Perioperative management of patients with increased risk of laparoscopy-induced hepatic hypoperfusion

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Abstract

Because hepatic hypoperfusion induced by laparoscopy has been underestimated, the aim of this article is to review the numerous factors influencing hepatosplanchnic blood flow during laparoscopy and to alert clinicians to the adverse consequences of hepatic hypoperfusion in high risk patients undergoing this procedure.

Reference


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Perioperative management of patients with increased risk of laparoscopy-induced hepatic hypoperfusion

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Summary

Because hepatic hypoperfusion induced by laparoscopy has been underestimated, the aim of this article is to review the numerous factors influencing hepatosplanchnic blood flow during laparoscopy and to alert clinicians to the adverse consequences of hepatic hypoperfusion in high risk patients undergoing this procedure.

Key words: laparoscopy; hepatic circulation; human studies; experimental studies; high-risk patients

Introduction

Indications for laparoscopic versus open surgery have rapidly widened, since the technique shortens postoperative recovery time and reduces postoperative complications [1–3]. Well-accepted indications for laparoscopy are cholecystectomy and diagnostic procedures such as evaluation of abdominal pain and haemorrhage and staging of malignant tumours [3]. Appendicectomy, exploration of the common bile duct, repair of inguinal hernia, colon resection, surgery for gastro-oesophageal reflux and peptic ulcer disease are gaining acceptance. Many other laparoscopic procedures are performed as technical limitations are addressed by numerous investigators and manufacturers.

During laparoscopy, a working space to facilitate surgery is established by continuously insufflating an inert gas in the peritoneal cavity whose volume should be large enough to facilitate surgery without increasing intra-abdominal pressure (IAP) over a threshold limit (usually 15 mm Hg). The most commonly used gas is CO₂, since it permits safe electrocautery and is rapidly absorbed and dissolved into vessels, thus minimising the risk of gas embolism. Potential complications described during intra-abdominal CO₂ insufflation include abdominal injuries and cardiovascular and respiratory disorders, which are easily avoided in low-risk patients undergoing short-time surgery. However, while the procedure is gaining acceptance among general surgeons, laparoscopy is now performed in high risk patients, including elderly patients with pre-existing cardiovascular diseases, patients with hepatic dysfunction and critically ill patients.

Because hepatic hypoperfusion induced by laparoscopy has been underestimated, the aim of this article is to review the numerous factors influencing hepatosplanchnic blood flow during laparoscopy (table 1) and to alert clinicians to the adverse consequences of hepatic hypoperfusion in high-risk patients undergoing this procedure.

Table 1
Factors influencing hepatic perfusion during laparoscopy.

<table>
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Pathophysiology of laparoscopy-induced hepatic hypoperfusion

Increase in intra-abdominal pressure (IAP)

Few studies have investigated the consequences of intraabdominal CO₂ insufflation on hepatosplanchnic perfusion. In healthy patients undergoing cholecystectomy and appendicectomy, Schilling et al. [4] measured the microcirculation of abdominal organs by introducing a laser Doppler into the peritoneum through a 5-mm trocar. During CO₂ insufflation (IAP = 15 mm Hg) there was a significant decrease in the gastric
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creasing IAP up to 20 mm Hg [14] or 12 mm Hg.

In contrast, in anaesthetised dogs and pigs, in-
put also decreases and hepatic hypoperfusion

increased while the hepatic blood flow was well main-
tained during open cholecystectomy.

In experimental studies, numerous but con-
troversial results have been published concerning
the modification of hepatic and mesenteric blood
flows during elevated IAP. Experimental designs
are likely to be responsible for these controversies.
In early studies, IAP was increased by infusing bal-
anced salt solutions [8, 9] or by inflating bags [10,
11] in the abdomen to mimic the increased IAP ob-
erved in massive ascites, bowel distention, and
omphalocoele in newborns. Recent studies have
used a pneumoperitoneum to increase IAP to mimic laparoscopy, but the gas insufflated was ei-
ther CO2 [12] or helium [13]. In anaesthetised pigs,
Diebel et al. [8] showed that increased IAP impairs
hepatic perfusion. In this study, despite the steady
mean arterial pressure and cardiac output mea-
sured during the procedure, portal vein blood and
hepatic artery blood flows fall to 65% and 45%
of the baseline value respectively at an IAP of 20 mm
Hg. When IAP reached 40 mm Hg, cardiac out-
put also decreases and hepatic hypoperfusion
worsens. Mesenteric blood flow also decreases
when IAP increases up to 20 mm Hg [9, 10, 13].
In contrast, in anaesthetised dogs and pigs, in-
creasing IAP up to 20 mm Hg [14] or 12 mm Hg
Finally, when IAP is increased by CO2 insufflation
in anaesthetised dogs, portal vein blood flow de-
creases but hepatic artery blood flow is maintained
[12].

Because portal vein blood and hepatic artery
blood flows may be differently regulated during
abdominal CO2 insufflation, several experimental
studies have investigated the “hepatic arterial
buffer response” which is defined as the inverse
change in hepatic arterial resistance in response to
modification of portal vein blood flow. Thus, in
physiological conditions, hepatic artery blood flow
increases to compensate for a decrease in portal
vein blood flow to maintain constant hepatic per-
fusion. Richter et al. [16] recently showed that in
anaesthetised rats the hepatic arterial buffer re-
response was altered during intra-abdominal CO2
insufflation, a fact which may represent a further
risk factor for hepatic hypoperfusion during la-
paroscopy.

Although interspecies differences may explain
the controversial effects of IAP increase on hepatic
blood flow, it is possible to conclude that in exper-
imental models hepatic blood flow is either main-
tained or decreased, and that local regulation of
hepatic and portal blood flows is impaired.

Changes of position

To increase the working space, head-down tilt
position is used during intestinal surgery while
head-up tilt position facilitates surgery in the eso-
gastric area. Because in awake volunteers, head-
down tilt increases the cardiopulmonary blood
volume with a concomitant increase in cardiac out-
put [17], the combination of intraabdominal CO2
insufflation and head-down tilt may be more ef-
effective in preserving hepatic perfusion than the
combination of head-up tilt position and intra-ab-
dominal CO2 insufflation. In 1972, Kelman et al.
[18] showed that a progressive increase in IAP up
to 20 cm H2O increases cardiac output in both the
horizontal and head-down tilts. This result was not
confirmed in healthy women who underwent la-
paroscopic hysterectomy. In this study, the combi-
nation of anaesthesia, head-down tilt and pneu-
operitoneum decreases cardiac output [19]. Fi-
ally, Joris et al. [20] showed that the combination
of anaesthesia, head-up tilt and pneumoperi-
toneum produces a 30% decrease in cardiac out-
put in healthy patients during laparoscopic chole-
cystectomy. No information is available concern-
ing modification of hepatic perfusion by a combi-
nation of position changes and intra-abdominal
CO2 insufflation in anaesthetised patients.

We showed that in anaesthetised pigs head-
down tilt before CO2 insufflation increases portal
vein blood flow, while head-up tilt decreases both
hepatic flows [21]. In this experimental model, the
combination of CO2 insufflation and changes in
position had a beneficial effect on hepatic perfu-
sion in the head-down tilt, whereas hepatic blood
flow was not modified by the combination of head-
up tilt and CO2 insufflation. Further studies are
therefore needed to obtain such information in
humans.

Anaesthetic technique

Anaesthetic drugs and volaemic status also in-
tereference with hepatosplanchnic perfusion. All drugs
that decrease cardiac output produce proportional
effects on hepatic blood flow. In addition, some
anaesthetic agents have more specific effects on
hepatic perfusion. For instance, while halothane
increases hepatic arterial resistance, isoflurane in-
creases regional blood flow. Other drugs such as
pancuronium and fentanyl do not significantly af-
flect hepatic blood flow [22]. Additionally, epidural
anaesthesia may modify hepatic flow to an extent
dependent on the block level.

Volaemic status

Volaemic status also interferes with haemody-
namic variables. Increasing IAP up to 40 mm Hg
decreases cardiac output by 53% in hypovolaemic
dogs and by 17% in normovolaemic dogs, but
raises cardiac output by 50% in hypervolaemic

(–53%), duodenal (–11%), jejunal (–32%), colonic
(–44%), hepatic (–39%), and peritoneal (–60%)
microcirculations. By inserting a pulsed Doppler
ultrasonic probe through the umbilicus, Takagi [5]
also showed that portal vein blood flow decreased
when IAP was ≥10 mm Hg in a similar group of
patients. The decreased blood flow is proportional
to the increase in IAP [6]. Finally, in healthy el-
derly patients Sato et al. [7] assessed hepatic blood
flow by transoesophageal Doppler echography of
the hepatic vein during cholecystectomy, and
showed that hepatic perfusion is significantly al-
terred while the hepatic blood flow was well main-
tained during open cholecystectomy.
dogs [23]. We have confirmed these results in normovolaemic pigs [21]. Since in clinical studies cardiac output is either unchanged [24, 25], increased [18] or decreased [19, 20, 26] during pneumoperitoneum, these conflicting findings may be explained by the volaemic status of anaesthetised patients.

**Effect of pH and pCO₂**

Low pH and high pCO₂ may modify hepatic blood flow during intra-abdominal CO₂ insufflation. Thus, CO₂ easily diffuses through the peritoneum, increasing blood pCO₂. Hypercarbic acidosis and its deleterious effects on cardiac output and regional circulation can easily be avoided by increasing ventilation. However, in patients with impaired cardiac output or pulmonary dysfunction, CO₂ transport from the peritoneal cavity to the lungs is slow and increased CO₂ retention must be detected. This is why several studies have assessed the consequences of insufflating different types of gas as well as the possibility of avoiding gas insufflation (abdominal-wall lift technique or retractor method) to limit the vascular effects of CO₂.

The consequences of high pCO₂ in blood have been investigated in experimental studies. Gelman et al. [27] showed that low pH and high pCO₂ increase hepatic arterial blood flow and reduce portal blood flow. In anaesthetised pigs we measured pH and pCO₂ in the portal vein during CO₂ insufflation and found minimal changes: pH and pCO₂ in the portal vein were 7.29 ± 0.02 and 8.50 ± 0.30 kPa respectively [21]. However, in this experimental model we modified mechanical ventilation to keep arterial pCO₂ within normal ranges. Because these changes were lower than those necessary to modify hepatic blood flow [27], we postulated that variations in pH and pCO₂ during laparoscopy had only a minor effect on hepatic blood flow [21]. Moreover, we recently showed that in isolated perfused rat livers metabolic and hypercarbic acidosis has no effect on hepatic flow and on the response to increasing doses of norepinephrine [28]. Hence, in clinical practice, modifications of pH and pCO₂ in hepatic vessels should have little effect on hepatic blood flow.

**Outcome**

As shown in the previous sections, numerous factors modify hepatic perfusion and the consequences of such hypoperfusion on hepatic function and outcome need to be further investigated. Few studies have investigated hepatic tests after laparoscopic surgery. Saber et al. [29] compared hepatic enzyme release following uneventful open and laparoscopic cholecystectomy by day 2 after surgery. They found that alanine aminotransferases doubled in 58% of healthy patients who had undergone laparoscopy and in only 6% of those who had undergone open surgery. The results were similar for aspartate aminotransferases, and all values returned to baseline by day 7. Similar results were published by Tan et al. [30] and Andrei et al. [31]. Interestingly, Kotabe et al. [32] showed that the degree of hepatic injury may be higher in laparoscopic colectomy than in laparoscopic cholecystectomy. Because the increase in IAP was similar for the two surgical procedures, the increased injury in cholecystectomy may result from patient position. Another important proposition has recently been advanced in a rat laparoscopy model [33]. Preconditioning (a 10-min insufflation followed by a 10-min deflation) may prevent the hepatic injury induced by a 60-min insufflation.

From these studies it may be concluded that in healthy patients undergoing laparoscopic surgery the transient hepatic hypoperfusion induced by laparoscopic surgery probably has no effect on hepatic function and outcome. However, hepatic enzyme release following long-lasting laparoscopy has never been published in large groups of patients. In these circumstances, prolonged hypoperfusion may be more deleterious [30].

**Clinical implications in high risk patients**

As stated previously, most publications on laparoscopic interventions involve young and healthy patients. With the rapid advances in laparoscopic technology and surgical skills the procedure is now performed in high risk patients, including elderly patients with pre-existing cardiovascular diseases, patients with hepatic dysfunction and critically ill patients. In these patients, the alterations in hepatic blood flow and hepatic functions are likely to be more deleterious that in healthy subjects. However, these questions have never been investigated.

It has long been known that postoperative complications and mortality within 30 days of surgery are high (30.1% and 11.6% respectively) in patients with cirrhosis undergoing all types of surgical intervention under anaesthesia [34]. In contrast, several studies have recently emphasised the uneventful outcome of laparoscopy in patients with cirrhosis. In 25 consecutive patients with mild cirrhosis (Child A and B) who underwent laparoscopic cholecystectomy, all patients survived and the hospital stay (1.7 days) was similar to that observed in healthy patients [35]. Postoperative complications were haematomas (n = 3), ascites (n = 3), and pneumonia (n = 2). Patients with mild cirrhosis tolerate laparoscopic cholecystectomy nearly as well as those without cirrhosis [36]. Advantages of laparoscopic cholecystectomy over open surgery have also been shown by Poggio et al. [37]. Patients with mild cirrhosis tolerate laparoscopy better than open surgery (including lower mortality,
shorter operative time and reduced hospital stay. If this is confirmed, the reason why laparoscopic surgery is well tolerated in patients with cirrhosis deserves further investigation.

Diagnostic laparoscopy for acute haemorrhagic trauma is also gaining acceptance among general surgeons. The indications for diagnostic laparoscopy are similar to those for diagnostic peritoneal lavage. Diagnostic laparoscopy is restricted to haemodynamically stable patients because abdominal CO2 insufflation is deleterious in patients with patent haemorrhagic shock [38]. Diagnostic laparoscopy has been safely performed in intensive care patients (n = 25) with suspicion of acute surgical abdomen [39]. No significant change in blood pressure, end-tidal CO2, arterial pH or cardiac output was observed during the procedure. The two complications reported (abdominal bleeding and small bowel injury) did not modify the patient outcome. Finally, laparoscopy has also been safely performed in patients with coexisting diseases, it is likely that hepatic blood flow would be further impaired in this group of patients.

Clinical implications

In healthy patients, hepatic blood flow decreases during laparoscopic cholecystectomy. In addition to increased IAP, numerous factors interfere with regional flow during the procedure: changes of position, anaesthesia and volaemic status, as well as changes in intravascular pH and pCO2. However, the consequences of hepatic hypoperfusion remain minor in healthy patients. No study has measured hepatic perfusion and hepatic functions in high risk patients and long-lasting surgery. Interestingly, in cirrhotic patients with mild disease, the benefits of laparoscopy seem similar to those observed in healthy patients. Thus, although successful laparoscopy has been performed in high risk patients, the numerous factors interfering with hepatic blood flow must be borne in mind. Low IAP, minimum tilt during changes in position, careful monitoring of volaemic status, haemodynamic stability during anaesthesia and correction of hypercarbic acidosis should prevent laparoscopy-induced hepatic hypoperfusion in high risk patients (table 2). Moreover, hepatic tests should be measured in the postoperative period to detect hepatic dysfunction.

Table 2
Clinical implications in high risk patients.

<table>
<thead>
<tr>
<th>Low IAP</th>
<th>Minimum tilt during changes of position</th>
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