Continuing Medical Education

Acute ventilatory complications during laparoscopic upper abdominal surgery

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Purpose: This article examines and summarizes the published reports dealing with subcutaneous emphysema, pneumothorax and carbon dioxide (CO₂) embolism during laparoscopic upper abdominal surgery. The purpose is to describe the expected clinical picture, the differential diagnosis and the management of these complications.

Source: The information was obtained from a Medline literature search and the annual meeting supplements of *Anesthesiology*, *Anesth Analg*, *Br J Anaesth* and *Can J Anaesth*.

Principal findings: An abrupt increase in PETCO₂ is the first sign of subcutaneous emphysema and of pneumothorax. Desaturation and increased airway pressure occur with pneumothorax, but not with subcutaneous emphysema alone. Desaturation and increased airway pressure also occur with bronchial intubation. The preliminary diagnosis is made by verifying the position of the tube, examination of the patient for swelling and crepitus and auscultation for air entry. Chest radiography and paracentesis confirm the diagnosis of pneumothorax, which frequently occurs with subcutaneous emphysema but is rarely of the tension type. Pulmonary embolism due to CO₂ during LUAS has not been reported, but the available data suggest that small, haemodynamically inconsequential CO₂ embolism occurs without change in PETCO₂. Massive

embolism is possible and will markedly decrease PETCO₂, arterial O₂ saturation (SpO₂) and blood pressure.

Conclusion: The immediate recognition of the three complications requires continuous monitoring of PETCO₂, arterial saturation, airway pressure, and an index of pulmonary compliance.

Objectif: Ce travail étudie et résume les publications portant sur l'emphysème sous-cutané, le pneumothorax et l'embolisation au gaz carbonique (CO₂) survenant pendant la chirurgie laparoscopique de l'étage supérieur de l'abdomen. L'objectif est de décrire le tableau clinique, le diagnostic différentiel et le traitement de ces complications.

Source: Recherche informatisée sur Medline et les suppléments annuels d'*Anesthesiology*, *Anesth Analg*, *Br J Anaesth* et *Can J Anaesth*.

Résultats: Une augmentation subite de la PETCO₂ précède l'emphysème cutané et le pneumothorax. La désaturation et l'augmentation de pression aux voies aériennes surviennent avec le pneumothorax mais non avec l'emphysème cutané seul. Le diagnostic préliminaire se fait en vérifiant la position de la canule trachéale, en recherchant le gonflement et les crépitations, et en auscultant le thorax. La radiographie pulmonaire et la paracentèse confirment la diagnostic de pneumothorax qui survient fréquemment avec l'emphysème sous-cutané mais est rarement sous tension. On n'a pas rapporté d'embolies pulmonaires au CO₂ pendant la chirurgie laparoscopique de l'abdomen supérieur, mais les données actuelles suggèrent que de petites embolies de CO₂ sans conséquences hémodynamiques se produisent sans modifier la PETCO₂. L'embolisation massive demeure possible et diminue fortement la PETCO₂, la saturation artérielle en O₂ (SPO₂) et la pression artérielle.

Conclusion: La détection immédiate des trois complications mentionnées nécessite un monitoring continu de la PETCO₂, de la saturation en oxygène, de la pression des voies aériennes et d'un indice de la compliance pulmonaire.

Key words

COMPLICATIONS: respiratory, subcutaneous emphysema, pneumothorax, pulmonary embolism;

LUNG: subcutaneous emphysema, pneumothorax, pulmonary embolism;

SURGERY: laparoscopy.

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The laparoscopic approach to upper abdominal surgery (LUAS) now includes a large variety of major procedures (Table I) performed on larger portions of the surgical population, many of whom can be American Society of Anesthesiology (ASA) physical class III and IV. The sheer increase in the number of patients and the variety of operations performed should lead us to expect serious complications, which must be recognized immediately and managed appropriately. Two complications in particular, pneumothorax (? carbothorax), which is often preceded by subcutaneous emphysema, and pulmonary gas embolism, are potentially lethal. A number of case reports, articles and letters to the Editors dealing with these complications have been published. The purpose of this presentation is to collate and summarize the pertinent features of these case reports to provide a clear picture of the clinical presentation and of the differential diagnosis. An algorithm to aid in the latter is provided. The mechanisms of these complications and their management will then be discussed. As will be seen, the first sign is an abrupt change in end-tidal CO_2 tension (PETCO_2). The latter can, in general, be used as an estimate of arterial PCO_2 (PaCO_2). There are times when this does not hold true. The arterial to end-tidal PCO_2 difference may be increased in healthy and in ASA 3–4 patients during LUAS,¹ such that PETCO_2 may underestimate PaCO_2 . End-tidal PCO_2 sometimes exceeds PaCO_2 (reversed gradient),¹ thus overestimating PaCO_2 . Figure 1 is a graphic representation of published data showing these three possibilities.² We believe that an early, modest increase in minute volume to maintain PETCO_2 at a constant, albeit increased, level is helpful in immediately detecting abrupt alterations in PETCO_2 .

Subcutaneous emphysema and pneumothorax

Subcutaneous emphysema (SCE) is not a serious complication but it may be the harbinger of pneumothorax.

Subcutaneous emphysema

The clinical presentation, accompanying problems and the diagnosis that will be presented are based on the information available in eight case reports^{3–10} of SCE in 12 patients (Table II) and the preliminary results of a prospective study.¹¹

CLINICAL PRESENTATION

In most instances, subcutaneous emphysema will be evident at about 45 min after the start, although it may appear later or only at the end of surgery. There is no apparent age or sex predilection. That complication frequently occurs during surgical manipulation around the oesophagus.¹¹

The most consistent finding is a sudden and brisk in-

TABLE I Laparoscopic upper abdominal surgery

1 Cholecystectomy and biliary surgery
2 Vagotomy and pyloroplasty
3 Nissen fundoplication
4 Oesophagogastrrectomy
5 Splenectomy
6 Colon resection
7 Nephrectomy
8 Abdominal trauma

FIGURE 1 Arterial: end-tidal PCO_2 gradients during laparoscopic cholecystectomy. In most instances the Pa-PETCO_2 gradient is maintained at a level that the data points will be within the parallelogram shown in the figure. This lies between the zero and 8 mmHg difference lines. At times, the gradient may be increased or reversed (the two rectangles). Under these two conditions, PETCO_2 will not provide a reliable index of PaCO_2 . Data from reference 2.

crease in PETCO_2 and a very marked increase in PaCO_2 (as high as 100 mmHg),^{3,5} coincident with the appearance of subcutaneous emphysema in the upper body (face, neck and thorax). An increase in airway pressure (Paw) or decreased lung compliance indicate the presence of pneumothorax.^{4,6,8} This was found in most instances, but was of the tension type in only two. Bilateral pneumothoraces and pneumomediastinum also occurred (Table II). A brisk increase in PETCO_2 should be followed by an examination of the patient for SCE and auscultation of the chest for air-entry. Chest radiography is essential to verify the presence of pneumothorax, unless one is dealing with a tension pneumothorax, with its attendant haemodynamic effects, which requires immediate thoracentesis.

MECHANISM

The source of the subcutaneous emphysema during LUAS is either supradiaphragmatic (from direct injury)

TABLE II Subcutaneous emphysema: data table

Ref #	Age	Sex	Op	IAP mmHg	Onset (min)	Δ PETCO ₂	Site of emphysema	Other signs	Remarks	Pneumo-thorax
3	1	61	F	LC	20	30–45 To 69 mmHg	Face, chest followed by legs	No. Paw unchanged	No evident diaphragmatic wrent. PaCO ₂ 104 mmHg. Increased ventilation to control PaCO ₂	No
4	1	21	M	LC	5–10	45 +13 mmHg	Head, neck, chest	Compliance decreased		Bilateral
5	1	69	F	LC	13	15 32 to 85	Neck, face	No Δ Paw	Minute volume 16 L but PaCO ₂ 100 mmHg	No
6	3	28	F	LC	28	50 Initially	Neck and chest	Inc Paw	Reduced QRS voltage	No
		32	M	LC		End Modest	Scrotum, trunk		Pneumomediastinum	No
		46	F	LC		End Not mentioned	Chest, neck, face		Pneumomediastinum	Yes
7	1	55	F	LC	16	45 Increased	Neck, chest			Bilateral
8	1		M	LC	?	45 Increased	Face, neck, chest	Inc Paw		Yes
9	2	33		LF	?	Late ?	Neck, chest	Nil	Pneumomediastinum	
		32		LF	?	Late ?	Neck, chest	Nil	Surgical injury recognized	Yes
10	2		M	V	15	? Increased	Left neck		Pneumomediastinum	
			M	V	11	? Increased	Left neck		Thoracentesis: CO ₂ <10%	Yes
11	16			LF		13 Marked increase			PETCO ₂ stabilized by increasing minute ventilation by 33.3 (6)% vs 8.9 (5)% in the other 9 patients	

*Prospective study of 25 patients.

Abbreviations: M = male; F = female; LC = laparoscopic cholecystectomy; LF = laparoscopic fundoplication; V = vagotomy; IAP = intra-abdominal pressure; Δ = change; Paw = airway pressure.

or infradiaphragmatic. Carbon dioxide can pass through the diaphragmatic foramina into the mediastinum and from there to the subcutaneous tissue planes of the head and neck.^{3,10,11} Alternatively, the insufflated gas can track along a low resistance conduit from the trocar into the subcutaneous tissue planes driven by the insufflating pressure,^{3,5} particularly with defective or improperly placed^{3,12} Verres needles. The gas may also be introduced directly in the thorax following inadvertent perforation of the diaphragm.

PARTICULAR FEATURES

There is no clear-cut level of insufflating pressure above which subcutaneous emphysema is more likely and no report has mentioned the volume of CO₂ insufflated. In uncomplicated laparoscopic cholecystectomy (LC), 27 $\pm$ 2.5 L CO₂ may be insufflated during the first 30 min.² Thus, any insufflated volume greater than expected should arouse suspicion. The volume of CO₂ that the lungs receive during LC with SCE is twice as large as that during uncomplicated LC, with a markedly increased PETCO₂.¹³ During the repair of inguinal herniae by the extraperitoneal approach (intentional SCE), the volume of CO₂ delivered to the lungs and the PETCO₂ are much larger than that during uncomplicated LC.¹⁴

Subcutaneous emphysema during LC has been reported in 9/40 patients in one study¹³ and 2/10 in another report,¹⁵ and is more likely during lengthy procedures, in particular, fundoplication during which SCE occurred in 16/25 patients.¹¹ Subcutaneous emphysema can cause upper airway obstruction by compression.¹⁶ Thus, close observation of the patient for four to six hours is necessary. In general, the emphysema will resolve within a day or two, whereas the pneumoperitoneum may persist for a week after surgery.¹⁷

MANAGEMENT

The presence of SCE requires the following:

- 1 Ruling out the presence of pneumothorax.
- 2 Increased ventilation to maintain an acceptable PETCO₂ and/or PaCO₂. This has been achieved by increasing minute volume by 33.3 $\pm$ 6%.¹¹
- 3 Evaluation of the upper airway at the end of the procedure.
- 4 Reassurance of the patient.

Pneumothorax

The following presentation of the clinical picture, pertinent features and diagnosis is based on the information available in 20 reported cases^{7–10,18–24} (Table III).

TABLE III Pneumothorax: data table

Ref	#	Age	M/F	Op.	Side	Onset (min)	IAP (mmHg)	Clinical monitors						Features and remarks
								SCE	Paw	BP	SpO ₂	PETCO ₂	x-ray	
4	1	21	M	LC	Bilat	45'	?	Yes	?	No	No	Inc	Yes	Decreased compliance
6	1	55	F	LC	Bilat	45'	16	Yes	Inc	Dec	Dec	Inc	Yes	
8	1	?	M	LC	Rt	45'	?	Yes	Inc	?	Dec	Inc	Yes	
18	1	60	F	LC	Lt	Early	?	?	Inc	Dec	Dec	?	Yes	Gas bubbles from thoracentesis tube. Thoracentesis CO ₂ > 10% Thoracentesis 800 ml 100% CO ₂
19	1	50	F	LC	Rt	Early	?	?	Inc	?	Dec	Inc	Yes	
20	1	61	F	LC	Rt	50'	?	?	No	No	Dec	No	Yes	
21	1	53	F	LC	Rt	After	?	?			Dec		Yes	
10	1	33	M	LV	Lt	?	15	Yes	No	No	Dec	Inc	Yes	Thoracentesis CO ₂ >10%. No bubbles during oesophagoscopy
	1	32	M	LV	Lt	?	11	Yes	?	No	Dec	Inc	Yes	
9	1	33	M	LF	Lt	After	?					Yes		
	1	75	F	LF		After		Yes					Yes	Pneumomediastinum
22	1	?	M	LF	Lt	Early	?	?	Inc	?	Dec	?	Yes	
23	1	29	M	LF	Lt	30'	12	No	Inc				Yes	Compliance decreased by 30 to 40% in the two patients
	1	41	F	LF	Lt	20'	12	No	Inc				Yes	
24	6			LF	Lt:5	During	14	Yes	Inc	?	No	Inc	Yes	Dec C dyn. PEEP 5 cm H ₂ O curative. Tear
					Bilat:1						Dec PaO ₂		In 2	During dissection over Lt diaphragmatic crux

Abbreviations: M = male; F = female; LC = laparoscopic cholecystectomy; LF = laparoscopic fundoplication; V = vagotomy; IAP = intra-abdominal pressure; Δ = change; Paw = airway pressure.

CLINICAL PRESENTATION

This complication can occur at any time during the procedure^{3,7,8,10,18-24} but may be noted only at the end.^{9,21} As with SCE, there is no predilection for age or sex, and it may occur during any LUAS. Pneumothorax is more likely to occur on the right side during LC (with two reported bilateral pneumothoraces) while performing surgery around the oesophagus, it occurred mostly on the left side. In the vast majority, SCE was noted. The three most consistent signs are an abrupt and marked increase in (1) PETCO₂, (2) airway pressure (Paw) and a decrease in dynamic compliance accompanied by (3) decreased arterial desaturation (reduced SpO₂). In some cases^{7,18} mean blood pressure (MBP) was reduced, raising the strong possibility that in those two cases a tension pneumothorax probably occurred. There is little documentation of the insufflation pressure used. The clinical diagnosis must be confirmed by auscultation of the chest, and if possible, by an immediate radiological examination. Thoracentesis will provide immediate relief and further confirmation.

The clinical presentation of pneumothorax may be confused with that of bronchial intubation. In both, an increase in Paw and a decrease in SpO₂ occur. However, with bronchial intubation, PETCO₂ should not increase initially. Further, bronchial intubation usually occurs during changes in patient position and during insufflation, which causes a cephalad shift of the diaphragm.

The differentiation between pneumothorax and bronchial intubation is by considering:

- (a) timing: earlier in the procedure in the case of bronchial intubation
- (b) PETCO₂: no initial increase with bronchial intubation
- (c) repositioning the endotracheal tube will rapidly improve SpO₂ and Paw with bronchial intubation.

Figure 2 is an algorithm to aid in the differential diagnosis, and also summarizes the differences between SCE, bronchial intubation and pneumothorax.

MECHANISM

There are several suspected mechanisms of LUAS-induced pneumothorax. (a) Diaphragmatic defects are often invoked.^{3,4,7,18,19} Improper closure of the communications between the pleural and peritoneal cavities during gestation result in diaphragmatic defects.⁴ A pleuro-peritoneal communication has been demonstrated in patients with ascites.²⁵ It is quite probable that high abdominal pressures open these potential canals. Diaphragmatic blebs, which may rupture causing pneumothorax, have been seen.⁴ The insufflated gas can reach the pleural space through the vena caval orifice in the diaphragm.^{7,10,18,20} The falciform ligament divides superiorly into two layers and leaves a "bare area" from which, if the ligament is perforated, gas can enter the mediastinum.^{20,21} Two other routes are the result of sur-

FIGURE 2 Algorithm: acute ventilatory complications during LUAS.

gical manipulation. The tissue space surrounding the oesophagus is continuous superiorly with the mediastinum. As this tissue plane is opened, as for example during laparoscopic vagotomy, the insufflated gas can track up into the mediastinum and create a pneumomediastinum. Further, the mediastinal pleura might be ruptured directly during dissection resulting in a pneumothorax.^{9,17} A remotely possible route is retroperitoneal insufflation. Finally, during laparoscopic hernia repair, gas can track along the inguinal blood vessels via the retroperitoneal space and into the mediastinum.¹⁶

The increased intraabdominal pressure and the head up position have both been implicated as contributory factors.

MANAGEMENT

- 1 The discontinuation of insufflation and release of the pneumoperitoneum.
- 2 Thoracentesis
- 3 Hyperventilation to limit the increase in PCO₂, with the knowledge that excessive hyperventilation can be deleterious.

An alternative method of treatment recently advocated is the use of 5 cm H₂O positive end-expiratory pressure (PEEP).²⁴ The reasoning is that PEEP should re-expand the lung sufficiently to permit reasonable gas exchange. In the five patients in whom this was attempted, arterial blood gases improved and Paw decreased.²⁴ However, this approach may not be indicated in all cases.

SUMMARY

An abrupt and marked increase in PETCO₂ is the first sign of SCE and/or pneumothorax. But an increase in PETCO₂ may also reflect an increased CO₂ load or an

increased cardiac output.¹ A tension pneumothorax will cause simultaneous desaturation and hypotension. The appropriate measures for either complication are (1) discontinue the insufflation temporarily (2) relieve the pneumothorax by chest tube drainage (3) increase minute ventilation. The use of 5 cm H₂O PEEP may be considered.

Carbon dioxide embolism

Massive air embolism²⁶ has occurred during laser laparoscopic cholecystectomy with the classical signs: (a) precipitous reduction in PetCO₂, (b) auscultation of a "mill-wheel" murmur (c) hypotension and (d) desaturation. We are unaware of documented cases of haemodynamically important CO₂ embolism during LUAS. The information regarding CO₂ embolism that will be presented is from illustrative cases of CO₂ embolism during gynaecological laparoscopies, from animal work and from preliminary reports utilising transoesophageal echocardiography (TEE) in patients undergoing LUAS.

Clinical presentation

Two case reports noted an increase rather than a decrease in PETCO₂.^{27,28} In the first, the authors noted a sudden 10% increase in PETCO₂ shortly after the detection of a "mill-wheel" murmur.²⁷ An increase in PETCO₂, prior to the severe hypotension, was also noted in the second case report.²⁸ This presentation contrasts with the abrupt reduction of PETCO₂ followed by hypotension²⁹ and the detection of the murmur. However, the sequence of events following the reported increase in PETCO₂ in the first two case reports explains the reason for this difference. In the first case,²⁷ insufflation was immediately stopped and the pneumoperitoneum released. In the second,²⁸ very severe hypotension followed and further information on PETCO₂ was not provided. The patient had to undergo complete cardiopulmonary by-pass to vent the trapped CO₂, with full recovery. It is possible that the reported increase in PETCO₂ merely reflects the usual peritoneal uptake of CO₂ prior to the embolization. This explanation is supported by the increased "noise" detected by TEE during LUAS, as will be discussed later.

Mechanism

In considering the conditions necessary for CO₂ embolism during LUAS, two points should be kept in mind. The first is that CO₂ is very soluble in blood, which also has a huge buffering capacity. The median lethal dose of CO₂ gas embolism in dogs is 25 ml · kg⁻¹ but only 5 ml · kg⁻¹ in the case of air.³⁰ That difference is due to the high solubility of CO₂ in blood compared with that in air. The other is that central venous and pul-

monary artery pressures are markedly increased during laparoscopic surgery in the reverse Trendelenburg position.¹ The "driving" pressure for embolization is the difference between intraabdominal pressure and intracardiac pressure. That pressure difference will determine the volume of gas introduced into the circulation. The CO₂ must come either from massive transperitoneal uptake or by intravenous injection under pressure to form a CO₂ "lock." The effects of intravenously injected CO₂ on PETCO₂, mean pulmonary artery pressure (MPAP) and systemic pressures in the pig depend on the volume injected.³¹ Volumes greater than 0.1 ml · kg⁻¹ · min⁻¹, either by bolus injection or by infusion, caused a reduction in PETCO₂, an increase in MPAP and hypotension. Smaller volumes resulted in insignificant changes in PETCO₂. The volume of CO₂ required for detection by transoesophageal echocardiography was 0.26 ± 0.24 ml · kg⁻¹ while that required to change PETCO₂ was 0.66 ± 0.51 ml · kg⁻¹. The response is thus determined by the volume of CO₂ injected. Another study examined the effects of various intraabdominal pressures during CO₂ insufflation on the occurrence of embolism with the vascular uptake of the gas occurring through a 5 mm cut in an iliac vein, thus more closely mimicking the clinical circumstance.³² At intraabdominal pressures <10 cm H₂O, only bleeding was seen through the laparoscope. In the pressure range of 10 to 25 cm H₂O, the transoesophageal Doppler consistently detected gas bubbling. Pressures >25 cm H₂O caused collapse of the veins and cessation of bleeding and gas bubbling. Thus, the difference between intraabdominal and intravascular pressure determines whether CO₂ embolism and/or haemorrhage will occur.

Transoesophageal echocardiographic monitoring

Transoesophageal echocardiography is considered the "gold standard" for the detection of emboli.³¹ Pulmonary gas emboli were detected by TEE in 10 of 15 patients undergoing laparoscopic cholecystectomy.³³ In five, the emboli occurred during insufflation and in the others during dissection, suggesting that small CO₂ emboli can occur either by transperitoneal absorption or by uptake via cut vessels. However, PETCO₂, blood pressure, heart rate and saturation were not altered during the episodes of CO₂ embolism. Haemodynamically inconsequential, small echogenic dots travelling at high speed, suggestive of pulmonary embolism, were noted in all patients in a similar prospective study of patients undergoing laparoscopic cholecystectomy.³⁴ No changes in PETCO₂ occurred.

Management

- 1 The patient must be placed immediately in the left lat-

eral position. This will relieve the mechanical obstruction of the gas "lock" to blood flow through the right ventricular outflow tract. The change in the position places the outflow tract below the right atrium.

- 2 Insufflation must be discontinued and the pneumoperitoneum released.
- 3 Full cardio-pulmonary support must be instituted.
- 4 Aspiration of gas by large bore catheters in the right heart and pulmonary artery may be of some value.
- 5 Complete cardiopulmonary by-pass may be required to evacuate the gas "lock"

Summary

Small CO₂ emboli occur during LUAS and can be detected by TEE. Such microemboli may increase PETCO₂. Macroemboli, sufficient to cause a gas "lock" and to decrease PETCO₂ and hypotension are also possible. Animal studies suggest that the volume of gas injected and pressure of injection are important. We should be aware that fatal CO₂ embolism can occur following the laparoscopy.³⁵

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References

- 1 Wahba RWM, Béique F, Kleiman SJ. Cardiopulmonary function and laparoscopic cholecystectomy. *Can J Anaesth* 1995; 42: 51–63.
- 2 Wahba RWM, Mamazza J. Ventilatory requirements during laparoscopic cholecystectomy. *Can J Anaesth* 1993; 40: 206–10.
- 3 Kent III RB. Subcutaneous emphysema and hypercarbia following laparoscopic cholecystectomy. *Arch Surg* 1991; 126: 1154–6.
- 4 Prystowsky JB, Jericho BG, Epstein HM. Spontaneous bilateral pneumothorax – complication of laparoscopic cholecystectomy. *Surgery* 1993; 114: 988–92.
- 5 Pearce DJ. Respiratory acidosis and subcutaneous emphysema during laparoscopic cholecystectomy. *Can J Anaesth* 1994; 41: 314–6.
- 6 Hasel R, Arora SK, Hickey DR. Intraoperative complications of laparoscopic cholecystectomy. *Can J Anaesth* 1993; 40: 459–64.
- 7 Woolner DF, Johnson DM. Bilateral pneumothorax and surgical emphysema associated with laparoscopic cholecystectomy. *Anaesth Intensive Care* 1993; 21: 108–10.
- 8 Massey SR, Balaji B. Surgical emphysema and laparoscopic surgery (Letter). *Anaesthesia* 1994; 49: 356–7.
- 9 Biswas TK, Smith JA. Laparoscopic total fundoplication:

- anaesthesia and complications (Letter). *Anaesth Intensive Care* 1993; 21: 127–8.
- 10 Chui PT, Gin T, Chung SCS. Subcutaneous emphysema, pneumomediastinum and pneumothorax complicating laparoscopic vagotomy. *Anaesthesia* 1993; 48: 978–81.
 - 11 Chiche JD, Joris J, Lamy M. Respiratory changes induced by subcutaneous emphysema during laparoscopic fundoplication. *Br J Anaesth* 1994; 72: A37.
 - 12 Bard PA, Chen L. Subcutaneous emphysema associated with laparoscopy (Letter). *Anesth Analg* 1990; 71: 101–2.
 - 13 Blobner MA, Felber AR, Gögler S, Weigl EM, Jelen-Esselborn S. Carbon dioxide uptake from pneumoperitoneum during laparoscopic cholecystectomy. *Anesthesiology* 1992; 77: A37.
 - 14 Mullett CE, Viale JP, Sagnard PE, et al. Pulmonary CO₂ elimination during surgical procedures using intra- or extraperitoneal CO₂ insufflation. *Anesth Analg* 1993; 76: 622–6.
 - 15 Wurst H, Schulte-Steiberg H, Finsterer U. Pulmonary CO₂-elimination in laparoscopic cholecystectomy. A clinical study (German). *Anaesthesist* 1993; 42: 427–34.
 - 16 Chien GL, Soifer BE. Pharyngeal emphysema with airway obstruction as a consequence of laparoscopic inguinal herniorrhaphy. *Anesth Analg* 1995; 80: 201–3.
 - 17 Millitz K, Moote DJ, Sparrow RK, Girotti MJ, Holliday RL, McLarty TD. Pneumoperitoneum after laparoscopic cholecystectomy: frequency and duration as seen on upright chest radiographs. *American Journal of Roentgenology* 1994; 163: 837–9.
 - 18 Seow LT, Khoo ST. Unilateral pneumothorax – an unexpected complication of laparoscopic cholecystectomy (Letter). *Can J Anaesth* 1993; 40: 1000–1.
 - 19 Whiston RJ, Eggers KA, Morris RW, Stamatakis JD. Tension pneumothorax during laparoscopic cholecystectomy (Letter). *Br J Surg* 1991; 78: 1325.
 - 20 Gabbott DA, Dunkley AB, Roberts FL. Carbon dioxide pneumothorax occurring during laparoscopic cholecystectomy. *Anaesthesia* 1992; 47: 587–8.
 - 21 Heddle RM, Platt AJ. Tension pneumothorax during laparoscopic cholecystectomy (Letter). *Br J Surg* 1992; 79: 374.
 - 22 Mangar D, Kirchoff GT, Leal JJ, Laborde R, Fu E. Pneumothorax during laparoscopic Nissen fundoplication. *Can J Anaesth* 1994; 41: 854–6.
 - 23 Mäkinen M-T, Yli-Hankala A, Kansanaho M. Early detection of CO₂ pneumothorax with continuous spirometry during laparoscopic fundoplication. *Acta Anaesthesiol Scand* 1995; 39: 411–3.
 - 24 Chiche JD, Joris J, Lamy M. PEEP for treatment of intra-operative pneumothorax during laparoscopic fundoplication. *Br J Anaesth* 1994; 72: A38.
 - 25 Verreault J, Lepage S, Bisson G, Plante A. Ascites and right pleural effusion: demonstration of a peritoneo-pleural communication. *J Nucl Med* 1986; 27: 1706–9.
 - 26 Greville AC, Clements EAF, Erwin DC, McMillan DL, Wellwood JMcK. Pulmonary air embolism during laparoscopic laser cholecystectomy. *Anaesthesia* 1991; 46: 113–4.
 - 27 Shulman D, Aronson HB. Capnography in the early diagnosis of carbon dioxide embolism during laparoscopy. *Can Anaesth Soc J* 1984; 31: 455–9.
 - 28 Diakun TA. Carbon dioxide embolism: successful resuscitation with cardiopulmonary bypass. *Anesthesiology* 1991; 74: 1151–3.
 - 29 Beck DH, McQuillan PJ. Fatal carbon dioxide embolism and severe haemorrhage during laparoscopic salpingectomy. *Br J Anaesth* 1994; 72: 243–5.
 - 30 Graff TD, Arbogast NR, Phillips OC, Harns LC, Frazier TM. Gas embolism: a comparative study of air and carbon dioxide as embolic agents in the systemic venous system. *Am J Obstet Gynecol* 1959; 78: 259–65.
 - 31 Couture P, Boudreault D, Derouin M, et al. Venous carbon dioxide embolism in pigs: an evaluation of end-tidal carbon dioxide, transesophageal echocardiography, pulmonary artery pressure, and precordial auscultation as monitoring modalities. *Anesth Analg* 1994; 79: 867–73.
 - 32 Gillart T, Bazin JE, Conio N, Rasson P, Schoeffler P. Visualization of venous injury during laparoscopy: assessment of pressure conditions promoting gas embolism. *Br J Anaesth* 1995; 74: A125.
 - 33 Derouin M, Boudreault D, Couture P, Girard D, Gravel D. Detection of CO₂ venous embolism during laparoscopic surgery. *Anesthesiology* 1994; 81: A560.
 - 34 Thio JM, Reichert CLA. Transesophageal echocardiographic assessment of venous carbon dioxide embolism during laparoscopic cholecystectomy. *Anesthesiology* 1994; 81: A112.
 - 35 Root B, Levy MN, Pollack S, Lubert M, Pathak K. Gas embolism death after laparoscopy delayed by “trapping” in portal circulation. *Anesth Analg* 1978; 57: 232–7.