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Elevated intrahepatic pressures and decreased hepatic tissue blood flow prevent gas embolus during limited laparoscopic liver resections

R. Ricciardi, S. Anwaruddin, B. K. Schaffer, S. H. Quarfordt, S. E. Donohue, S. M. Wheeler, K. A. Gallagher, M. P. Callery, D. E. M. Litwin, W. C. Meyers

Department of Surgery, University of Massachusetts Medical School, 55 Lake Avenue, North, Worcester, MA 01655, USA

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Abstract
Background: As new techniques are emerging for laparoscopic liver resections, concerns have been raised about the development of gas embolus related to the CO_2 pneumoperitoneum. We hypothesized that elevated intrahepatic vascular pressures and decreased hepatic tissue blood flow (LQ_B) would prevent gas embolus during laparoscopic liver resections under conventional pneumoperitoneum.
Methods: Intrahepatic vascular pressures and LQ_B were measured in nine pigs with varying CO_2 pneumoperitoneum. Gas embolus was determined after hepatic incision by monitoring pulmonary arterial pressure (PAP), hepatic venous P_{CO_2}, systemic blood pressure (SBP), and suprahepatic vena cava ultrasound.
Results: As the pneumoperitoneum was increased from 0 to 15 mmHg, intrahepatic vascular pressures increased significantly ($p < 0.05$), while LQ_B decreased significantly ($p < 0.05$). A 2.0-cm hepatic incision at 4, 8, 15, and 20mmHg produced no ultrasound evidence of gas embolus and no changes in PAP, SBP, or hepatic venous P_{CO_2} (p = NS).
Conclusion: These data suggest that the risk of significant embolus under conventional pneumoperitoneum is minimal during laparoscopic liver resections.

Key words: Laparoscopy — Liver — Embolus — Blood flow — CO_2

By now, laparoscopy has become an integral part of a number of surgical procedures because it offers a shorter postoperative course, better cosmesis, and less pain than conventional methods. Indeed, both the medical and financial outcomes are more favorable with these new techniques. To take advantage of the benefits of minimally invasive surgery, more complex procedures are now being attempted laparoscopically. In the area of hepatobiliary surgery, small case studies have shown the utility of laparoscopy in liver resections [4, 11], but the technical limitations of laparoscopic hepatic surgery continue to be investigated.

Laparoscopic hepatic procedures can be performed with one of two methods—either the conventional CO_2 pneumoperitoneum [3] or an abdominal wall lift device [5]. Although each of these techniques has certain advantages, they also have their disadvantages. For instance, the abdominal wall lift device has been criticized for not providing adequate intraabdominal visualization for complex hepatic resections [11]. On the other hand, one potentially catastrophic complication of conventional pneumoperitoneum is the presumed risk of gas embolus during the inadvertent incision of a large venous channel, either in the liver or elsewhere [5, 11]. Although several studies have focused on the feasibility and safety of minimally invasive laparoscopic hepatic resections, the physiological mechanisms responsible for preventing gas embolization are not well understood.

Hepatic blood flow alterations have been described in many clinical conditions, including sepsis, ischemia reperfusion, and cirrhosis. We hypothesized that hepatic blood flow alterations occur with raised intraabdominal pressure during conventional pneumoperitoneum and act to prevent CO_2 embolization during laparoscopic liver resections. Therefore, we set out to evaluate intrahepatic hemodynamic parameters and their role in gas embolus during conventional laparoscopy.

Materials and methods

Animal preparation

Nine Yorkshire pigs weighing 30–50 kg were utilized for these experiments. All protocols were approved by the institution's animal review board.

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Correspondence to: R. Ricciardi

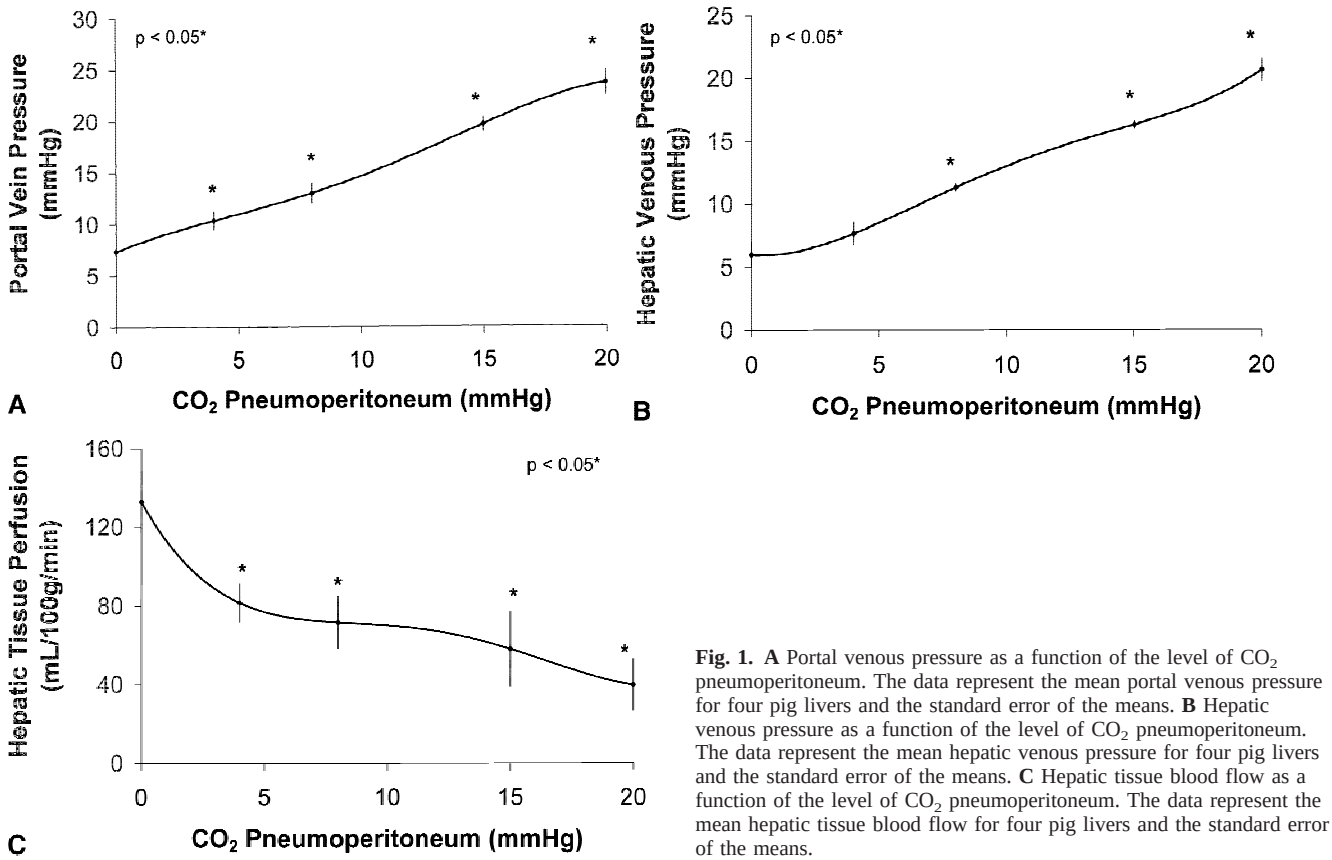

Fig. 1. **A** Portal venous pressure as a function of the level of CO_2 pneumoperitoneum. The data represent the mean portal venous pressure for four pig livers and the standard error of the means. **B** Hepatic venous pressure as a function of the level of CO_2 pneumoperitoneum. The data represent the mean hepatic venous pressure for four pig livers and the standard error of the means. **C** Hepatic tissue blood flow as a function of the level of CO_2 pneumoperitoneum. The data represent the mean hepatic tissue blood flow for four pig livers and the standard error of the means.

Sedation was provided with a mixture (1.0 ml/kg) of telazol (Ft. Dodge Animal Health, Ft. Dodge, IA, USA), ketamine, 50 mg/ml (Ft. Dodge Animal Health), and xylazine 10 mg/ml (Phoenix Pharmaceuticals, St. Joseph, MO, USA). The animals were endotracheally intubated, and anesthesia was provided by inhalation isoflurane (1.5%). The right femoral artery and right external jugular vein were cannulated for continuous blood pressure monitoring and intravenous infusions. A 24in-5-Fr cannula (Daig Co., Minnetonka, MN, USA) was placed in the left femoral vein and gently manipulated up to the mouth of the hepatic veins. A similar cannula was placed in the splenic vein through a small 4-cm subcostal incision prior to laparoscopy. The incision was closed and remained airtight throughout the laparoscopic portion of the experiment. A Swan-Ganz catheter was inserted via the left external jugular vein to monitor pulmonary artery pressure.

Laparoscopic protocol

A Veress needle was inserted midline at the level of the umbilicus. The peritoneal cavity was insufflated to a pneumoperitoneum of 15 mmHg. The laparoscopic camera was inserted through this umbilical trocar. Additional trocars (5–10 mm) were triangulated for the laparoscopic ultrasound and other surgical instruments. The laparoscopic ultrasound probe (B-K Medical Systems, Billerica, MA, USA) was maintained over the suprahepatic inferior vena cava after takedown of the falciform ligament. Accuracy of ultrasound placement was determined by color flow Doppler and venous waveform. A thermistor flow probe (Thermal Technologies, Cambridge MA, USA) was inserted into the liver parenchyma via a 14-gauge angiocatheter (Becton Dickenson, Sandy, UT, USA). Thermistor probes can delineate flow to one-tenth of a milliliter per 100 g of tissue per min.

Experimental protocol

After the peritoneum was desufflated, hepatic venous blood gases, hepatic venous pressure (HVP), portal venous pressure (PVP), hepatic tissue blood flow (LQ_B), pulmonary artery pressure (PAP), and systolic blood pressure (SBP) were recorded. These same measurements were repeated with CO_2 pneumoperitoneums of 4, 8, 15, and 20 mmHg.

After these data were collected, a 2.0-cm incision was made in the central portion of the liver parenchyma at a pneumoperitoneum of 4 mmHg. Hepatic venous blood gases, PAP, and SBP were recorded prior to and following the hepatic incision at 60 sec. The laparoscopic ultrasound image of the inferior vena cava at the hepatic veins was monitored continuously for 3 min following hepatic incisions. The procedure was repeated at pneumoperitoneums of 8, 15, and 20 mmHg. In other studies, changes in PAP, hepatic venous P_{CO_2}, SBP, and visualization of gas on ultrasound have all been utilized in detecting gas embolus.

To determine what volume of CO_2 would produce changes in PAP, hepatic venous P_{CO_2}, and SBP, as well as ultrasonic evidence of gas embolus, CO_2 gas was infused directly by Veress needle into the liver parenchyma. The volume of CO_2 gas infused was measured with 10- and 60-ml syringes. These experiments were performed under a conventional pneumoperitoneum of 15 mmHg.

Statistical analysis

Statistical analyses were determined with Student's *t*-test and ANOVA and considered significant when the p value was <0.05.

Results

Hepatic hemodynamics

As the pneumoperitoneum was increased from 0 to 20 mmHg, intrahepatic pressures increased significantly. PVP was 7.3 ± 0.3 at native intraabdominal pressure (0 mmHg) and increased significantly to 23.7 ± 1.2 mmHg at 20 mmHg ($p = 0.005$) (Fig. 1A). HVP was 6.0 ± 1.0 at native

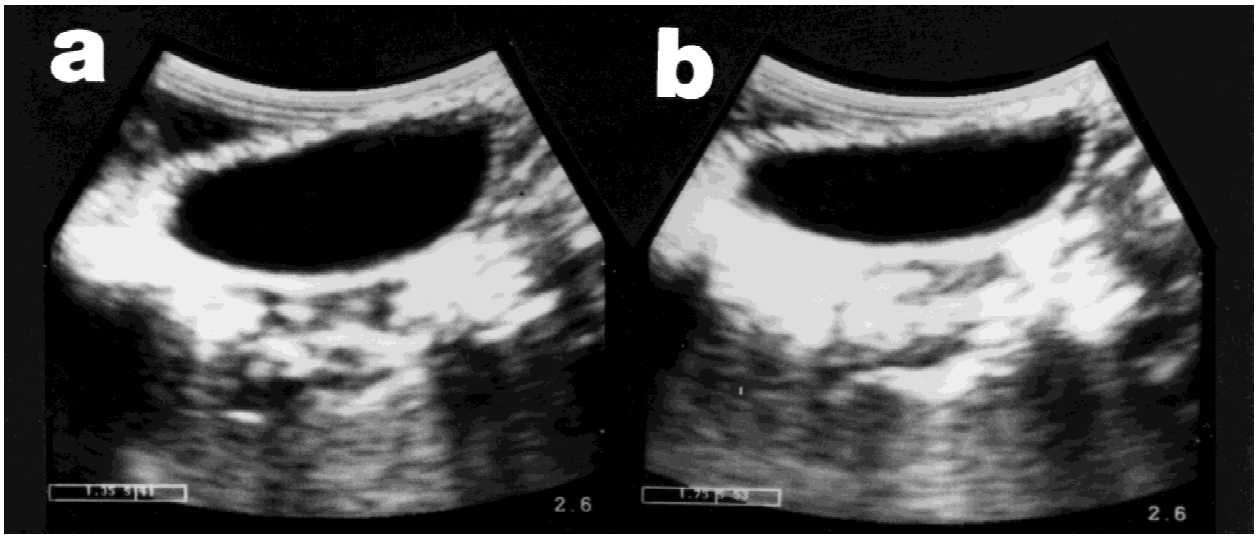

Fig. 2. **A** Ultrasound image of the suprahepatic inferior vena cava without evidence of gas embolus. **B** Ultrasound image of the suprahepatic inferior vena cava after infusion of 10 ml of CO_2 gas.

intraabdominal pressure and increased significantly to 20.7 ± 0.9 mmHg ($p = 0.001$) at a pneumoperitoneum of 20 mmHg (Fig. 1B). Conversely, there was a statistically significant decrease in LQ_B as intraabdominal pressure was increased from 0 to 20 mmHg (Fig. 1C). LQ_B at native intraabdominal pressure was 132.8 ± 15.7 and decreased to 39.8 ± 13.1 ml/100g/min at 20 mmHg ($p = 0.004$). Interestingly, there was no significant change in liver blood flow when comparing LQ_B at pneumoperitoneums of 4 and 15 mmHg. These data indicate that once a pneumoperitoneum is established, even minimal intraabdominal pressures significantly alter hepatic tissue blood flow.

Hepatic incisions

A 2.0-cm incision was placed centrally in the hepatic parenchyma. Following hepatic incisions, no evidence of gas embolus was noted with laparoscopic ultrasound at pneumoperitoneums of 4, 8, 15, and 20 mmHg (Fig. 2). Hepatic venous P_{CO_2} was also unchanged after all incisions at 4, 8, 15, and 20 mmHg (p = NS) (Table 1). Similarly, no difference in PAP or SBP was noted when comparing pressures before and after the parenchymal incision at the given pneumoperitoneum (p = NS). Similar purposeful incisions of large central vascular structures also produced no ultrasound evidence of CO_2 embolus or changes in PAP, SBP, or hepatic venous P_{CO_2}.

CO_2 gas infusions

Laparoscopic ultrasound evidence of gas embolus was observed only with direct infusions of CO_2 gas into the hepatic parenchyma through a Veress needle. A direct infusion of <10 ml of CO_2 produced evidence of gas embolus on ultrasound (Fig. 3). Significant changes in hepatic venous P_{CO_2}, PAP, and SBP, however, were only noted with larger volumes of infused CO_2 gas (Table 2). A significant increase in hepatic venous P_{CO_2} occurred upon infusion of

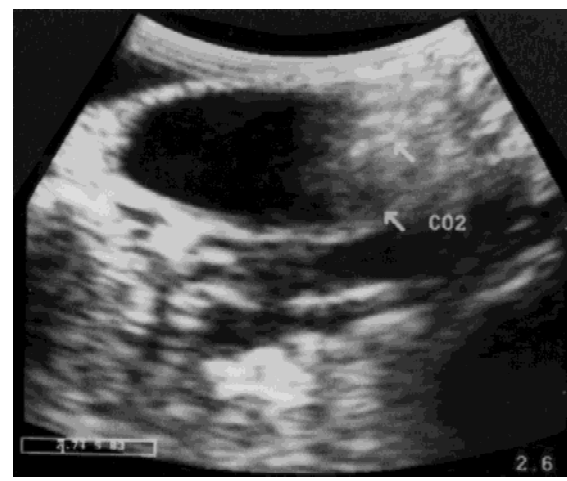

Fig. 3. Ultrasound image of the suprahepatic vena cava following an infusion of 10 ml of CO_2 gas. Arrows indicate gas embolus.

≥60 ml of CO_2 gas ($p = 0.04$). Prior to the infusion of 60 ml of CO_2 gas, no change in PAP was noted. Yet with direct infusion of 60 ml of CO_2 gas, a significant increase in PAP was noted ($p = 0.05$). Although both hepatic venous P_{CO_2} and PAP increased during infusions of 60 ml of CO_2 gas, no change in SBP was noted with this infused volume. A statistically significant decrease in SBP was only noted upon a total infusion of 300 ml of CO_2 ($p = 0.03$).

Discussion

The presumed risk of gas embolus with laparoscopic hepatic resections has limited the acceptance of these minimally invasive techniques. The present study indicates that the risk of gas embolus is minimal, even when large central vascular channels are incised. No evidence of gas embolus was detected by laparoscopic ultrasound, and no alterations of hepatic venous P_{CO_2}, pulmonary artery pressure, or systolic blood pressure were noted during hepatic incisions at any

Table 1. Mean pulmonary artery pressure, systolic blood pressure, and hepatic venous P_{CO_2} before and after a 2.0-cm incision in the central hepatic parenchyma of four pig livers

	CO_2 peritoneum	Gas embolus on ultrasound	Pulmonary artery pressure (mmHg)	Hepatic venous P_{CO_2} (mmHg)	Systolic blood pressure (mmHg)
Precut	4 mmHg	NA	26 ± 1.9	62 ± 2.2	97 ± 12.0
Postcut	4 mmHg	None	26 ± 3.2	60 ± 4.5	91 ± 8.0
Precut	8 mmHg	NA	32 ± 2.6	61 ± 6.3	110 ± 19.0
Postcut	8 mmHg	None	34 ± 3.0	60 ± 4.6	97 ± 11.0
Precut	15 mmHg	NA	32 ± 1.9	61 ± 4.4	99 ± 8.0
Postcut	15 mmHg	None	34 ± 2.4	61 ± 1.4	97 ± 8.0
Precut	20 mmHg	NA	33 ± 1.0	65 ± 2.4	105 ± 13.0
Postcut	20 mmHg	None	34 ± 2.9	67 ± 3.5	98 ± 12.0

Table 2. Mean pulmonary artery pressure, systolic blood pressure, and hepatic venous P_{CO_2}

Volume of CO_2 gas infused (ml)	Gas embolus on ultrasound	Pulmonary artery pressure (mmHg)	Hepatic venous P_{CO_2} (mmHg)	Systolic blood pressure (mmHg)
No gas	–	30.3 ± 0.2	55.4 ± 1.4	105.7 ± 3.9
2	+++	NA	NA	NA
10	+++	30.3 ± 1.0	56.5 ± 1.6	101.0 ± 2.3
20	+++	33.0 ± 0.9	64.4 ± 2.5	95.3 ± 1.7
40	+++	34.7 ± 0.7	65.3 ± 3.1	92.7 ± 1.8
60	+++	38.3 ± 1.0[a]	65.0 ± 1.3[a]	94.0 ± 4.5
120	+++	41.0 ± 1.5[a]	66.5 ± 3.5	85.7 ± 6.0
180	+++	43.3 ± 2.1	68.5 ± 3.0	84.3 ± 6.0
240	+++	41.0 ± 0.9[a]	75.0 ± 0.6[a]	79.7 ± 5.6
300	+++	39.0 ± 0.7	72.7 ± 3.8	77.0 ± 5.1[a]

[a] Denotes a statistically significant change as compared to time at which no gas was infused

level of CO_2 pneumoperitoneum. The lack of gas embolus may be explained by the protective effect of hepatic tissue stasis during the pneumoperitoneum. With elevations in intraabdominal pressure, portal venous and hepatic venous pressures also became elevated. Elevations in intraabdominal pressure are also associated with significant decreases in hepatic tissue blood flow as the pneumoperitoneum is raised from native intraabdominal pressure to 20 mmHg. These intrahepatic blood flow alterations may protect against gas embolus during hepatic resections.

Minimally invasive techniques have recently been developed in the area of hepatic surgery. One technique involves the limited resection of liver parenchyma under a standard pneumoperitoneum [3]. However, case reports have documented gas embolization as a consequence of the pneumoperitoneum, either during inadvertant Veress needle insertion [7] or due to use of an argon beam [2]. A second approach developed to minimize this complication utilizes an abdominal wall lift device without pneumoperitoneum to provide clear visualization of the intraabdominal contents [5]. In addition, a hybrid procedure has also been devised in which an abdominal wall lift device is utilized with a partial pneumoperitoneum of 4 mmHg in order to improve visualization [11]. It is assumed that this hybrid procedure entails little risk of gas embolus because the pneumoperitoneum pressure is lower than the native central venous and portal venous pressures. There are few data that demonstrate the relative safety of hepatic resections under CO_2 pneumoperitoneum, and no studies have attempted to evaluate the changes in hepatic vasculature and tissue blood flow on the production of gas embolus under a standard pneumoperitoneum.

As the pneumoperitoneum was raised from native pressure to 20 mmHg, the hepatic venous and portal venous pressures also rose significantly. In fact, these intrahepatic vascular pressures, both hepatic and portal venous, were consistently higher than the intraabdominal pressure set. A similar elevation of central venous pressure with increasing pneumoperitoneum has been described previously in a study evaluating laparoscopy in large animals [9]. Given the observed elevations in intrahepatic vascular pressures during conventional pneumoperitoneum, CO_2 gas must flow from an area of lower pressure (the peritoneum) to an area of higher pressure (the blood vessels) to produce an embolus. This possibility is highly unlikely. Another possibility may include dissolving of gaseous CO_2 into plasma during contact with surface-active agents at the incised hepatic surface. Yet our results demonstrate no change in hepatic venous P_{CO_2} following liver incisions, also making this possibility unlikely.

In addition to the observed increases in intrahepatic pressures of the conventional pneumoperitoneum, our data also demonstrate a considerable decrease in hepatic tissue blood flow. Other investigators have reported a similar reduction in tissue blood flow to the gastrointestinal organs with increasing intraabdominal pressures [1, 8, 10]. The decreased perfusion of the liver raises the question of whether increased intraabdominal pressure leads to functional damage of the liver. In those patients with limited hepatic reserve—i.e., cirrhosis—the potential risk of hepatic failure due to decreased hepatic tissue blood flow secondary to elevated intraabdominal pressure is an important consideration [6]. For this reason, it has been suggested that laparoscopic procedures should not be performed with a conventional pneumoperitoneum in patients with poor hepatic reserve. Yet, although hepatic tissue blood flow was significantly reduced when native intraabdominal pressure was increased to 15 mmHg, tissue flows at 15 mmHg were not significantly less than the tissue flows at 4 mmHg. These results indicate that the initiation of the pneumoperitoneum is a key contributor to decreased hepatic tissue blood flow. In addition, a standard pneumoperitoneum does not impact hepatic blood flow to a greater extent than a partial pneumoperitoneum in laparoscopic hepatic procedures.

Evidence of gas embolus was only observed with direct

infusion of CO_2 gas through a Veress needle inserted into the hepatic parenchyma. Infusions of minimal quantities of CO_2 gas (<10 ml) produced ultrasound evidence of gas embolus. With larger infusions of CO_2 gas, hepatic venous P_{CO_2}, pulmonary artery pressure and systolic blood pressure were similarly altered, indicating gas embolus. These data suggest that gas embolus can only occur when pressure surrounding the hepatic vasculature is overcome, as with forceful infusion of CO_2 gas under high pressure. This pathophysiology has been confirmed by case reports describing CO_2 embolization during Veress needle insertion [7]. Given the range of the pneumoperitoneums (4–20 mmHg) tested in the present study, intrahepatic pressures always remain higher than intraabdominal pressure, thereby preventing gas embolus.

The findings of the present study indicate that the intrahepatic pressure and blood flow alterations secondary to increased intraabdominal pressure protect against CO_2 embolus during minimally invasive hepatic resections. Although the risk of gas embolus is significant with accidental intravascular insertion of the Veress needle, the likelihood of a gas embolus from peritoneal gas entering through the hepatic parenchyma is minimal. Since visualization is better with a conventional pneumoperitoneum than with abdominal wall lift devices, trials of limited hepatic resections with a conventional pneumoperitoneum can proceed with minimal concern about the potential for gas embolus.

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