Cardiovascular Collapse during Laparoscopy: A Brief Overview

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Abstract

This review article considers the physiology, differential diagnosis and immediate management of vasovagal response, vascular injury and carbon dioxide embolism caused during creation of the laparoscopic pneumoperitonium. These pathologies account for over half of all laparoscopic complications and therefore, by taking a systematic approach to these possibly life-threatening events, laparoscopy can become even safer.

Introduction

The introduction of laparoscopy has revolutionized modern surgery. As more procedures are performed with laparoscopic techniques, the use of laparotomy and other open techniques have declined. Patients undergoing these minimally invasive procedures are having shorter lengths of hospital stay, less pain, quicker recovery and, in many instances, the overall cost of the procedure is less[1-3]. However, the introduction of laparoscopy has brought unique complications. This review article will discuss the rare yet possibly fatal risk of cardiovascular collapse during laparoscopy.

The first laparoscope was performed in the early 20$^{\text{th}}$century. Quickly pneumoperitoneum protocols were established to improve intraabdominal visualization. By 1968, the Verres needle was invented and carbon dioxide was used routinely as an insufflation gas. Carbon dioxide has become a mainstay of laparoscopy because it is inexpensive, readily available, non-flammable, colorless and highly soluble in blood. Early laparoscopic techniques were developed in the gynecological realm specifically to cause permanent sterilization. Early laparoscopic gynecologists would insert the Verres needle into the posterior cul-de-sac or even into the cervix and through the uterine fundus to begin insufflation[4,5]. These techniques encountered some complications and therefore placing the insufflating device through the abdominal wall has become more commonplace.

The combination of blind entry into the abdomen and the exposure of the patient to carbon dioxide gas has created some unique opportunities for cardiovascular collapse to occur during laparoscopic procedures. Estimates of laparoscopic mortality range from 1-4/100,000 cases[3,5,6]with 50% of all major laparoscopic complications occurring at abdominal entry and insufflation[1]. There is a 1/2,500 incidence of cardiac arrest during laparoscopy[6] with a 14% rate of dysrhythmias[3]. This review article will briefly review the normal physiological alterations caused by a carbon dioxide pneumoperitoneum and then discuss the leading causes of cardiovascular collapse related to the formation of that pneumoperitoneum. We will then touch upon the differential diagnosis of these causes and immediate management of the patient experiencing cardiovascular collapse.

Physiology

Carbon dioxide is found in the blood of all living humans as it is a major by-product of metabolism. With a normally functioning cardiopulmonary system, that carbon dioxide is carried out of the body and eliminated by lung exhalation. If this carbon dioxide is not eliminated, it can cause respiratory acidemia with a myriad

of complications to the patient including cardia arrhythmias. The creation of a pneumoperitoneum increases the amount of carbon dioxide in the body. Carbon dioxide can be absorbed when coming into contact with the peritoneum or can be inadvertently introduced into an injured blood vessel. Carbon dioxide gas can also unintentionally be introduced into the subcutaneous tissue causing subcutaneous emphysema. It is thought this occurs in up to 2% of laparoscopies leaving another reservoir of carbon dioxide that must be ventilated off[7]. One study found that peak serum carbon dioxide levels occur 10 minutes after insufflation[3,8]and persist for 45 minutes after the pneumoperitoneum is released[3]. Most patients, especially when ventilated, can easily clear this excess carbon dioxide. Anesthesiologists often adjust minute ventilation to account for this increase in carbon dioxide uptake, allowing a normal acid-base status to be maintained during surgery.

The act of creating the pneumoperitoneum does create some boundaries to clearing that carbon dioxide. The pneumoperitoneum places upwards pressure on the abdominal wall to increase visualization but also increases pressure on the diaphragm and inferior vena cava. Pressure on the diaphragm decreases lung volume and therefore gas exchange making it harder for carbon dioxide to be eliminated. Trendelenburg position is frequently used in laparoscopy and it also causes pressure on the diaphragm by shifting the abdominal consents against the diaphragm. All of these changes serve to worsen lung compliance, lowering tidal volumes and increasing the risk of barotrauma with mechanical ventilation. While well-tolerated in young and healthy patients, this ventilator impairment can pose a challenge for older or morbidly-obese patients. Pressure on the inferior vena cava will also decrease blood return to the heart and hence cardiac output. Therefore, less carbon dioxide makes it to the lungs to be eliminated and heart perfusion is decreased. Decrease in myocardial oxygen delivery also leads to arrhythmia. One study found that when the pneumoperitoneum exceeds 15 mmHg cardiac output can be decreased by 28%[4]. Again, these physiological changes can be compensated for in a healthy ventilated patient. Obese patients and patients with pre-existing heart and lung disease will need closer monitoring to compensate for their pathophysiology[7]. The speed at which the pneumoperitoneum is created also has effects on the sympathetic nervous system. The rapid stretching of the peritoneum with insufflation greatly increases vagal tone[8]. This rush of sympathetic response leads to hypotension and bradycardia even to the point of asystole.

Leading causes

The above-mentioned vagal response is a cause of cardiovascular collapse during laparoscopy. This pathophysiology is usually self-limiting in a healthy patient. By releasing the pneumoperitoneum and supporting the patient while the sympathetic nervous system resets, normal cardiopulmonary physiology can usually be reestablished. Additionally, antimuscarinic agents (either glycopyrrolate or atropine) are often administered by the anesthesia team to combat bradycardia, interrupting the vagal signaling arc and decreasing the incidence of bradycardia. Glycopyrrolate has a longer duration of action and does not cause central anticholinergic syndrome, but is the slower-onset of the two commonly-used antimuscarinics. Occasionally, sympathomimetics (ephedrine or epinephrine) are needed to raise the heart rate if unresponsive to atropine. The other two leading causes of cardiovascular collapse can be much more malignant and require more intervention. These are accidental vascular injury and/or carbon dioxide embolism caused in relation to the formation of the pneumoperitoneum.

The rate of vascular injury is 0.03%-0.06%[3]. An Australian study reported a rate of 7/10,000 for vascular injury with a 23-56% mortality rate resulting[9]. The most common injured major blood vessels are the abdominal aorta followed by the common iliac vessels however mesenteric and all other intraabdominal vessels are at risk. The injury of these large vessels leads to exsanguination and resulting hemorrhagic sock. This condition is diagnosed first by a flash of blood through the insufflating device followed by tachycardia and hypotension. These are the hallmarks of acute blood loss. Cardiovascular collapse ensues when intravascular volume and pressure are lost and the heart, as well as other organs, are no longer perfused.

The incidence of carbon dioxide embolism during laparoscopy is much more elusive. Trans-esophageal echocardiogram during routine laparoscopy has shown gas bubbles present in the right heart in 6.25 to 100% of all cases[10,11]. Granted, the majority of these emboli are asymptomatic. It is estimated that clinically relevant emboli occur in 0.001% of laparoscopies of which 28% are fatal[10]. Dog and pig studies have

demonstrated that it would take greater than 70 cc/minute of direct injection of intravascular carbon dioxide to cause cardiovascular collapse[4,11]. Normal laparoscopic low flow rates should only introduce 5% of that volume[10]. Carbon dioxide embolism causes gas bubbles to be trapped and occlude the pulmonary vasculature. This results in a V-Q mismatch which causes hypoxia and hypercapnia. This loss of oxygen and respiratory acidemia results in decreased oxygenation and acidosis of the heart and other organs and organ failure. Carbon dioxide embolism is diagnosed by cyanosis, bradycardia and hypotension. End tidal carbon dioxide, measured during ventilation, rapidly increases then drops off dramatically. The pathognomonic mill-wheel murmur is heard as trapped gas bubbles are heard in the right heart. Precordial Doppler has been used in the past to also recognize this trapped gas. While rare, carbon dioxide may also cross a patent foramen ovale and cause arterial occlusion. This is considerably less dangerous than arterial air given the solubility of carbon dioxide in blood, but nonetheless is a potential cause of embolic stroke.

Immediate management

The key to managing this possibly life-threatening complications lies in making the correct diagnosis. Vascular injury, after identified, should start with leaving the insufflation device in place. The Veress needle, or other insufflating device, can serve to occlude the damaged vessel and removing the instrument may allow blood to rapidly leave the vasculature and even make the rent in the vessel larger. An exploratory laparotomy can then be performed where the damaged vessel is identified, the insufflation instrument removed under direct visualization and the vessel repaired. It would be advantageous at this point to recruit the assistance of vascular surgery or other specialists well practiced in large vessel repair. This event should be immediately communicated to the anesthesia team who can begin volume resuscitation, replacement of blood products and even possible pressor support to maintain perfusion and avoid further hemorrhagic shock. Consideration should also be made for aortic cross-clamping or using an aortic occlusion balloon, both of which are routinely used in aortic surgery by vascular surgeons for hemorrhage control. Care should be taken to place the clamp as distal as possible, to avoid renal and gastrointestinal tract ischemia, and to avoid clamping the inferior vena cava.

The management of carbon dioxide embolism is significantly different and therefore the need to make the correct diagnosis. The mainstay of carbon dioxide embolism treatment is to stop insufflating gas into the patient. The gas should be turned off and the pneumoperitoneum released. All of these actions will decrease more carbon dioxide from entering the blood stream. Again, this should be communicated to the anesthesia team who can also help with the diagnosis (end tidal carbon dioxide readings and mill-wheel murmur). The anesthesiologist can then hyperventilate the patient to remove the carbon dioxide and position the patient on their left side to trap carbon dioxide bubbles in the upper right atrium and away from the pulmonary vasculature. In extreme cases, a central venous line can be placed and gas bubbles removed from the right heart, though typically by the time the line is placed the carbon dioxide has already been absorbed: this is most helpful in patients with indwelling central venous catheters. Hyperbaric therapy has been used to treat cerebral gas emboli which arrived in the brain by a patent foramen ovale (paradoxical emboli), an uncommon sequela of this uncommon condition.

Future Questions

This review article has covered why the pneumoperitoneum, unique to laparoscopy, can cause life threatening complications. Pneumoperitoneum physiology, common reasons for cardiovascular collapse on insufflation and their respective management have been covered. However, there are a few questions that remain to be addressed in relation to this topic. 1) If a vagal response or small carbon dioxide embolism is successfully managed if and when should re-insufflation be attempted. How much slower a rate of insufflation and pressure should be used? 2) What is the safest entry into the abdominal cavity to allow insufflation. A Cochrane review has tried to address this issue and even a recent update to that review has still shown inconclusive results[12].

Medicolegal and Ethical Considerations

These further research questions also bring up some ethical and legal areas of interest. The above mentioned

pathologies are rare and frequently self-limited. Quality care is transparent care and therefore these possibly fatal complications should be presented to our patients during the consent process. We should also be clear to our patients that there are limited ways to mitigate these risks and that entry techniques vary greatly between surgeons[13]. If these events occur, they should be communicated to the patient in the post-operative area. These events, and their management, should also be documented in the operative report. This communication will be invaluable for the next laparoscopist and anesthesiologist.

Sandberg et al found that more than a third of Dutch laparoscopic malpractice claims were entry related. The number of claims has stayed relatively constant over the last twenty years[14]. However, the United Kingdom case of Palmer v Cardiff and Vale NHS has established precedent, at least in England, that injury during laparoscopic entry is considered negligent[13]. If litigation were to be pursued, then a clear and thorough documentation of events and the thought process behind management will assist in making intent very clear to other parties.

Intraoperative decisions frequently need to be made without the assistance of our patients. If a vagal response or carbon dioxide emboli is managed successfully, the decision to reattempt insufflation is made solely by the patient's surgeon and anesthesiologist. Shared decision making becomes near impossible when one half of the parties involved are under general anesthesia. There appears to be no way around this but it is a humbling thought none-the-less and again speaks to a thorough consent process.

Finally, should laparoscopy be performed in a setting where an anesthesiologist and/or surgeon trained in large vessel repair are not readily available? Safety is paramount but limiting access to the benefits of minimally invasive procedures seems to be an undue burden for such rare events. The benefits of minimally invasive laparoscopic procedures are great and with further research and this review article hopefully the risks can be further mitigated.

Conflict of Interest Statement

The authors have no conflict of interest to declare

Author Contribution Statement

BW, DB and MAB all contributed to this review conception and design, interpreting the relevant literature and manuscript preparation and revision. BW, DB and MAB all have reviewed and approve the final manuscript.

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