Cardiovascular collapse after laparoscopic liver biopsy

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Summary
We describe a unique complication of laparoscopic liver biopsy. The increased intra-abdominal pressure associated with carbon dioxide pneumoperitoneum produced haemodynamic stability by tamponading a trochar-induced retroperitoneal haemorrhage. After deflation of the abdomen, release of the tamponade resulted in acute cardiovascular collapse. Other complications associated with laparoscopic surgery are also discussed. (Br. J. Anaesth. 1995; 75: 782–784)

Key words
Surgery, laparoscopy, Complications, haemorrhage.

Laparoscopic surgery has many advantages compared with conventional surgery, including decreases in postoperative pain, pulmonary dysfunction and duration of hospital stay [1, 2]. In addition, recent reports have documented the efficacy and safety of laparoscopy for previously contraindicated conditions, such as previous abdominal surgery, severe cardiopulmonary disease, cirrhosis, portal hypertension and bleeding diathesis [3, 4]. An increasing number of patients with liver dysfunction have successfully undergone laparoscopic procedures [4, 5]. We report a complication of laparoscopic liver biopsy not described previously—cardiovascular collapse and death from retroperitoneal haemorrhage. Other potential complications unique to laparoscopic surgery are reviewed to emphasize the risks likely to be encountered by the anaesthetist and surgeon when using this technique.

Case report
A 52-yr-old, 55-kg Caucasian female presented for liver transplant evaluation, including laparoscopic abdominal exploration and liver biopsy. The laparoscopic approach was proposed in order to minimize postoperative pain and pulmonary dysfunction. Her past medical history included 10 yr of steroid-dependent ulcerative colitis with sequelae including leucocytoclastic vasculitis, arthritis, nephritis, post-transfusion chronic hepatitis C and increased aspartate aminotransferase (AST) (250 iu litre−1) and alanine aminotransferase (ALT) (350 iu litre−1) concentrations. In addition, she had a history of chronic renal insufficiency with a serum creatinine concentration of 177 mmol litre−1. She was receiving medication with frusemide, prednisolone, 6-mercaptopurine and verapamil. Recently, she had developed ascites refractory to medical therapy and her bilirubin concentration had increased to 236 mmol dl−1 and alkaline phosphatase to 200 iu litre−1. A diagnosis of sclerosing cholangitis was proposed.

There was no family history of anaesthesia-related difficulties and the patient underwent uncomplicated Caesarean delivery at 23 yr of age. On physical examination, she was jaundiced and had a cushingoid appearance. Examination of the airway, heart and lungs was unremarkable. Her abdomen was distended, but there was no hepatosplenomegaly. She had bilateral pitting oedema extending up to the knees. Preoperative laboratory studies revealed a prothrombin time of 17.5 s (normal 11–13 s) and platelet count of 34000. ECG and chest x-ray were unremarkable and her haemoglobin concentration was 14.0 g dl−1. Echocardiography revealed normal left ventricular function. Pulmonary function testing revealed a mild restrictive pattern.

The patient was taken to the operating room and ECG, arterial pressure cuff and pulse oximeter monitors were attached. Anaesthesia and neuromuscular block were induced via a left internal jugular, triple-lumen i.v. catheter using fentanyl 100 μg, thiopentone 250 mg and atracurium 30 mg. Anaesthesia was maintained with isoflurane and nitrous oxide in oxygen. Throughout induction and maintenance of anaesthesia, arterial pressure remained unchanged at approximately 100/70 mm Hg and heart rate at 80–90 beat min−1. A Verres’ needle was inserted below the umbilicus and the abdomen insufflated to a pressure of 15 mm Hg. A 10-mm pyramidal tip trochar was inserted into the peritoneal cavity above the umbilicus and a cannula positioned at this site maintained insufflation for surgery at a pressure of 15 mm Hg. Ascites fluid (1600 ml) was evacuated. Two additional trochars and cannulae were placed in the upper abdomen under direct visualization, one near the right midclavicular line at the level of the umbilicus and one to the left of the midline at the level of the 10th rib. The lungs were ventilated artificially to maintain endtidal carbon dioxide partial pressure at approximately 4.0 kPa. Coagulopathy was treated by i.v. administration of 4 u. of fresh frozen plasma and 6 u. of platelets before liver biopsy. Lactated Ringer’s solution (600 ml) was given. Exploration and liver biopsy proceeded without incident. Urinary output

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during the 1-h procedure was 100 ml. The laparoscope and trochar were removed after abdominal deflation.

During skin closure, while the lungs were ventilated with an \( P_{\text{F}}O_2 \) of 1.0 and end-tidal isoflurane concentration decreased to 0.1 %, the patient's arterial pressure decreased to 85/60 mm Hg. Heart rate remained unchanged at 80 beat min\(^{-1}\) and \( S_{\text{PO}}2 \) was 98 %. Normal saline 1 litre was given. One minute later, her arterial pressure was 60/45 mm Hg, without a change in heart rate or oxygen saturation. Crystalloid fluid administration was continued, phenylephrine 100 \( \mu \)g was given and isoflurane was discontinued. Oxygen flow was maintained, without a change in heart rate or oxygen desaturation. The trachea was midline and breath sounds were normal and heard bilaterally.

During the next minute, the patient developed bradycardia and oxygenation desaturation to \( S_{\text{PO}}2 \) of 90 %, and arterial pressure became unmeasurable by cuff. Despite administration of another 100 \( \mu \)g of phenylephrine, rapid deterioration to an agonal rhythm was apparent on the ECG. No pulse was palpable and external cardiac compression was commenced. Adrenaline 1.0 mg was administered by both the i.v. and tracheal routes (2 mg total). An arterial blood gas sample was obtained at an \( P_{\text{F}}O_2 \) of 1.0 with \( pH = 7.37, P_{\text{CO}}2 = 4.3 \text{ kPa}, P_{\text{O}}2 = 70 \text{ kPa}, HCO_3^- = 18.8 \text{ mmol litre}^{-1}; \) haemoglobin concentration = 5.4 g dl\(^{-1}\); Na = 133 mmol litre\(^{-1}\) and K = 4.7 mmol litre\(^{-1}\). An 8.5-French gauge i.v. catheter was placed in the right internal jugular vein to facilitate fluid administration.

After rapid preparation, laparotomy was performed. Exploration revealed no significant blood collection in the abdominal cavity. The incision was extended cephalad to perform a sternotomy, which revealed no major fluid collection in the mediastinum or left thoracic cavity. A right thoracostomy tube was inserted and this drained only 200 ml of serosanguineous fluid. The aorta was cross-clamped and direct cardiac massage was commenced. The surgeon reported inadequate filling of the ventricles, and colloid, crystalloid and packed red blood cells were administered via an in-line fluid warmer. A total of 7 mg of adrenaline was administered in divided doses by the i.v., tracheal and intracardiac routes. Atropine 1 mg and calcium chloride 1 g were also administered i.v. Direct cardioversion was attempted without success. A total of 7.5 ml of packed red blood cells, 2.5 litre of colloid solution and 1.5 litre of crystalloid solution were administered, after which the surgeon noted that the ventricles remained inadequately filled. After 40 min of resuscitation the patient still had no intrinsic cardiac rhythm, pulse or arterial pressure. At this point, resuscitation efforts were discontinued.

After discussion with the family, a post-mortem examination was performed. Significant observations at post-mortem examination included: a haemorrhage of 16 × 8 × 8 cm surrounding the right kidney, and purpura and lividity of the upper back; also noted were micronodular cirrhosis of the liver secondary to hepatitis C, small bilateral pleural effusions and acute pericardial haemorrhages secondary to direct cardiac compression. There was no evidence of coronary artery disease. A specific area of disruption could not be identified by visual inspection of the inferior vena cava or aorta. Death was attributed to acute retroperitoneal haemorrhage that became apparent after decompression of the abdominal cavity.

**Discussion**

Although minimally invasive, laparoscopy entails anaesthetic management of the same degree of complexity as open surgery. For example, the incidence of technical and haemorrhagic complications is similar [1, 6]. Although mortality is reported to be substantially lower with the laparoscopic approach (0.07 vs 1.8 %) [1, 6], this finding may have resulted from the better general health of the patients undergoing laparoscopy compared with open surgery [7].

Perioperative complications associated with laparoscopy are attributable to trochar insertion, Trendelenburg or reverse Trendelenburg position, direct effects of pneumoperitoneum and systemic effects of carbon dioxide [3, 8]. The Trendelenburg position may cause atelectasis and worsen intra-pulmonary shunt, while the reverse Trendelenburg position may cause decreased venous return. A potential for endobronchial intubation and regurgitation are recognized hazards of the Trendelenburg position.

Pneumoperitoneum may result in subcutaneous emphysema, pneumothorax, carbon dioxide embolism, arrhythmias (usually bradycardia) caused by visceral distension and impaired venous return with reduced cardiac output [3, 8]. A diagnosis of pneumothorax was considered in our patient, but the absence of bilateral breath sounds and the lack of clinical response to thoracic decompression excluded this aetiology. The presence of satisfactory oxygenation and ventilation, as indicated by arterial blood-gas analysis, also excluded the possibility of pneumothorax or venous gas embolism.

Exogenous carbon dioxide absorption produces direct systemic effects. Hypercapnia increases the tendency towards arrhythmias, especially in the presence of halothane anaesthesia [3]. An increased tendency to respiratory acidosis has been reported in patients with cardiac or pulmonary disease [9]. However, ventilation in our patient was adequate, as evidenced by arterial blood-gas analysis.

Other causes of acute cardiovascular collapse include electrolyte abnormalities, pulmonary embolism, myocardial infarction and anaphylaxis. Laboratory studies of blood obtained at resuscitation and inability of vigorous fluid therapy to fill the ventricle during open cardiac massage make these diagnoses less tenable. Post-mortem examination of our patient also provided no evidence for these causes, only the signs of vigorous direct compression manoeuvres.

Epigastric vessel injuries which may result in free bleeding or haematoma formation are known complications related to the type of trochar used and also
the site of insertion [10, 11]. Perforation of the gastrointestinal tract and tearing of the viscera (liver and spleen) also have been reported [8]. Major vascular disruption has occurred, the most common site being the aortic bifurcation [12]. Haemorrhagic complications associated with laparoscopy, such as hepatic subcapsular haematoma, capsular laceration and epigastric vessel injury, have been described in previous reports [8, 10, 11]. Many have occurred at a significant distance from the operative site and been neither suspected nor detected because of the limited range of vision of the laparoscope.

Post-mortem examination of our patient revealed haemorrhage around the right kidney and purpura of the back, indicating that retroperitoneal haemorrhage had caused the acute hypovolaemia. Given the rapidity of the patient’s cardiovascular collapse, disruption along a great vessel, in this case at the level of the renal system, was the most likely cause. Laboratory studies of the immediate resuscitation level of the renal system, was the most likely cause.

The fluids given during resuscitation did not enter the cardiac chambers, but apparently followed the path of least resistance and entered the retroperitoneal space at the level of the renal vein. Because of the low pressure of the venous system, potential haemorrhage may be subject to tamponade and not clinically apparent until after the pneumoperitoneum is decompressed.

This case is unique from other reports of haemorrhage associated with laparoscopy. In other reports, the delay in discovering bleeding was attributed to the limited field of view afforded by the laparoscope [10]. In our case, clinically detectable haemorrhage was delayed because of the tamponade effect produced by the pneumoperitoneum. There was no evidence of injury on visual examination of the peritoneum before deflation. Laparoscopy has been shown to detect preoperative retroperitoneal trauma [13]. However, cases of venous injury may be only first noted after deflation of the pneumoperitoneum [14]. In this case, our patient’s coagulopathy certainly contributed to the haemorrhage.

Transoesophageal echocardiography has been suggested as a means to assess left ventricular ejection fraction, preload and afterload in selected patients undergoing laparoscopic surgery [15]. This technique may provide important information in acute situations for those patients predisposed to cardiac dysfunction. In addition, application of this monitor might allow documentation of carbon dioxide embolism or pneumopericardium.

In summary, we have described cardiac arrest and death secondary to hypovolaemia after laparoscopy. Intraoperative bleeding was not detected clinically because of the tamponade effects of the pneumoperitoneum. Deflation of the abdomen resulted in cardiovascular collapse and the patient was unresponsive to fluid resuscitation. Post-mortem examination revealed marked retroperitoneal haematoma as the likely site of haemorrhage. The laparoscopic technique may carry significant risks for the patient with major cardiac, pulmonary or hepatic disease. Further study on morbidity and mortality of laparoscopy for this compromised patient population would provide guidelines for clinicians in selecting the optimal surgical and anaesthetic management.

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References