Anaesthesia and minimally invasive surgery

Michael G Irwin

Stanley SC Wong

Abstract

Minimally invasive surgery is commonly performed because of various advantages such as reduced postoperative pain, faster recovery, and reduced postoperative pulmonary complications. However, anaesthesia for laparoscopy can be difficult and potentially hazardous in long, complex surgical procedures and in sick patients. Establishment of CO_2 pneumoperitoneum produces adverse pathophysiological changes due to increased intra-abdominal pressure and hypercapnia, and these are further altered by postural changes. Laparoscopy is also associated with potential complications such as extraperitoneal gas insufflation and pneumothorax. It is important for the anaesthetist to understand the advantages and potential risks. General anaesthesia with endotracheal intubation is the most common anaesthetic technique, but supraglottic airway devices can sometimes be used. Neuroaxial anaesthesia has been used in some laparoscopic procedures as the sole anaesthetic technique. This article will focus on the pathophysiological changes caused by CO_2 pneumoperitoneum, the anaesthetic management for patients undergoing laparoscopy, and potential complications.

Keywords Anaesthesia; complications; laparoscopy; laryngeal mask airway; minimally invasive surgery; pathophysiological changes; pneumoperitoeum; regional; supraglottic airway device; Trendelenburg

Learning objectives

After reading this article you should be able to:

- understand the pathophysiological changes of CO_2 pneumoperitoneum on the cardiovascular and respiratory system
- give four reasons why CO_2 is the most commonly used gas for pneumoperitoneum
- list four potential complications of laparoscopic surgery

Introduction

Minimally invasive surgery involves the use of precise instruments designed to decrease the size of incisions as well as spare surrounding tissue. They are usually performed with endoscopic visualization. Laparoscopy is the commonest technique and used for diagnostic and staging procedures, emergency operations such as appendicectomy and cholecystectomy, as well as elective surgeries including oesophagectomy, hemicolectomy, liver resection, gastrectomy, nephrectomy, radical prostatectomy, cystectomy, and distal pancreatectomy.[1,2] This review will focus primarily on how to manage patients undergoing laparoscopy.

Laparoscopy requires inflating gas into the peritoneum to provide a surgical view. Usually 2.5–5.0 litres of carbon dioxide (CO_2) is inflated.[1] An intra-abdominal pressure (IAP) of up to 15 mmHg is usually used for abdominal surgeries. CO_2 is used because it does not support combustion, is cheap, is cleared more rapidly than other gases, and is highly soluble in blood which reduces the risk of gas embolism. The disadvantage is that vascular absorption of CO_2 can cause hypercapnia and respiratory acidosis. Pneumoperitoneum itself produces mechanical effects also, and it is important for the anaesthetist to understand these changes in order to provide optimal care.

Laparoscopic surgery offers several advantages over open surgery. There is a reduction in stress response, postoperative pain, intraoperative bleeding, rate of postoperative wound infection, impairment of respiratory function, and pulmonary complications. Recovery time is shorter and cosmetic appearance is improved.[1,2] These advantages need to be balanced with potential adverse effects caused by CO_2 pneumoperitoneum.

Laparoscopic surgery is usually performed under general anaesthesia. Endotracheal intubation is common but supraglottic airway devices such as laryngeal mask airways (LMA), particularly those with the facility for gastric drainage, are becoming more popular.[1,3] Neuroaxial anaesthesia has also been used.[1,4,5]

Physiological effects of pneumoperitoneum

Pneumoperitoneum with CO_2 insufflation produces adverse pathophysiological effects via increase in IAP and hypercapnia. While well tolerated in healthy individuals, these can be harmful in patients with cardiopulmonary disease.

Respiratory effects (Table 1)

During pneumoperitoneum, delivery of CO_2 to the lungs may increase by up to 50%.[6] Arterial ($PaCO_2$), alveolar ($PACO_2$), and mixed venous PCO_2 levels usually rise by 10 mmHg 5 minutes after insufflation.[7] This can cause hypercapnia and respiratory acidosis.

Increased IAP shifts the diaphragm cephalad and reduces diaphragmatic excursion. This reduces functional residual capacity by up to 20%[7] and causes atelectasis. Oxygenation is minimally affected with no significant change in alveolar arterial oxygen gradient.[7,8] At an IAP of 15 mmHg, pulmonary compliance decreases by up to 47% and peak airway pressures can increase by 50%.[7] Work of breathing is increased and controlled ventilation is prudent in most cases.

Postoperative lung function tests show significant reduction in forced expiratory volume in 1 second (FEV_1), peak expiratory flow (PEF), and forced vital capacity (FVC) in both open and laparoscopic surgery.[6] However, atelectasis and impairment of

Michael G Irwin MB ChB MD FRCA FHKAM is Professor and Head, Department of Anaesthesiology, University of Hong Kong, and Honorary Consultant in Anaesthesia and Administrative Director of the Intensive Care Unit at Queen Mary Hospital, Hong Kong, China. Conflicts of interest: none declared.

Stanley SC Wong MBBS is a Resident in Anaesthesia at Queen Mary Hospital, Hong Kong, China. Conflicts of interest: none declared.

Effect of CO_2 pneumoperitoneum on respiratory function

Respiratory factor	Effect
Functional residual capacity	Decrease
Atelectasis	Increase
Peak airway pressure	Increase
Lung compliance	Decrease
Work of breathing	Increase
Alveolar arterial oxygen gradient	Unchanged
$PaCO_2$	Increase
Forced expiratory volume in 1 second (FEV_1)	Decrease
Peak expiratory flow (PEF)	Decrease
Forced vital capacity	Decrease

Table 1

lung function test parameters are significantly less in patients undergoing laparoscopy.

Laparoscopy produces adverse intraoperative respiratory effects, but results in less postoperative respiratory impairment.

Cardiovascular effects (Table 2)

CO_2 pneumoperitoneum produces significant cardiovascular effects due mostly to increased IAP, but also hypercapnia.

Effects due to increased intra-abdominal pressure: IAP, patient position and intravascular volume all affect haemodynamic function during pneumoperitoneum. At an IAP of 15 mmHg, cardiac output and stroke volume have been shown to decrease by 30%[6] due to reduced venous return and increased afterload. Reduction in cardiac output is greater with higher IAP. When comparing IAP of 7 mmHg versus 15 mmHg during laparoscopic cholecystectomy, stroke volume and cardiac output were significantly more depressed at higher IAP.[9] Reduction in cardiac output in the obese appears to be less, probably because they usually have a chronically high IAP of 9–10 mmHg.[8] Cardiac depression is usually transient with cardiac index returning to baseline levels within 10–15 minutes after abdominal insufflation.[10]

At an IAP of around 15 mmHg, there is an increase in systemic vascular resistance (SVR) and afterload due to compression of the abdominal aorta and increased catecholamine, vasopressin and renin angiotension activity.[2,7] Raised SVR increases mean arterial pressure. Heart rate is also elevated and increased afterload can increase ventricular wall tension. Risk of myocardial ischaemia may therefore be increased.[6]

Effect of CO_2 pneumoperitoneum on cardiovascular function

Cardiac factor	Effect
Heart rate	Increase
Stroke volume	Decrease
Cardiac output	Decrease
Systemic vascular resistance	Increase
Mean arterial blood pressure	Increase
Central venous pressure	Increase

Table 2

Intense vagal stimulation due to insertion of Veress needle/trocar, peritoneal stretch, carbon dioxide embolization, or stimulation of Fallopian tubes during bipolar electrocauterization can cause sinus bradycardia, nodal bradycardia, atrioventricular dissociation and even asystole.[1] It is unusual and can be treated with atropine and withdrawal of the stimulus.

Effects due to hypercapnia: a $PaCO_2$ of 45–50 mmHg creates little haemodynamic effect. At a $PaCO_2$ of 55–70 mmHg, hypercapnia and acidosis have direct and indirect actions. Hypercapnia directly depresses myocardial contractility and causes vasodilation. This is counteracted by central sympathetic stimulation, which enhances heart rate, myocardial contractility and vasoconstriction. Overall, the indirect effect due to sympathetic stimulation is greater.

Renal effects

Increased IAP reduces renal blood flow, glomerular filtration rate, and urine output.[6] In animal models, oliguria and reduction in renal blood flow is worse with higher IAP. Reduced renal blood flow may be due to a direct pressure effect on renal cortical blood flow and renal vasculature, as well as an increase in antidiuretic hormone, aldosterone and renin.

Urinary N-acetyl-β-D-glucosaminidase (U-NAG) is a sensitive marker for tubular cell damage. Pneumoperitoneum has no obvious effect on U-NAG or serum creatinine levels.[6] There is no evidence to suggest long-term harmful effects on renal function.

Effects on other systems

Pneumoperitoneum increases intracranial pressure (ICP) and reduces cerebral perfusion pressure which has little clinical significance in healthy individuals.

Increased IAP reduces femoral venous flow, indicating an increase in venous stasis.[8] This is due to increased pressure on the inferior vena cava and iliac veins, which reduces venous blood flow in the lower limbs.

Postoperative C-reactive protein (CRP) and interleukin-6 (IL-6) levels are less elevated after laparoscopic surgeries compared to open surgery, suggesting an attenuation of the surgical inflammatory response.[6] IL-6 is an important cytokine mediating the acute phase response, and its level increases in proportion to tissue trauma.[6] CRP is lower when CO_2 is used compared to helium and abdominal wall lifting.[6] IL-6 is also lower when CO_2 is used compared to air.[6] CO_2 may somehow independently reduce the inflammatory response.

Pneumoperitoneum has been shown to reduce portal blood flow by 53%,[8] which may lead to transient elevation of liver enzymes (ALT and AST).

Trendelenburg position

Head down position increases venous return, which tends to increase stroke volume and cardiac output back towards normal.

Respiratory function is further impaired because cephalad shifting of the diaphragm is exaggerated. Functional residual capacity, total lung volume, and lung compliance are reduced, and atelectasis is worsened. Intracranial pressure is increased more.