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REVIEW ARTICLE

CO_2-Pneumoperitoneum in Laparoscopic Surgery: Pathophysiologic Effects and Clinical Significance

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ABSTRACT

Background: Knowledge of the pathophysiological basis of laparoscopic procedures, in particular the impact of CO_2-pneumoperitoneum (PNP) on the body, can prevent onset of complications during laparoscopy.

Design and Methods: Standard intra-abdominal pressure (IAP), which is used during laparoscopic surgery, is 12 to 15 mm Hg. The direct effect of CO_2-pneumoperitoneum is a consequence of the mechanical action of the gas, and increased intra-abdominal pressure. The indirect effect of CO_2-pneumoperitoneum caused by the absorption of gas from the abdomen. Analysis of articles that evaluated the effects of CO_2-pneumoperitoneum on the body and intra-abdominal organs contributes to an even better use of the laparoscopic method.

Results: The results of numerous experimental and clinical studies have confirmed that increased IAP and CO_2-pneumoperitoneum intraoperatively causing reduction the portal venous blood flow, increasing venous stasis, reduced glomerular filtration, reduced Tiffeneau-index and pulmonary compliance what it can lead to hemodynamic and cardiac disorders. Consecutive intraoperative acidosis and hipercarbia impact the function of intra-abdominal organs and heart.

Conclusion: To avoid the side effects of CO_2-pneumoperitoneum, which is important in patients with ASA II and more often as necessary to be operate with low pressure (IAP: 6-8 mm Hg) or use gasless laparoscopy.

Keywords: Laparoscopic surgery, CO_2-pneumoperitoneum, Hepatic, Renal, Pulmonary and cardial changes, Venous stasis.

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INTRODUCTION

The pneumoperitoneum (PNP) is the crucial element in laparoscopic surgery. The surgeons performing laparoscopy should understand the basic physiologic changes occurring during PNP, recognize the clinical changes and make appropriate intraoperative adjustments to minimize the adverse changes.

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Controlled intra-abdominal pressure (IAP) within the abdominal cavity is tasked to facilitate smooth operation of the surgeon, raising the anterior abdominal wall up and suppressing the other abdominal organs and soft tissues of the back. Georg Kelling's first 1901th described technique of establishing PNP and first did a review of the method of the abdomen, which was then named after him celioscopy, now known as laparoskopy.[1,2] It is believed that Zollikofer, in 1924, first described the use of CO_2 for establishing PNP. Pneumoperitoneum with CO_2 has been used in clinical practice, since the introduction of laparoscopic cholecystectomy in the late 1980s.

Carbon dioxide is the most suitable gas for insufflation into the abdominal cavity, because it meets several important criteria: not flammable and it is possible to use electrocoagulation, very soluble in blood and tissues, it is easily eliminated through the lungs, is nontoxic and it is inexpensive.[3,4]

PHYSIOLOGIC EFFECTS OF CO_2-PNEUMOPERITONEUM

Physiological changes that occur in the body during laparoscopy are the result of different influences. First of all, present the effects of increased IAP on the body, the effects of the gas absorption, and should not be neglected surgical trauma caused by the surgery itself.[3,4] To a certain gas was used to establish PNP in laparoscopy must have certain qualities and meet certain criteria (Table 1).

CO_2 as an inert gas, which is found in the body and is very cheap, most are in use and more convenient than nitrous oxide.[5,6] Diffusion and decomposition of gas through the organism does not pose a risk to the patient or risk of embolism, since that its elimination from the body via the

Table 1: Features of ideal gas for insufflation in laparoscopy

- Antiknock
- Fireproof
- Limited ability resorption
- Limited physiological effects on the body after absorption
- Rapid excretion from the body after absorption
- Does not support the occurrence of burns
- Limited physiological effects in the case of intravascular embolization
- Very soluble in blood
- Colorless

Table 2: Comparison of gases used for PNP during laparoscopy

Gas	*Solubility/diffusion*	*Risk*		*Biological effects*
		Burns	*Gas embolism*	
CO_2	+ + +	Yes	Very low	Many
He	– – –	No	Very low	Inert
N_2O	+ +	Yes	Low	Anesthesia
Air	O_2: –	Yes	High	Oxidation
	N_2: +	No	High	Inert

lungs physiological. Using electrocoagulation is allowed since CO_2 is nonflammable. Sometimes, at longer laparoscopic procedures, carbon dioxide may lead to peritoneal irritation and postoperative pain. After being absorbed from the stomach sometimes leads to laparoscopic acidosis with the possibility of cardiac arrhythmias.[3-5]

Some surgeons for short laparoscopic procedures using nitrous oxide (N_2O), which does not lead to peritoneal irritation with resorption and does not lead to acidosis. It should be noted that more nitrous oxide is not soluble in the blood and, in theory, it is very risky in terms of the possibility of gas embolism. The use of electrocautery during N_2O-pneumoperitoneum is associated with the possibility of occurrence of burns because the gas is flammable.[6] The use of other gases, such as helium, argon, xenon and krypton was not entered into routine use in laparoscopy, because they are very expensive, although they are suitable for insufflation because of its properties.[7-9] Comparison of gases used for PNP during laparoscopy are shown in Table 2.

Physiological changes that occur in the body during laparoscopy are the result of different influences. Transient physiologic abnormalities in the body that may result in CO_2-pneumoperitoneum during laparoscopic procedures are shown in Table 3.

Intraoperative Acid-base Balance Changes

Carbon dioxide is currently the most commonly used gas for creating PNP. Significant elevation in serum levels of CO_2 (pCO_2) or end tidal CO_2 levels along with a concomitant fall in serum pH levels, have been observed during CO_2-pneumoperitoneum.[3,4,10] The effects of CO_2-pneumoperitoneum on acid-base balance are shown in Table 4.

Pneumoperitoneum can result in systemic absorption of CO_2 and alteration of acid-base balance. Hypercarbia is primarily due to transperitoneal absorption of intraperitoneal CO_2. In patients with severe cardiac or pulmonary disease has been associated with the development of more profound hypercarbia and acidemia during carboperitoneum that would be otherwise be seen in patients with normal cardiopulmonary function.[10] With respect of changes in oxygenation, the effect of PNP appears to be small and clinically unimportant. Hasukić et al[11] found no significant difference between the two group patients laparoscopic cholecystectomy *vs* open cholecystectomy (LC *vs* OC) in $PaCO_2$ and pH undergoing LC and OC 24 hours after operation (Table 5).

Pulmonary Changes

Physiologically, when the act breaths due to increased IAP, there is shifting abdominal wall forward. The muscles involved in this process are primarily the diaphragm, intercostal muscles and sternocostal, assisted and pectoral muscles. Expiratory flow is a passive process, with the exception of lung obstruction, when in the process include the expiratory abdominal muscles, and when this process becomes active.[12] Pains in the laparotomy wound and prevent these excursions too passive expiration converted to active process.[11-13] Changes in lung function during laparoscopy caused by PNP are shown in Table 6.

The underlying cause of respiratory function changes in the lung is able to reduce respiratory movements of the diaphragm resulting in decreased respiratory lung expansion. Reduce lung compliance caused a reduction in total lung capacity, functional residual capacity and residual volume[11,13-15] reduced vital capacity (VC), functional residual capacity (FRC) and forced expiratory volume in the first second (FEV1) are well documented.[10,13,15,16]

Intra-abdominal pressure value of 15 mm Hg reduces respiratory compliance by 50%.[16] Absorption of CO_2 leads

Table 3: Physiologic effects of PNP during laparoscopy

- Intraoperative acid-base balance changes
- Pulmonary changes
- Cardiovascular and hemodynamic changes
- Hepatic function changes in intraoperative portal venous flow
- Venous stasis
- Changes in intracranial pressure

Table 4: The effects of CO_2-pneumoperitoneum on acid-base balance

Function	*Status*
Arterial blood gas	
$PaCO_2$	↑
PaO_2	Unchanged
Bicarbonate	↓
Base excess	↓
pH	Unchanged/↓

Table 5: Mean (SD) values for blood gas measurements and pH preoperatively and 24 hours after laparoscopic and open cholecystectomy[11] (LC and OC)

	Preoperative		24 hours after operation	
	Laparoscopic (N = 30)	Open (N = 28)	Laparoscopic (N = 30)	Open (N = 28)
$PaCO_2$ (kPa)	4.3 (0.8)[a]	5.0 (0.5)	4.6 (1.1)[b]	5.4 (0.6)[b]
PaO_2 (kPa)	13.1 (4.5)	11.1 (0.8)	11.9 (3.5)[b]	10.4 (0.9)[b]
pH	7.4 (0.05)	7.4 (0.03)	7.4 (0.03)[b]	7.4 (0.02)[b]

[a]Values are mean (SD); [b]Not significant

Table 6: Pulmonary changes during laparoscopy

Function	Status
Respiratory mechanics	
Peak inspiratory pressure (PIP)	↑
Respiratory compliance	↓
Ventilatory changes	
Respiratory rate	↑
Tidal volume	↓
Minute ventilation (MV)	↑
Tiffeneau-index	↓

to hypercapnia, which along with hypoxemia may lead to transient acidose.[15-17] Position the patient on the operating table can also affect the respiratory disturbances during laparoscopy.[16,17] These changes were more pronounced in patients with cardiac and pulmonary diseases.

Diaphragm contractility reduce hypoxemia, acidosis, neuromuscular disease, malnutrition or surgery in the upper abdomen. Disturbances that have arisen directly affect the exchange of gases and which is becoming limited as it manifests itself through various ventilatory disorders.[11,12,17] In laparoscopic procedures, the intensity changes of respiratory function is less, recovery and normalization of spirometry significantly faster. Problems that might occur due to PNP, CO_2 absorption, and the occurrence of intraoperative hypercapnia and acidosis, quickly solve the need for cessation of gas.

Postoperative pulmonary ventilation disorders have a restrictive character.[11,14-17] The Tiffeneau index, the ratio between forced expiratory volume in 1 second (FEV1) and forced vital capacity (FVC) help to differentiate airflow limitations from restrictive abnormalities.[11,14-16] A meta-analysis of the literature was carried out, concerning the postoperative pulmonary function in both surgical techniques, focused on the Tiffeneau index. Hasukić et al[11] found a significant decrease in FEV1 and FVC on postoperative day 1 in both the LC and OC groups, but the decline was more important in the former group. In the current study, a significant decrease (15.5%) in $FEF_{25-75\%}$, as compared with preoperative values, was observed only on postoperative day 2 in the patients who underwent open surgery (Table 7).

Crema et al[18] found that esophagogastric surgery causes a transitory decrease in pulmonary function, and that this reduction is less pronounced in laparoscopic surgery than in open surgery. Damiani et al[19] in their meta-analysis of the Tiffeneau index in patients undergoing laparoscopic and open cholecystectomy suggests that the laparoscopic compared with the laparotomic technique can reduce airflow limitations and avoid postoperative pulmonary restriction. Laparoscopic cholecystectomy is associated with a significantly shorter hospital stay and a quicker convalescence compared with the classical OC.[20]

Cardiovascular and Hemodynamic Changes

Effects of increased IAP on the heart and major blood vessels, and hemodynamic disturbances which may occur, are generally well-tested. Patients during laparoscopy occur following physiological changes: decreased venous flow to the heart, increase in systemic vascular resistance is and increase in intrathoracic pressure.[3,4,12,16,20] Cardiovascular and hemodynamic changes caused by PNP are shown in Table 8.

Intra-abdominal pressure support splanchnic vasoconstriction and reduction in blood flow through the inferior cava vein, renal and portal vein, and all results in decreased venous flow to the heart.[3,4,10,16,26,27] An increase in systemic vascular resistance (SVR) is a consequence of activation neurohumoral vasoactive systems, which include: sympathetic activity, secretion of antidiuretic hormone (ADH) and the renin-angiotensin-aldosterone system (RAAS).[21-23] With increasing IAP, an increase of central venous pressure (CVP). PNP with moderately elevated IAP causes an increase in intrathoracic pressure, thus increasing the

Table 7: Mean (SD) values for pulmonary function tests preoperatively and 24 hours after laparoscopic and open cholecystectomy[11]

	Preoperative		24 hours after operation	
	Laparoscopic (N = 30)	Open (N = 28)	Laparoscopic (N = 30)	Open (N = 28)
FVC	3.5 (0.9)[a]	3.6 (0.8)	2.9 (1.0)[1]	2.4 (0.6)[2]
FEV_1	3.1 (0.7)	2.9 (0.6)	2.3 (0.8)[3]	1.4 (0.7)[4]
$FEF_{25-75\%}$	1.9 (0.9)	1.8 (0.4)	1.6 (0.7)[b]	1.4 (0.5)

FVC: Forced vital capacity; FEV1: Forced expiratory volume in 1 second; $FEF_{25-75\%}$: Aid expiratory phase of forced expiratory flow; [a]Values are mean (SD); [b]Not significant; 1 *vs* 2, $p < 0.01$; 3 *vs* 4, $p < 0.0001$

CVP.[24,25] Significantly greater increase in IAP squeeze blood from intra-abdominal organs in the venous reservoir. In these situations, an increase CVP does not reflect the real situation of effective blood volume.[21] Increased IAP leads to an increase in pulmonary vascular resistance to 65% of its normal value.[4,10,21]

The organism can occur and significant hemodynamic changes: increase heart rate, increase blood pressure and systemic vascular resistance[4,12] with increasing IAP, an increase in mean arterial pressure. Increase in systemic arterial pressure leads to an increase in intracranial pressure.

Hemodynamic stress response to the CO_2-pneumoperitoneum leads to increased O_2 consumption at the level of the heart muscle, which is very harmful for patients with heart disease. Effect of CO_2-pneumoperitoneum on cardiac output is reflected in the increase of the same or unchanged. In patients with hypovolemia venous flow to the heart is even smaller. Sufficient crystalloid volume replacement may improve venous flow to the heart and reduce the hemodynamic changes.[4,10,21] The insufflation of gas into the peritoneal cavity can provoke arrhythmias. Their incidence is as high as 14 to 27% of laparoscopies which is higher than in 'open' surgery.[28]

The increase in plasma renin activator in the blood plays an important role in the regulation of blood flow in the body during laparoscopy. Antidiuretic hormone (ADH) controls blood pressure through the receptors in the wall of blood vessels and plays a key role in the regulation of blood flow in the body during laparoscopy.[4,16,21,28]

Hepatic Function and Changes in Intraoperative Portal Venous Flow

An IAP of 15 mm Hg used in laparoscopic surgery is higher than the normal portal blood pressure (7-10 mm Hg). This PNP could therefore reduce portal flow and cause alteration in liver function. In healthy patients, increased IAP of 10 mm Hg at l5 mm Hg leads to a significant reduction in blood flow through various abdominal organs: the stomach by 54%, jejunum by 32%, in the colon for 4%, in the liver by 39%, peritoneum by 60 and 11% for duodenum. Splanchnic ischemia time was identical duration increased IAP.[27,29] The reduction of blood flow in the mesenteric blood vessels can occasionally lead to mesenteric ischemia.[27]

During the PNP of 12 mm Hg in dogs demonstrated a lower blood flow through the liver, the superior mesenteric artery and portal vein, 24% of preoperative values.[27] Reduction of intra-abdominal organ perfusion can be viewed as transitory effects of increased pre sure in the abdomen, which operates in three places: the liver and portal vein, splanchnic region and kidneys (Table 9).

Hepatic perfusion is characterized by a unique autoregulatory mechanism known as the 'hepatic arterial buffer response' (HABR), which indicates the relationship between the flow through the portal vein and hepatic artery. Reduced flow through the portal vein leading to reduced flow resistance through the hepatic artery, which thereby increases the allocation of liver blood and *vice versa*. It is proved that this mechanism automatically regulates blood flow through the liver is sufficient for normal IAP.[30]

In animal and human studies, the increased IAP at 15 mm Hg has been shown to reduce portal venous flow.[31] In an experimental study with rats, Richter et al[32] documented that during an IAP of 12 to 15 mm Hg, the rats experienced a loss of physiologic hepatic blood flow control (HABR). High-pressure PNP and its consequent intra-abdominal hypertension-induced hepatic ischemia appear to be the cause. These changes are clinically silent in patients with normal liver function. However, in patients with pre-existing liver dysfunction, these changes may be associated with a significant clinical course.[33-35]

In a clinical study of LC, Jakimowicz et al[36] reported a 53% reduction in portal blood flow with abdominal insufflation to 14 mm Hg. A reduction in portal venous blood flow during PNP may lead to hepatic hypoperfusion and acute hepatocyte injury. Hepatic hypoperfusion can lead to transient elevation of liver enzymes. Hasukic et al[35] reported transient increases in the level of hepatic transaminases

Table 8: Effects of PNP on cardiovascular and hemodynamic changes

Function	*Status*
Hemodynamics	
Heart rate	↑
Mean arterial pressure	↑
Cardiac function	
Cardiac output	Unchanged/↓
Stroke volume	↓
Systemic vascular resistance	↑
Filling pressures	
Mean pulmonary artery pressure	↑
Pulmonary artery wedge pressure	Unchanged
Central venous pressure	↑

Table 9: Hepatoportal and splanchnic changes during laparoscopic surgery

Function	*Status*
Intraoperative changes	
Portal venous flow	↓
Splanchnic flow	↓
Postoperative liver enzymes	
Aspartate aminotransferase (ASAT)	↑
Alanine aminotransferase (ALAT)	↑
Alkaline phosphatase (ALP)	Unchanged
Gamma-glutamyl transpeptidase (GGT)	Unchanged

(ALAT and ASAT) after LC, which returned to baseline by 48 hours postoperatively after comparison of postoperative hepatic function between LC and OC. Hasukić showed a worsening of the postoperative liver function tests (LFTs) when they compared high- and low-pressure PNP (14 *vs* 7 mm Hg) (Table 10).[37]

Many other studies have also confirmed the transient disturbances in liver enzymes after LC. Elevation of hepatic transaminases (ALAT and ASAT) occurred after LC but were transient and clinically silent in patients with normal liver function.[38-40] Laparoscopic cholecystectomy performed under a low pressure PNP or gasless LC using abdominal wall retractors might be feasible in patients with hepatic disfunction or cirrhosis.[41-43]

Application of laparoscopic procedures in the lower abdomen, such as laparoscopic colon resection does not lead to an increase in aminotransferase. This suggests that increased IAP due to a PNP, although important, is not the only cause of reduced blood flow through the liver during laparoscopic procedures. Location, type and extent of surgical organ manipulation represent an additional factor that locally in the tissues lead to certain disorders.[33] In their study, Ahmad[43] suggests that mild-to-moderate elevation in preoperative LFTs may not be associated with any deleterious effect, and, in the absence of clinical indications, routine preoperative or postoperative liver function testing is unnecessary.

Changes in Renal Function

Renal function during laparoscopy characterized by increased vascular resistance, with a secreted vasopressin and reduced 'cardiac output', leading to a reduction in renal blood flow and reduced glomerular filtration. The increase in plasma renin activator in the blood plays an important role in the regulation of blood flow in the body during laparoscopic surgery.[44] The increased IAP during laparoscopy has been shown to alter renal function (Table 11).

A reduction in intraoperative urine output has been well documented during laparoscopic operations.[44-46] The mechanism for oliguria is related to the acute increased IAP.[23] In contrast to these studies, Micali et al[46] compared 31 patients who underwent laparoscopic procedures with 28 similar patients treated by the open method. They found no difference in urinary N-acetyl-beta-(D)glucosaminidase levels in the urine and concluded that no significant renal tubular injury occurs during PNP.

Although this decrease in renal blood flow is well documented, it is unclear whether this is of any clinical significance. It is likely that these changes in renal blood flow are not significant in healthy patients under most normal conditions, but may be important in cases wherein renal blood flow is already compromised. Although the data demonstrate that renal function is decreased during PNP, the clinical significance of this phenomenon is not certain because it appears that renal function returns to normal after PNP is released.[34,45,46]

Venous Stasis during Laparoscopic Surgery

The true incidence of deep venous thrombosis after laparoscopic compared with open operation is unknown; however, some of the factors relating to Virchow's triad (endothelial injury, hypercoagulability and venous stasis) are altered during laparoscopy. The main factor that is adversely affected during laparoscopy is venous stasis. The increased IAP and reverse Trendelenburg position during laparoscopy have been shown to reduce femoral venous flow.[47,48] Nguyen et al[49] reported that increased IAP and reverse Trendelenburg positioning are independent factors that resulted in decreased femoral peak systolic velocity.

Many studies evaluating D-dimer during PNP and postoperative. D-dimer values were significantly higher in the examinees who underwent LC than in those operated by the classical method. In every subsequent measurement those values increased, particularly seen in the laparoscopic group of patients in the measurements taken after the 5th day post-surgery. The increase in D-dimer values in the LCH patients was far more expressed after the operation in the period after

Table 10: Doubling of alanine aminotransferase (ALAT) and aspartate aminotransferase (ASAT) from preoperative values 48 hours after operation in low- and high-pressure laparoscopic cholecystectomy (LC)[35,37]

Liver function tests		*Doubling values 48 hours after surgery*		*p-value**
		Patients	*%*	
ALAT				
	SPLC (N = 25)	11	44	0.0029[a]
	LPLC (N = 25)	2	4	0.0001[b]
	OC (N = 50)	5	10	0.0069[a]
ASAT				
	SPLC (N = 25)	8	32	0.0069[a]
	LPLC (N = 25)	0	0	
	OC (N = 50)	6	12	0.0004[b]

*Chi-square test; LPLC: Low-pressure laparoscopic cholecystectomy; SPLC: Standard-pressure laparoscopic cholecystectomy; OC: Open cholecystectomy; [a]SPLC *vs* LPLC; [b]LC *vs* OC the 5th day, when there was no prophylaxis, while in the course of and 24 hours after the operation, during the period of active prevention, this increase was insignificant.[50,51]

Increased IAP, and with the presence of reverse Trendelenburg's position (head up) patient who is present in the majority of laparoscopic procedures increases venous stasis in the lower extremities and reduces the return of blood from the lower extremities for more than 40%. Potential risk of deep vein thrombosis is present in these patients.[47]

The negative effects of venous stasis in the lower extremities during laparoscopic procedures are given suppress the use of lower limb compression elastic bandages and giving prophylactic low-molecular heparin.[51] Millard et al[52] and Schwenk et al[53] reported that the use of sequential compre-ssion devices (SCD) during LC was effective in reversing the reduced femoral systolic velocity to baseline values. Effects of low-pressure or gasless laparoscopy on thromboembolic complications during laparoscopy are not well tested but expect the positive effects of these methods for the prevention of thromboembolism during laparoscopy.

Intracranial Pressure during PNP

During laparoscopic procedures, increased IAP leads to increased intracranial pressure (ICP), disrupts the flow of blood through the intracranial blood vessels and leads to abnormal resorption of cerebrospinal fluid. Increased ICP is quickly returning to normal after a gas leak from the abdomen. It was not proven that there are specific clinical consequences of increased ICP during laparoscopy.[54] Pathophysiological studies suggest that the increase in IAP leads to abnormal venous drainage of the lumbar venous plexus, which has a direct impact on reducing the absorption of cerebrospinal fluid during insufflation gas into the abdomen. Consequently, hypothetically possible that increased IAP directly leads to an increase in ICP which results in systemic pressure increase caused by the action of CNS. However, the exact pathophysiology of increased ICP during PNP remains unknown so far.[55] Experimental and clinical studies have established that the hemodynamic changes in the body during the PNP's followed directly by increasing ICP. Consequently, patients with intracranial injury or increased ICP due to other reasons, are not suitable for intra-abdominal laparoscopic procedures.[56] If needed laparoscopic treatment for these patients is a necessary quality monitoring, with the ability to use gasless laparoscopy, an operation with low pressure (6-8 mm Hg).[57] Using CO_2-pneumoperitoneum during laparoscopy may lead to hipercarbia and acidosis, with an impact on cerebral circulation.[54-56] Hypoventilation and hipercarbia lead to increased ICP compared with hyperventilation and hipocarbia. In acute increase in ICP, hyperventilation can not so effectively reduce ICP.[56] Intermittent pneumatic compression of the lower extremities increases cerebral oxygenation during laparoscopic surgery.[58]

Table 11: Effects of pneumoperitoneum on renal function and intraoperative urine output

Function	*Status*
Intraoperative urine output	↓
Intraoperative hormonal changes/renal function	
Antidiuretic hormone	↑
Aldosterone	↑
Plasma renin activity	↑
Glomerular filtration	↓
Postoperative renal function	
Blood urea nitrogen	↓
Creatinine	↓
Creatinine clearance	Unchanged

CONCLUSION

Increased IAP during laparoscopy has different effects on different abdominal organs acting through two mechanisms: directly and indirectly. The direct effect of PNP is a consequence of the mechanical action of the gas, and increased IAP. The indirect effect of PNP is caused by absorption of CO_2. Increased IAP mechanical effect on all intra-abdominal organs and tissues, bringing different pathophysiological responses, which were mostly transient. Increased IAP supports splanchnic vasoconstriction and reduction of flow through the inferior cava vein, renal vein and portal vein, all resulting in decreased venous flow to the heart. During laparoscopic procedures, increased IAP leads to increased ICP, disrupts the flow of blood through the intracranial blood vessels and leads to abnormal resorption of cerebrospinal fluid. Working with low pressure or gassless laparoscopy proline can drastically reduce the negative effects of increased IAP during laparoscopy.

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