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REVIEW

Effect of laparoscopic abdominal surgery on splanchnic circulation: Historical developments

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Abstract

With the developments in medical technology and increased surgical experience, advanced laparoscopic surgical procedures are performed successfully. Laparoscopic abdominal surgery is one of the best examples of advanced laparoscopic surgery (LS). Today, laparoscopic abdominal surgery in general surgery clinics is the basis of all abdominal surgical interventions. Laparoscopic abdominal surgery is associated with systemic and splanchnic hemodynamic alterations. Inadequate splanchnic perfusion in critically ill patients is associated with increased morbidity and mortality. The underlying pathophysiological mechanisms are still not well understood. With experience and with an increase in the number and diversity of the resulting data, the pathophysiology of laparoscopic abdominal surgery is now better understood. The normal physiology and pathophysiology of local and systemic effects of laparoscopic abdominal surgery is extremely important for safe and effective LS. Future research projects should focus on the interplay between the physiological regulatory mechanisms in the splanchnic circulation (SC), organs, and diseases. In this review, we discuss the effects of laparoscopic abdominal surgery on the SC.

Key words: Laparoscopic abdominal surgery; Splanchnic circulation; Intra-abdominal pressure; Abdominal compartment syndrome; Safe laparoscopic surgery

Core tip: With the developments in medical technology and increased surgical experience, advanced laparoscopic surgical procedures are performed successfully. Laparoscopic abdominal surgery is one of the best examples of advanced laparoscopic surgery (LS). However, it is associated with systemic and splanchnic hemodynamic alterations. Inadequate splanchnic perfusion in critically ill patients is associated with increased morbidity and mortality. For a safe and effective approach in LS, the anatomy and pathophysiology of the splanchnic circulation (SC), and surgical outcomes should be understood. The aim of this review was to evaluate the effects of LS on the SC.

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INTRODUCTION

The concept of laparoscopic surgery (LS) was proposed more than 100 years ago, but its efficient use in gastrointestinal surgery has only occurred within the last 40-50 years. With the developments in LS, many abdominal surgical procedures have been performed using this method. Progress continues in this area of surgery.

LS is associated with systemic and splanchnic hemodynamic changes. Decreased splanchnic perfusion in critically ill patients is associated with increased morbidity and mortality. Underlying pathophysiological mechanisms are still not well understood. Insufficient splanchnic blood flow may be the result of many diseases, treatment modalities and their interplay. Therefore, it is important to appreciate under which experimental or clinical conditions the effects of vasoactive drugs on splanchnic blood flow are assessed. Unfortunately, many of the available monitoring tools for hepato-splanchnic perfusion and metabolism are difficult to apply in clinical settings and interpretation of the obtained results is difficult. Therefore, splanchnic resuscitation concepts have not yet been established. Future research projects should focus on the interplay between the physiological regulatory mechanisms in splanchnic organs, diseases and treatments. The aim of this review was to evaluate the effects of LS on the splanchnic circulation (SC).

ANATOMY AND PHYSIOLOGY OF THE PERITONEAL SPACE AND SC

The peritoneal space is the most extensive part of the extravascular space. The parietal peritoneum that covers the inner surface of the abdominal wall is supported by the fascia transversalis which is a continuation of the endoabdominal fascia. The visceral peritoneum forms the largest part of the peritoneal surface and covers the surface of the internal organs. The parietal peritoneum and visceral peritoneum are an uninterrupted continuation of each other. The peritoneal space is completely closed except for an opening for the fallopian tubes in women. The surface of the peritoneum in adults is approximately equal to the surface of the skin. This surface varies from 1.6 to 2.1 m^2. Lubricity of the parietal and visceral peritoneum is provided by a clear transudate fluid that can reach 50-70 mL in volume.

The physiological pressure within the abdominal cavity is little more than atmospheric pressure. However, even small increases in intraabdominal pressure (IAP) may have adverse effects on renal function, cardiac output, hepatic blood flow, respiratory mechanics, splanchnic perfusion and intracranial pressure. Although IAP can be measured directly *via* an invasive method, a non-invasive bedside measurement of IAP through a urinary bladder catheter is generally used[1].

The intestinal arterial circulation is principally provided by the superior mesenteric artery (SMA) and its branches with collateral pathways from the inferior mesenteric artery (IMA) and celiac artery. The SMA provides blood supply to embryological midgut organs, whereas the IMA provides blood supply to the embryological hindgut organs.

The mesenteric vascular capillary network is different. The vascular capillary network has very low resistance. Therefore, if the main channel is narrowed, there is potential for the development of collateral blood flow. When chronic ischemia develops, a significant amount of other aortic branches will receive blood flow. These branches are the lumbar, intercostal, renal, internal mammary (*via* the deep epigastric branches), middle sacral and hypogastric arteries (*via* the inferior and superior rectal arteries).

Veins are a part of the dynamic and complex system that returns venous blood to the heart against the force of gravity in an upright position. Venous blood flow is dependent on multiple factors such as gravity, venous valves, cardiac and respiratory cycles, blood volume and the calf muscle pump. Alterations in the complex balance of these factors can result in venous pathology[2,3]. Veins are thin-walled, highly distensible and collapsible structures. Therefore, splanchnic venous structures are affected more than arteries when there is an increase in IAP. When an individual is in the upright position, the veins are maximally distended and their diameter may be several times greater than those in the supine position[2,3]. Unidirectional blood flow is achieved *via* multiple venous valves. Some veins such as the inferior vena cava (IVC), common iliac veins, portal venous system and cranial sinuses are valveless[3].

The superior mesenteric vein and splenic vein converge behind the pancreas and the portal vein (PV) is formed. The inferior mesenteric vein opens into the splenic vein or directly into the PV[4]. The PV is divided into left and right lobar branches at the porta before entering the liver. The portal lobar veins lie posterior to the hepatic arteries and the bile ducts. Nutrients carried by the PV leave the liver before the PV enters the liver and then *via* the hepatic vein and IVC which drain into the right atrium. This is known as the portal circulation[5]. The portal circulation is a system containing the luminal gastrointestinal tract, spleen, gall bladder and pancreas, and includes the venous drainage from intraperitoneal organs created by low-pressure (< 10 mmHg).

When portal venous pressure is at least 5 mmHg greater than the pressure in the non-PVs (for example, IVC), portal hypertension (PHT) occurs. Normally, the portal venous system pressure is higher than the caval venous system pressure. Portosystemic collateral vessels develop to equalize the pressure in these two venous systems.

INTRINSIC AND EXTRINSIC MECHANISMS REGULATING THE SC

The blood flow in the different layers of the intestine is

not uniform and is closely related to the function of the tissue. The amount of blood flow increases up to 200% of the baseline value following digestion of a meal and this increase lasts for 2-3 h. The increase in blood flow is greatest in the mucosal layer. In general, autoregulation of the blood flow in these layers is affected by decreased pO_2, pH, osmolarity, increased pCO_2, or by metabolic factors such as adenosine. This autoregulation is essential for tissue oxygen, nutrient supply, and waste discharge[5]. During the rest period, the vascular tone of the gastrointestinal circulation has several control mechanisms. Metabolic autoregulation of the resting state of the enteric circulation plays an important role in the regulation of vascular tone. Neural regulation plays a role in the primary vasodilation and vasoconstriction of the great arteries and arterioles[5]. The SC has intrinsic, neurological and humoral controls[5]. Functional and reactive hyperemia that results from increased oxygen demand and in response to arterial occlusion, respectively, are examples of intrinsic control. Splanchnic arterial and arteriolar vasoconstriction after the stimulation of sympathetic postganglionic fibers of the splanchnic nerves is part of neurological control. Humoral control consists of the vasoconstricting effects of alpha-adrenergic agonists, vasopressin, prostaglandin-F2, angiotensin-Ⅱ, digitalis and the vasodilating effects of beta-adrenergic agonists, prostaglandin-E1, cholecystokinin, gastrin and glucagon.

The splanchnic microcirculation is controlled by precapillary sphincters and arteriolar resistance. The most important local mechanism in the control of the total splanchnic blood flow is arteriolar resistance. Precapillary sphincters control the capillary blood supply. The SC also depends on the systemic blood pressure. Endogenous and exogenous catecholamines in the circulation induce primary vasoconstriction of the mesenteric postcapillary venules, thus regulating splanchnic vascular volume. Autonomic factors include the opposite effects of alpha and beta-adrenergic stimuli that produce vasoconstriction and vasodilatation, respectively. Renin, angiotensin, vasopressin, thromboxanes or leukotrienes induce intense persistent vasoconstriction, as well as non-occlusive mesenteric ischemia that may result in intestinal necrosis.

If there is no serious damage, the normal intestinal circulation can be established again after low blood flow and perfusion pressure. Mesenteric capillaries have 4-5 times less blood flow during their fasting state with respect to their postprandial state. When intestinal ischemia is initiated, the other remaining capillaries become functional. In the case of moderate ischemia, the ischemic tissue increases its capacity to take up oxygen. Thus, an impaired balance in oxygen can be compensated. However, if the blood flow decreases below 30 mL/100 g of tissue or the systemic blood pressure drops below 40-70 mmHg, the oxygen uptake becomes dependent on the flow[6]. Even moderate ischemia of the intestines due to the stretched intestinal wall caused by obstructive colitis may result in irreversible ischemia due to decreased blood flow. High concentrations of xanthine dehydrogenase in the mesenteric endothelium undergoing intestinal reperfusion injury seems to be one of the culprits in this situation[6].

An imbalance in oxygen supply and consumption can result in acute intestinal ischemia. An experimental study demonstrated that the lipid content in enteral formulations may cause intestinal ischemia after the ischemia-reperfusion state[7].

In conclusion, both intrinsic and extrinsic mechanisms regulate the intestinal blood flow. Intrinsic factors include local myogenic and metabolic control, locally produced vasoactive substances and local reflexes. The intrinsic factors are responsible for phenomena such as pressure/flow autoregulation, reactive hyperemia and hypoxic vasodilation. Extrinsic factors are circulating vasoactive substances, sympathetic innervation and systemic hemodynamic changes. The infusion of drugs acting on adrenergic receptors result in intestinal vasoconstriction, whereas pharmacological stimulation of adrenergic receptors is associated with intestinal vasodilation. Angiotensin, vasopressin and endothelin are potent intestinal vasoconstrictors. In low-flow states, activation of the renin-angiotensin axis is a predominant feature of splanchnic vasoconstriction. Mesenteric angiotensin Ⅱ-mediated vasoconstriction can be inhibited by pharmacological maintenance of perfusion pressure with sodium nitroprusside. Splanchnic venous and arterial pressure, gravity, intrathoracic pressure, intra-abdominal pressure, blood volume, and cardiac function are affected by venous and arterial tonus.

HISTORICAL DEVELOPMENTS IN LS

During LS, the abdominal cavity must be insufflated and expanded by a gas in order to facilitate and enlarge the work area. The gas is pumped into the abdominal cavity between the visceral peritoneum and parietal peritoneum which creates a pneumoperitoneum. Air was first used to create a pneumoperitoneum in 1901, when Kelling carried out an experiment on dogs[8]. In 1910, Jacabaeus used air to create a pneumoperitoneum in humans, where he applied air into the abdominal cavity using an injector[9]. The needle insufflation technique was developped by Veres and was used for the first time in 1938, and this method is still used today. In 1944, Palmer monitored IAP and automatic insufflators were introduced in the 1960s by Semm[10,11]. Automatic insufflators provide the ability to monitor and control IAP and 10-15 mmHg pressure is generally sufficient for most laparoscopic interventions.

LAPAROSCOPIC ABDOMİNAL SURGERY AND THE SC

There are direct and indirect effects of LS on the SC. A controlled increase in IAP develops due to the created pneumoperitoneum during LS. Intraperitoneal pressure is

related to the amount of gas supplied to the peritoneum. An increase in IAP produced by insufflation results in direct mechanical suppression of splanchnic blood flow. During laparoscopy, intra-abdominal hypertension (IAH) can result from the pressure to the anterior abdominal wall (due to the trocars and cannulas placed), excessive gas flow, high pressure of the irrigation fluid, misposition of the cannula and/or trocar, folding of insufflation tubing, misplacement of the Veress needle into the intestinal lumen, omentum or retroperitoneum, and inadequate anesthesia or muscular blockade.

For operative laparoscopy, the maximum flow rate capacity of the automatic insufflator should be 9-16 L/min. This capacity is important when dealing with gas leak during aspiration, where insufflation must be performed quickly. Fast gas movements during LS (*i.e.*, inflow and/or outflow) can lead to problems, especially in patients with cardiac and/or respiratory compromise. In patients with advanced age or cardiac/respiratory problems, reflex cardiac rhythm disturbances, hypotension and cardiovascular collapse may occur as a result of achieving rapid and high insufflation pressure[12-16]. For this reason, an automatic flow rate adjustment mechanism must be provided in insufflators.

Air, oxygen, nitrogen dioxide and carbon dioxide have been used to create a pneumoperitoneum. Gases other than carbon dioxide are not currently used today due to the possibility of gas embolism, decreased venous return, respiratory distress, severe abdominal pain and burns during thermocoagulation. Carbon dioxide is colorless, nonflammable, does not affect vision, clean, cheap and can be obtained pure. CO_2 easily passes into the bloodstream due to its high solubility. According to air-contrast radiographic studies, up to 10 mL/min of CO_2 can be absorbed into the circulation.

PATHOPHYSIOLOGICAL CHANGES IN THE SC DURING LAPAROSCOPIC ABDOMİNAL SURGERY

A significant amount of CO_2 is used during laparoscopy and this is absorbed by the peritoneum. Some of the total amount of CO_2 is washed away from the lungs, while some is stored in the body. The human body has a storage capacity for 120 L of CO_2. The bones are the largest storage field and they play a significant role in acting as a repository, especially in the case of chronic hypercarbia. Some of the skeletal muscles and other tissues can also store CO_2. There is a direct relationship between arterial pCO_2 and the amount of CO_2 used in laparoscopy. Elimination of CO_2 is associated with cardiac output and ventilation rate. Therefore, ventilation control by the anesthesiologist can minimize the amount of residual CO_2 during laparoscopy. A large amount of storage in the tissues can escape and enter the circulation in the postoperative period causing hypercarbia[17-19].

The risk of gas embolism can be minimized using CO_2 due to its high solubility and rapid absorption. The risk of gas embolism appears to be 1/65000[20]. The formation of emboli occurs with the placement of the insufflation needle directly into a blood vessel. Carbon dioxide can also enter veins during the dissection. Direct insufflation of the gas into a large vessel may have fatal results. If this occurs, the insufflation should be stopped immediately and abdominal gas should be eliminated quickly. Gas embolism may ocur at any time in patients undergoing laparoscopic liver resection due to high IAP. Gas embolism is a serious complication of laparoscopic liver surgery. The risk of significant embolus under conventional pneumoperitoneum is minimal during laparoscopic liver resection[21]. Thus, low IAP or noninsufflating techniques are recommended.

In addition to massive embolism, cardiovascular collapse may also occur due to decreased venous return to the heart or deep vasovagal responses. The effects of elevated IAP on systemic hemodynamics and splanchnic blood flow created by insufflation of carbon dioxide were examined in anesthetized dogs, and IAP from 8 to 12 mmHg was recommended for LS to avoid complications caused by hemodynamic derangements (*e.g.*, cardiovascular collapse)[22]. Such major changes in hemodynamics are rarely encountered and small changes in blood pressure, heart rate and venous return are generally observed[23]. If cardiovascular collapse occurs, insufflation must be stopped, the gas must be eliminated with the patient in the reverse Trendelenburg position. Other measures must be applied for cardiovascular stability.

Insufflation with CO_2 results in high pCO_2 and low pH. The value of pCO_2 can be increased up to 50 mmHg. pH levels below 7.30 can result in respiratory acidosis. The main reason for acidosis is the direct absorption of CO_2 from the peritoneal cavity. Elevation of the diaphragm with increased IAP and reduced tidal volume as a result of this does not seem to play an important role in pH and pCO_2 changes[15,24-27]. The acidosis and systemic effects of the pneumoperitoneum can be controlled by altering ventilation rates and/or using vasoactive agents, but such interventions have a minimal ability to preserve end-organ blood flow and oxygen delivery. Local tissue perfusion is regulated by a physiological response known as hypoxic vasodilation in which tissue oxygen requirements are directly coupled to blood flow[28].

CO_2 has direct vasodilating effects on the blood vessels. In addition, it has direct effects on the myocardium leading to decreased contraction. Plasma vasoactive peptides, epinephrine, norepinephrine and angiotensin, increase due to sympathetic stimulation as a result of increased pCO_2. Therefore, heart rate, cardiac output, diastolic and systolic blood pressures rise, blood vessel vasoconstriction occurs and renal output decreases[29-31].

Venous resistance also increases as a result of increased intra-abdominal insufflation pressure. Central

venous pressure increases from 3.3 to 10.6 cmH_2O when IAP is increased from 15 to 20 mmHg. Increased return of blood to the chest cavity *via* both IVC and abdominal organs and sympathetic stimulation in response to increasing pCO_2 results in an approximately 25% increase in cardiac output. Other gases used instead of CO_2 do not result in sympathetic stimulation and reduced cardiac output, peripheral vascular resistance and increased heart rate. This prevents continuing research for an alternative to CO_2[32-36]. Argon insufflation impairs the liver blood flow. However, helium may be advantageous compared with CO_2 insufflation[37].

Intraabdominally insufflated CO_2 during laparoscopy may have specific effects on the SC that may be unrelated to the effects of increased IAP alone. In an experimental animal study, the influences of insufflation with CO_2 *vs* air on the SC were compared and intraabdominal insufflation with CO_2 resulted in a moderate splanchnic hyperemia at an IAP $\leqslant$ 12 mmHg compared with air insufflation. At higher IAP values, pressure-induced changes became more important than the type of gas used[38].

Although a pneumoperitoneum decreases venous return, stroke volume, cardiac output and renal blood flow, it increases central venous pressure, heart rate, systemic vascular resistance, myocardial oxygen consumption and peripheral venous stasis. Thus, hypotension, hypertension, arrhythmia, myocardial ischemia, deep venous thrombosis (VT), pulmonary embolism, and renal ischemia may develop as a consequence of these effects[39]. Increased IAP after insufflation may cause respiratory distress, especially in patients with decreased lung capacity, which occurs when the pressure exceeds 15 mmHg. Controlled and assisted ventilation reduces this risk. The role of the anesthesiologist is crucial in the monitoring of respiratory pressure changes and early diagnosis of distress. Thoracic, epidural or local anesthesia is also valid for LS, but these patients are more prone to lung complications due to abdominal distension. During these processes, intra-abdominal pressure should be below 10 mmHg.

Elevation of IAP by means of gas insufflation produces hemodynamic disturbances in the peritoneal viscera, leading to splanchnic ischemia. Elevated IAP due to gas insufflation for LS may result in regional blood flow changes[40,41]. Impairments in hepatic, splanchnic and renal blood flow during peritoneal insufflation have been reported[42]. The CO_2 pneumoperitoneum appears to be a predisposing factor in the development of intestinal ischemia due to physiological alterations such as decreased cardiac output due to increased systemic vascular resistance, decreased venous return and elevated intrathoracic pressure. Moreover, a significant reduction in splanchnic blood flow results from mechanical compression of the mesenteric veins, humoral vasoconstriction of the mesenteric bed and increased portal venous pressure caused by hypercapnia, local absorption of CO_2 and increased release of vasopressin. The reduction in cardiac output and mesenteric blood flow associated with the CO_2 pneumoperitoneum seems to be insufficient to cause significant intestinal ischemia in healthy patients. Progression to critical ischemia depends on underlying vasculopathy or an inciting event.

LAPAROSCOPIC ABDOMİNAL SURGERY AND INTRA-ABDOMINAL HYPERTENSION

Splanchnic and systemic circulation are under the effect of IAP during LS and IAP should be kept as low as possible. Changes in intra-abdominal pressure, flow rate, average and maximum pressure values of the gas used are made by adjustable gas pumps during laparoscopy. The targeted mean IAP is 12-15 mmHg. An average of 3-6 L of gas can be given into the peritoneal cavity of an adult patient within these pressure limits. Reducing the inflow of CO_2 is not a tenable option because an IAP of 12-15 mmHg is generally required to produce enough space within the peritoneal cavity for organ visualization, multiple tool insertions and surgical manipulations.

With the development of laparoscopy, both anesthesiologists and surgeons have been exposed to IAH and its negative effects on the circulatory system. Kron *et al*[43] were the first general surgeons to report a study on laparoscopy-induced IAH. Cardiovascular, urinary and respiratory systems, the splanchnic area, brain, anterior abdominal wall were all affected by IAH.

Pulmonary derangements due to IAH were first established by Marey and Burt. Wendt denoted a decrease in the amount of urine with IAH and Quirin stated that an IAP of 20 mmHg may lead to a reduction in the amount of urine. Thorington and Schmidt[44] demonstrated a decrease in the amount of urine in patients with severe ascites and with IAP values of 15-20 mmHg or more. They also demonstrated that the cut-off pressure for urine was 30 mmHg. Some researchers demonstrated a 27% decrease in glomerular filtration rate (GFR) in healthy volunteers with an IAP of 20 mmHg. Experimental animal studies demonstrated a reduced amount of urine due to flow restriction in the renal veins. Guyton and Adkins[45] demonstrated a collapse in the IVC with IAP values above 20 mmHg in animal studies in 1954. Moreover, Thorington and Schmidt[44], Cooper and Scalea[46] and Burch *et al*[47] reported local clogging in the IVC due to massive ascites with IAP values of 15-20 mmHg.

IAP has been defined as a static pressure between organs in the abdominal cavity. A continuous or recurrent increase in the IAP above 12 mmHg (1.6 kPa) is regarded as IAH. Abdominal compartment syndrome (ACS) has been defined as a continuous IAP above 20 mmHg (2.67 kPa) with coexisting organ dysfunction or failure. Mortality of patients with recognized ACS may be as high as 42%. The diagnosis of IAH is based on the measurement of IAP only. The World Society

of the ACS (WSACS) advises screening of IAP in all patients admitted to intensive care units with certain risk factors[48]. The measurement of IAP should be made in the end-expiratory phase, in the flat supine position, after relaxation of abdominal muscles and the median axillary line should be referred to as zero-level. The IAP should be measured in the urinary bladder filled with 25 mL of sterile solution of 0.9% NaCl, according to the WSACS guidelines. In confirmed cases of abdominal hypertension and/or ACS, immediate action should be taken, which consists of the evacuation of gastric and bowel contents, maintenance of adequate blood pressure, diuretics and/or ultrafiltration and deeper sedation and/or muscle relaxation[49].

ACS is the result of the deleterious effects of IAH on the pulmonary, cardiovascular, splanchnic, musculoskeletal/integumentary (abdominal wall), urinary and central nervous systems. Abnormal and sudden increase in the volume of any component of the intraperitoneal or retroperitoneal spaces (occurring postoperatively or subsequent to hemorrhagic trauma, pneumoperitoneum, reperfusion edema, intestinal distention, acute pancreatitis) results in IAH. Both animal and human studies have demonstrated that in 20%-80% of cases with IAH, IAP can be transferred to the thoracic cavity. Increased thoracic pressure decreases blood inflow to the heart and decreases the end-diastolic volume of ventricles, resulting in lower contractility of the myocardium. All this leads to a decrease in cardiac output. Increased IAP transferred to the thoracic cavity can interfere with the measurements of central venous pressure and pulmonary capillary wedge pressure.

Sustained abdominal hypertension leading to ACS is always fatal when unrecognized or untreated. Measurement of urinary bladder pressure is the best validated technique for the diagnosis of IAH. It should be used routinely for the minimally invasive follow-up of IAP in patients with severe thoraco-abdominal trauma or after major abdominal operations (laparoscopic or open surgery). Identification of patients at risk, early recognition and appropriate and timed intervention are the keys to effective management of this condition. Treatment consists of adequate fluid resuscitation and surgical decompression when necessary[50,51].

In 2006, the WSACS published definitions regarding IAH and ACS[48-51]. IAH is graded as follows: Grade-Ⅰ: 12-15 mmHg (1.6-2.0 kPa); Grade-Ⅱ: 16-20 mmHg (2.1-2.6 kPa); Grade-Ⅲ: 21-25 mmHg (2.8-3.3 kPa); Grade-Ⅳ: > 25 mmHg (> 3.3 kPa).

Elevated IAP has been shown to decrease mesenteric blood flow and cause intestinal damage[52]. Development of IAH during LS is disturbing. Pressure values above 20 mmHg may result in reduced perfusion of the intestinal mucosa and submucosa. Even low levels of IAP, such as 15 mmHg, can result in intestinal ischemia after a period of time due to the systemic inflammatory response activated by cytokines released from the intestinal wall. In addition, experimental studies have shown that multiple factors may cause bacterial translocation[52-54]. The formation of bacterial translocation leads to multiple organ failure and sepsis, contributing to mortality[1]. Patient selection, maintaining IAP at 15 mmHg or below and intermittent decompression of the gas are the best options for preventing this complication.

Adverse effects of IAP on hepatic arterial, portal and microcirculatory blood flow have been shown with pressures above 20 mmHg. A progressive decline in perfusion through these vessels occurs as IAP increases, despite cardiac output and systemic blood pressure being maintained at normal levels. Splanchnic vascular resistance is a major determinant in the regulation of hepatic arterial and portal venous blood flow. Elevated IAP can become the main factor in establishing mesenteric vascular resistance and finally abdominal organ perfusion. These abnormalities are amplified in the presence of hypovolemia and hemorrhage, and can only partly be corrected by physiologic and resuscitative improvements in cardiac output[55].

The increased intraperitoneal pressure necessary to perform laparoscopic operations substantially decreases portal venous blood flow. The extent of the volume flow reduction is related to the level of intraperitoneal pressure. This reduction in flow may suppress hepatic reticular endothelial function. Thus, the reduced portal blood flow may enhance the cryoablation effect during laparoscopic cryosurgery for metastatic liver disease by decreasing the heat sink effect[56].

According to a study in experimental animals, pneumoperitoneum with CO_2 during standing laparoscopy in healthy horses does not cause adverse alterations in cardiopulmonary or laboratory variables, but causes a mild inflammatory response within the peritoneal cavity. High pressure (15 mmHg) pneumoperitoneum in standing sedated mature horses undergoing LS can be performed safely without any short-term or cumulative adverse effects on hemodynamic or cardiopulmonary functions[57].

Severe progressive reduction in mesenteric blood flow has been shown with graded elevation in IAP from approximately 70% of baseline at 20 mmHg to 30% at 40 mmHg[55]. Intestinal mucosal perfusion, measured by a laser flow probe, has been shown to be impaired at IAP above 10 mmHg. Metabolic changes resulting from impaired intestinal mucosal perfusion have been shown by tonometry measurements and revealed worsening acidosis in the mucosal cells with increasing IAH. Similarly, abnormalities in intestinal oxygenation have been shown with elevations in IAP above 15 mmHg. Impairment of bowel tissue oxygenation occurs without corresponding reductions in subcutaneous tissue oxygenation. This phenomenon indicates the selective effect of IAP on organ perfusion. Not surprisingly, reductions in mesenteric blood flow have been shown to be greatly exacerbated during resuscitation after hemorrhagic shock[55].

A direct relationship between IAP and intra-cranial

pressure (ICP) has been demonstrated. An increase in ICP results from increased intra-thoracic pressure due to the transmission of pressure from the abdominal cavity. Due to the reasons mentioned, the measurement of IAP is recommended in all patients with pathology likely to increase ICP, particularly in patients with risk factors for IAH. Increased ICP has also been observed during diagnostic laparoscopy, therefore, this procedure is not recommended for patients with cranial trauma[52].

COMPLICATIONS RELATED TO THE SC DURING LAPAROSCOPIC ABDOMİNAL SURGERY

Veress needle-like tools used for the creation of a pneumoperitoneum can result in direct mechanical injury to splanchnic vessels. Moreover, intra-abdominal laparoscopic instruments may cause direct mechanical compression injury and affect the SC. Care must be taken especially with the first trocar, which is blindly inserted into the abdomen. Incorrect and careless practices can lead to colon, IVC and aortic vascular perforations, splanchnic vascular injury, mesenteric injury and even death[58,59]. Abdominal wall hematoma may also develop during trocar insertion.

Some surgeons prefer open insertion of the first trocar to the closed method. If the first trocar is placed *via* the closed method the index finger must be used as a guide to avoid injury. The camera must be the first instrument inserted into the abdominal cavity. The first thing to look for after introduction of the trocar is whether there is any bleeding or organ injury. The remaining trocars must be placed under the guidance of the video camera inserted after the first trocar, to avoid injury to splanchnic vessels, and the anatomical structure of the vessels in the anterior abdominal wall can be seen due to the camera light. Nasogastric drainage and bladder catheterization are recommended prior to establishment of the pneumoperitoneum.

Complications due to sustained elevated intraperitoneal pressure in cardiovascular, pulmonary and renal systems have been well documented in several experimental and clinical studies. Alterations in the SC have also been reported in animal experiments, but details of the exact hemodynamic changes in the flow to solid intraabdominal organs caused by raised intraperitoneal pressure in humans are not available.

Several factors should be considered in the evaluation of hepatic perfusion. During LS, hepatic blood flow is reduced as a result of increased sympathetic activity (caused by hypercarbia), as well as increased IAP. Splanchnic vascular resistance may increase with certain modes of mechanical ventilation used during anesthesia. Moreover, anesthetics may decrease cardiac output and proportionally reduce the total hepatic blood flow that is already deranged by the cardiovascular effects of positive-pressure pneumoperitoneum.

After insufflation, cardiac output and blood flow in the SMA and PV decrease progressively and return to the pre-insufflation values following deflation. Hepatic arterial blood flow does not change significantly, perhaps due to compensatory mechanisms for maintenance of hepatic blood flow. Mechanical compression of the splanchnic capillary beds due to elevated IAP may possibly reflect the increase in systemic vascular resistance causing decreased cardiac output. To prevent this effect, intermittent decompression of gas during surgical laparoscopy is recommended[60,61]. A study involving 31 patients undergoing elective laparoscopic cholecystectomy which measured the changes in hepatic venous and left portal venous blood flow *via* transesophageal echocardiography showed decreased hepatic blood flow due to increased intraperitoneal pressure[62].

An experimental animal study demonstrated a significant reduction in cardiac output and mean arterial pressure during CO_2 laparoscopy[63]. Cardiovascular hemodynamics normalized to baseline values after rapid desufflation. Blood flow in the hollow viscus organs was less disturbed than that in the solid organs during laparoscopy. Although small and large bowel blood flow was reduced significantly, gastric blood flow remained unchanged. Total hepatic blood flow was influenced predominantly by portal blood flow, which was particularly decreased; hepatic arterial flow remained stable. Severe alterations in cardiovascular hemodynamics and in the hepatic and SC rapidly occurred during CO_2 laparoscopy. It can be presumed that both increased IAP and hypercapnia which are the main factors underlying these disturbances[63]. Another experimental study in animals suggested that there is a compensatory increase in hepatic arterial flow, known as the "hepatic arterial buffer response" that may maintain liver blood supply during laparoscopy-associated portal venous flow reduction[64].

Splanchnic macrocirculatory changes during high-pressure CO_2 pneumoperitoneum include decreased mesenteric arterial blood flow and decreased gastric perfusion with a drop in gastric pH. Microcirculatory changes in abdominal organs under a low pressure CO_2 pneumoperitoneum are unknown. It is suggested that laparoscopic procedures with CO_2 pneumoperitoneum should be performed at a pressure of 10 mmHg or below to avoid splanchnic microcirculatory disturbances[65].

The abdominal wall itself is also adversely impacted by elevations in IAP. Significant abnormalities in the rectus abdominis muscle blood flow have been documented with progressive elevations in IAP. These perfusion abnormalities are roughly on a par with the changes in the abdominal visceral perfusion with graded increases in IAP. Clinically, this condition results in derangement of abdominal wound healing, including fascial dehiscence and surgical site infection[55].

Renal blood flow, tubular functions and glomerular filtration rate are affected by numerous multifactorial

and complex mechanisms resulting in decreased urine output. Cardiovascular effects of elevated positive-pressure pneumoperitoneum include impairment of venous return, reduced cardiac preload, decreased cardiac output and eventually reduced renal blood flow. Another putative mechanism for decreased urine output is mechanical compression of IVC, renal vasculature and the parenchyma.

Positive-pressure pneumoperitoneum causes increased sympathetic activity that can lead to renal cortical vasoconstriction[66]. Central and peripheral regulatory mechanisms increase secretion of antidiuretic hormone, which acts on the collecting ducts and the thick loop of Henle. Reduced renal perfusion together with sympathetic stimulation activates the renin-angiotensin-aldosterone system, inducing its vascular and metabolic effects. Increased levels of catecholamines and endothelin-1 have also been detected. All of these factors are in addition to the major effect of surgical stress on renal function. Anesthetic drugs usually decrease glomerular filtration and renal blood flow secondary to their endocrine, sympathetic and cardiovascular effects. Other drug interactions and preexisting renal disease contribute to this complex situation. Indeed, positive-pressure ventilation can induce derangement of kidney function through cardiovascular and neurohumoral mechanisms[66]. Both hepatic blood flow and renal blood flow decrease as IAP increases. Therefore, in order to carry out laparoscopic abdominal surgery safely in patients with hepatic or renal impairment, low IAP or non-insufflating techniques are recommended[32].

PHT leads to considerable hemodynamic changes in the SC. In experimental models, chronic PHT was shown to result in splanchnic vasodilatation and hyperdynamic circulation[67]. The exact pathogenesis of hyperdynamic circulation is not well understood. However, an increased quantity of vascular endothelial vasodilators and reduced response to endogenous vasoconstrictors are proposed. The powerful vasodilator properties of nitric oxide on the microcirculation have been known for a long time. During hemodynamic regulation of the microcirculation in PHT the synthesis of nitric oxide is stopped[68-70]. Local vasodilators such as endothelium-derived nitric oxide and prostaglandins cause vasodilatation in response to microcirculatory changes resulting in clinically significant changes[71]. Therefore, LS is not appropriate for patients with splanchnic circulatory disorders and those with clinically significant PHT.

One of the most important pathologies in the splanchnic venous system is the development VT. IAH is one of the risk factors for VT as it can result in vein collapse. Three conditions, first described by Rudolf Virchow in 1862, contribute to VT formation. These are stasis of blood flow, endothelial damage and hypercoagulability. Acquired risk factors for VT include advanced age, hospitalization and immobilization, hormone replacement and oral contraceptive therapy, pregnancy and postpartum state, prior VT, malignancy, major surgery, intra-abdominal hypertension, obesity, nephrotic syndrome, trauma and spinal cord injury, long-haul travel (> 6 h), varicose veins, antiphospholipid syndrome, myeloproliferative disorders and polycythemia. Heritable risk factors include factor V Leiden; prothrombin 20210A gene variant; antithrombin, protein C and protein S deficiencies; and dysfibrinogenemias. Thrombophilia may have both heritable and acquired components in some patients. These mixed causes include homocysteinemia; factor Ⅶ, Ⅷ, Ⅸ and Ⅹ Ⅰ elevations; hyperfibrinogenemia; and activated protein C resistance in the absence of factor V Leiden[72]. Preoperative risk assessment for the development of VT should be performed in patients scheduled for LS.

In a series of 21 cases with acute mesenteric ischemia, intestine viability was 100%, 56% and 18% in patients diagnosed within the first 12 h, 12-24 h and after 24 h of symptom initiation, respectively[73]. The first 6 h are particularly important for the diagnosis of ischemia, and restoration of blood flow improves the prognosis[6,74,75].

SC DURING LAPAROSCOPIC ABDOMİNAL SURGERY IN THE ELDERLY

Even with small gas pressures, peritoneal gas insufflation induces many splanchnic hemodynamic changes related to both the direct mechanical compression of intraabdominal vessels, and to the systemic hemodynamic and acid-base effects derived from increased IAP and from carbon dioxide absorption. Laparoscopy is increasingly performed in older and often more unfit patients, in whom the procedure may last considerably longer. LS in the elderly, especially in emergency cases, results in higher morbidity and mortality due to comorbid diseases[76]. Risk factors for ischemic events in patients over the age of 50 are heart valve diseases, cardiac arrhythmias, history of recent myocardial infarction, congestive heart failure, hypotension and hypovolemia[77]. Chronic mesenteric ischemia in asymptomatic patients can result in ischemic events following LS. Detailed knowledge of risk factors and preoperative evaluation of patients are important. Only 5% of cases with mesenteric ischemia are due to chronic mesenteric ischemia and most are caused by atherosclerotic diseases. Males and females are equally affected with a mean age of 68 years[78].

Patients, in whom the SC is thought to be impaired, based on preexisting atherosclerosis or other risk factors, should be handled with great care or offered an open laparotomy rather than the laparoscopic approach. A high index of suspicion and early evaluation by ultrasound followed by laparotomy may be helpful in improving survival rates. It is generally agreed that periodic decompression of the pneumoperitoneum, while maintaining the IAP at 15 mmHg or below, represents the best available option for preventing this complication.

In the elderly, increasing dysfunction is observed at the cellular level and organ systems due to the aging

process. Diseases which require surgical intervention are less tolerated in this population. Diseases of the gastrointestinal system are generally the result of the aging process itself. In the elderly, gastrointestinal surgery can be performed safely with good control of comorbid diseases.

SC CHANGES RELATED TO OBESITY AND PATIENT POSITION DURING LAPAROSCOPIC ABDOMİNAL SURGERY

The SC can be affected depending on the position of the patient during LS. The Trendelenburg position is used for some surgical procedures including gynecological interventions, appendectomy and colorectal surgery when performed by the laparoscopic method. The effects of the IAP are exacerbated due to further pushing of the diaphragm by gravity. Intrathoracic pressure rises, residual volume decreases, and the probability of atelectasis and hypoxemia increases. In addition, the increase in splanchnic venous return and cardiac output alleviates the adverse cardiovascular effects to some degree. Cerebral perfusion may be corrupted due to venous congestion in the head and neck region. Intracranial pressure and intraocular pressure also increase.

The lithotomy position and subsequent pneumoperitoneum increase preload, probably due to blood shifting from the abdomen to the thorax by compression of splanchnic vessels. Careful fluid management, maintaining low abdominal pressure and use of the reverse Trendelenburg position are favored to prevent adverse hemodynamic effects during LS[79]. The head-up position and intraperitoneal pressure greater than 12 mmHg should be avoided during LS because they compromise hepatic and renal blood flow. LS causes a reduction in hepatic blood flow due to a number of factors, including increased IAP, neurohumoral response to surgical stress and the effect of patient position[80]. The clinical significance of this phenomenon is not fully understood.

In obese patients with large amounts of omental and intestinal fat, more extreme positions may be required for a good surgical view. Therefore, more changes occur in the splanchnic vascular area. Effects related to positional changes in patients with hypovolemia and hypotension are exacerbated[81,82]. In obese patients, IAP may reach 13.2 or even 13.7 mmHg (1.7-1.8 kPa). The clinical implications of IAP abnormalities in morbidly obese patients have not yet been studied[49].

CONCLUSION

Laparoscopic abdominal surgery has rapidly become a popular and widely used technique. Recent advances in operative procedures and surgical instrumentation have enabled the application of laparoscopic techniques in more complex surgeries. However, complex procedures require extended insufflation times and consequently patients experience prolonged periods of elevated IAP. LS with computer-assisted surgery such as robotic surgery have also become more popular. Both the number of operations and the variety of operations have increased. Therefore, a longer operative time has become routine for complicated laparoscopic surgical procedures. Despite its benefits, pneumoperitoneum is not without physiological consequences. In humans, even brief periods of CO_2 insufflation (45-60 min) have been shown to significantly reduce blood flow to organs within the peritoneal space. This reduction promotes anaerobic metabolism leading to lactic acidosis with postoperative manifestations including alteration in liver enzymes, subclinical hepatic dysfunction and the appearance of oxidative stress markers. Tissue ischemia and altered postoperative organ function are major causes of laparoscopy-associated morbidity and mortality. A method to maintain organ blood flow during LS has not yet been developed. For a safe and effective approach to LS, anatomy of the abdomen, pathophysiology of the SC and surgical outcomes should be known. The most important points in LS are as follows: (1) for the patient, an unhurried and calm working style is necessary for safe laparoscopic abdominal surgery; (2) the lowest possible IAP range should be achieved for the safe application of pneumoperitoneum; (3) a minimal number and effective use of laparoscopic instruments and materials; (4) Good preoperative clinical evaluation and determination of possible risk factors; (5) optimal perioperative monitoring of vital signs and urine output, and good collaboration with anesthetists; (6) minimize the LS time as much as possible; and (7) close monitoring of the patient in the postoperative period regarding complications of SC derangement.

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