insufflation about 15°-20° trendelenberg position is used initially. After achieving capnoperitoneum steep reverse trendelenberg position with right lateral tilt is used to facilitate gall bladder retraction. The trendelenberg position facilitates movement of mobile intrabdominal viscera due to gravity there by reducing chances of injury during veeres needle insertion. The trendelenberg position increases venous return, right atrial pressure cardiac output .CVP is increased by 8mm of Hg with 15mm of Hg Intrabdominal pressure in supine position. In trendelenberg position an additional 6mm of Hg increase is noted[5,6]. FRC, TLC and tidal volume. And lung compliance is reduced. There is potential risk of endobronchial intubation due to cephalic movement of diaphragm and carina. Nerve compression is possible in prolonged trendelenberg position particularly brachial plexus. There is increased ICP and IOP; with the trendelenberg position[3,7,8,15] is used in gynecological surgeries. The lithotomy position further exaggerates the changes seen in trendelenberg position[12].For laparoscopic cholecystectomy after initial trendelenberg position; position is changed to steep reverse trendelenberg plus right 20°-30° lateral tilt. This is more favorable for respiration. However there is reduced venous return, right atrial pressures, PCWP. These changes lead to reduced CO and MAP. Compression of inferior venacava further worsens

the situation. The venous stasis leads to increased risk of thromboembolism. In a series of 13 patients undergoing laparoscopic cholecystectomy use of TEE has shown reduced left ventricular end-diastolic area in reverse trendelenberg position indicating reduced venous return. LVEF has be shown to maintain throught the procedure in an otherwise healthy patients[3,22,35,43]

GLOSSARY :

CO_2-Carbon dioxide, CO-cardiac output, DBP-Diastolic blood pressure, $EtCO_2$- End tidal carbon dioxide, ICP-intra cranial pressure, IAP-Intrabdominal pressure, IOP- Intraocular pressure, HR-Heart rate, LVEF-left ventricular ejection fraction, MAP-mean arterial pressure, P_aCO_2-Partial pressure of arterial carbon dioxide, P_aO_2- Partial pressure of arterial oxygen, P_ACO_2- Partial pressure of alveolar carbon dioxide, P_AO_2-partial pressure of alveolar oxygen, PAP-Pulmonary artery pressure, PCWP-pulmonary capillary wedge pressure, PVR-Pulmonary vascular resistance, SBP-systolic blood pressure, SVR-Systemic vascular resistance, SV-stroke volume.

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