Review Article

Cardiopulmonary function and laparoscopic cholecystectomy

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This review analyzes the literature dealing with cardiopulmonary function during and pulmonary function following laparoscopic cholecystectomy in order to describe the patterns of changes in these functions and the mechanisms involved as well as to identify areas of concern and lacunae in our knowledge. Information was obtained from a Medline literature search and the annual meeting supplements of Anesthesiology, Anesth Analg, Br J Anaesth, and Can J Anaesth. The principal findings were that changes in cardiovascular function due to the insufflation are characterized by an immediate decrease in cardiac index and an increase in mean arterial blood pressure and systemic vascular resistance. In the next few minutes there is partial restoration of cardiac index and resistance but blood pressure and heart rate do not change. The pattern is the result of the interaction between increased abdominal pressure, neurohumoral responses and absorbed CO_2. Pulmonary function changes are characterized by reduced compliance without large alterations in PaO_2, but tissue oxygenation can be adversely affected due to reduced O_2 delivery. A major difficulty in maintaining normocarbia is due to the abdominal distention reducing pulmonary compliance and to CO_2 absorption. End tidal CO_2 tension is not a reliable index of $PaCO_2$, particularly in ASA III–IV patients. The pattern of lung function following LC is characterized by a transient reduction in lung volumes and capacities with a restrictive breathing pattern and the loss of the abdominal contribution to breathing. Atelectasis also occurs. These changes are qualitatively similar to but of a lesser magnitude than those following "open" abdominal operations. It is concluded that the changes in cardiopulmonary function during laparoscopic upper abdominal surgery lead us to suggest judicious invasive monitoring and careful interpretation in ASA III–IV patients. Lung function following extensive procedures in sick patients has not been reported.

Ce survol analyse la littérature portant sur la fonction cardiopulmonaire pendant et la fonction pulmonaire après la cholécystectomie laparoscopique dans le but d'en décrire le profil des changements et les mécanismes engagés, et identifier les domaines d'intérêt particuliers et les lacunes de nos connaissances. Les renseignements ont été compilés à partir des données de Medline et des suppléments d'Anesthesiology, Anesth Analg, Br J Anaesth, et Can J Anaesth. Les répercussions cardio-vasculaires causées par l'insufflation sont caractérisées par une chute immédiate de l'index cardiaque et une augmentation de la pression artérielle moyenne et de la résistance vasculaire ont constitué les constatations les plus importantes. Dans les minutes qui suivent, l'index cardiaque et la résistance se rétablissent partiellement, mais la pression artérielle et la fréquence cardiaque ne changent pas. Cet état résulte de l'interaction entre l'augmentation de la pression abdominale, les réponses neuro-humorales et le CO_2 absorbé. Les changements de la fonction pulmonaire sont caractérisés par une baisse de la compliance sans modifications significatives de la PaO_2, mais l'oxygénation des tissus peut être désavantagée par la baisse du transport de l'O_2. Le maintien de la CO_2 téléexpiratoire représente un problème majeur à cause de la distension l'abdominale qui réduit la compliance pulmonaire et par la réabsorption du CO_2. Le CO_2 téléexpiratoire évalue mal la $PaCO_2$ spécialement chez les patients ASA III et IV. L'état de la fonction pulmonaire est caractérisé par une réduction transitoire des volumes et des capacités pulmonaires avec un profil restrictif et une perte de la contribution abdominale à la respiration. Il existe aussi de l'atélectasie. Ces changements sont identiques à ceux qui suivent la cholécystectomie traditionnelle mais à un moindre degré. Les répercussions sur la

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fonction cardio-pulmonaire pendant la chirurgie laparoscopique haute nous incitent à suggérer un monitorage effractif pertinent interprété avec perspicacité chez les patients ASA III et IV. On n'a pas fait d'observation sur la fonction pulmonaire de patients tarés après des interventions complexes.

Laparoscopy for diagnostic and operative purposes offers specific advantages to the patient. Apart from the aesthetic appeal, the shorter hospital stay and decreased postoperative morbidity are particularly strong arguments for laparoscopic surgery. This is reflected in the trend towards the laparoscopic approach for a wide variety of major abdominal operations in older and sicker patients. Currently, the most frequently performed laparoscopic upper abdominal operation is laparoscopic cholecystectomy (LC). A sufficient number of clinical studies of the physiological changes during and after LC has now been reported to warrant analytical examination. This review will examine these studies to describe the perioperative cardiopulmonary effects of LC as an example of laparoscopic upper abdominal surgery (LUAS), which is usually performed with patients in the reverse Trendelenburg position (rT). The discussion will describe the probable mechanisms involved in the sequential changes in cardiopulmonary function and identify lacunae in our knowledge. Finally, general and specific recommendations for the care of patients with pre-existing cardiac or lung disease will be made based upon the analysis.

Cardiovascular function

The pattern of change

One must clearly distinguish between the sequential haemodynamic effects of anaesthesia, positioning (usually 20° rT), mechanical and neuro-endocrine effects of the pneumoperitoneum and those of absorbed carbon dioxide (CO_2). Thus, studies reporting serial rather than single measurements are most informative. The data available in a number of articles and abstracts, in which CO_2 was used for insufflation, are summarized in Table I. The first four studies reported serial measurements of cardiac index (CI) in mostly healthy patients. The clearest picture of the pattern of haemodynamic changes is from a study reporting serial CI determinations in 15 healthy adults.[1] Induction of anaesthesia and positioning in rT prior to insufflation caused a 35 to 40% reduction in CI. A further reduction to 50% of awake values occurred during the initial phase of CO_2 insufflation in rT. Approximately five to ten minutes after the start of the insufflation (at a constant pressure of 14 mmHg), a gradual restoration of CI began. The biphasic pattern of an early reduction followed by partial recovery of CI was present in other studies of serial measurements[2,3] and is supported by studies reporting single measurements of CI during the creation of the pneumoperitoneum.[5-7]

The decrease in CI observed with CO_2 insufflation in the rT position might be avoided if CO_2 insufflation is performed in the supine position. One study reported no large changes in ejection fraction or heart rate during supine CO_2 insufflation. Left ventricular end systolic and end diastolic areas (LVESA, LVEDA), measured by transoesophageal echocardiography (TEE), were not altered during the insufflation.[4] Further investigations are needed to confirm these findings. Subsequent positioning in rT following insufflation resulted in reduced LVEDA, implying that stroke volume and thus CI (the values of which were not reported), must also have decreased, since heart rate and ejection fraction did not change.

The filling pressures of the heart (pulmonary artery occlusion (wedge) pressure (PAOP) and central venous pressure (CVP)), which are markedly reduced by the induction and by positioning in rT, increase when CO_2 insufflation starts.[1,5,8-10] The increase in filling pressures is not associated with an increase in LVEDA or LVESA as described above.[4] Most studies reported an increase in mean arterial blood pressure (MABP) during the insufflation.[1-7,9,10] The increase in MABP represents increased afterload[2] and is associated with an increase in left ventricular wall stress.[4] Calculated systemic vascular resistance (SVR) increases markedly,[1,5,9,10] particularly during the early phase of insufflation, with partial restoration starting 10 to 15 min after the creation of the pneumoperitoneum.

It is important to note that (a) the improvement in CI and the reduction in SVR occur simultaneously, and that (b) CI recovers in spite of an increased MABP. Some but not all authors report an increase in heart rate (HR) during the recovery phase.[1,3] The pattern of change in measured and calculated indices of cardiovascular function in healthy patients is graphically shown in the two left hand panels of Figure 1.

In patients with mild heart disease[8] or those with clinically severe systemic diseases (ASA III–IV),[9,10] the pattern of changes in CI, MABP and SVR is qualitatively very similar to that in healthy patients (Figure 1, right hand panels). The most striking features are the considerable initial reduction in CI occurring simultaneously with large increases in PAOP, MABP and SVR followed by a recovery of the CI as described in the healthy patients. Left ventricular stroke work index (LVSWI) is also substantially increased causing an increase in myocardial oxygen demand.[11] In one study of ASA III and IV patients mixed venous oxygen saturation ($S\bar{v}O_2$) increased during surgery in about 50% of these patients, but decrease in the rest.[10] In the latter group, the reduction in CI was of greater magnitude than in the former, with a higher MABP (after-load) and reduced oxygen delivery.

TABLE I Cardiovascular function during CO_2 pneumoperitoneum

Ref.	*Function examined*	*Investigative method*	*Sample details*	*Results and observations*
1	CI and MABP	Thermodilution	15 Healthy	Induction and head up tilt: −25%. A further reduction to 50% of awake values with the start of the insufflation, followed by a gradual restoration but no compensatory tachycardia. MABP increased.
2		Doppler pulse from oesophageal probe	21 Healthy; 4 with CAD	Variably reduced (average c−25%) with head up position and insufflation; partial restoration with time. MABP and heart rate increased.
3		Bio-impedance	10 ASA I–III, no CAD	Initially decreased ventricular preload and CI then gradual improvement by tachycardia. MABP increased.
4		Ejection fraction by TEE	13 Healthy	Insufflation in the supine position: no change in ejection fraction, heart rate or LVEDV; subsequent tilt: LVEDA decreased, thus stroke volume and CI must have decreased.
5		Bio-impedance	15 Healthy & 1 CABG	−30% with positioning and insufflation. MABP increased.
6		TEE pulse Doppler	18 Healthy	−24% with position and insufflation. MABP increased.
7		TEE pulse Doppler	16 Healthy	No change noted at peak $PaCO_2$ (43.7 mmHg). MABP increased.
8		Thermodilution	5 Cardiac patients	50% reduction with positioning and insufflation. Partial restoration with time. No BP change.
9		Thermodilution	9 ASA 3–4: no details	Initial reduction with positioning and insufflation, then normalization. BP increased.
10		Thermodilution	15 cardiac patients (ASA 3–4)	Marked reduction during the insufflation phase. BP increased.
1, 6	PAOP and CVP	Pulmonary a. catheter	Healthy	Initial 60% increase (1). Initial doubling and elevated throughout (6). Increase related to insufflation pressure (8).
8, 9, 10		Pulmonary a. catheter	Cardiac history or ASA I–IV	Initial increase during insufflation in both reports.
1, 5, 9, 10	Systemic vascular resistance	Calculated		Markedly increased at start of insufflation, then gradual, partial restoration.

CI: cardiac index; MABP: mean arterial blood pressure; CABG: coronary artery bypass graft; CAD: coronary artery disease; TEE: transoesophageal echocardiography; LVEDV: left ventricular end-diastolic volume; PAOP: pulmonary artery occluded pressure; CVP: central venous pressure: ASA: American Society of Anesthesiologists.

The direction and magnitude of the changes in $S\bar{v}O_2$ could not be predicted from the ASA classification. The reduction in CI could be corrected by volume loading in some instances.

The mechanisms

The initial reduction in CI is due to the effects of induction of general anaesthesia and of positioning. The direct myocardial depressant and the vasodilatatory effects of the anaesthetic together with the loss of sympathetic tone cause the initial reduction in CI and PAOP. This change is followed by the reduction in venous return (preload) due to positioning in rT causing a further decrease in CI and PAOP. The effects of abdominal distention with CO_2 are more complex and it is important to distinguish between the direct mechanical (compressive) effects, the neuro-humoral responses and those of absorbed CO_2.

INCREASED ABDOMINAL PRESSURE

The increased abdominal pressure will have a biphasic effect on venous return: an initial transient increase due to compression of the abdominal capacitance vessels followed by impedance of venous return from the abdomen (decreased preload) and the lower limbs. In fact, during abdominal CO_2 insufflation, the measured pressure in the femoral veins is increased with reduced flow velocity indicating decreased venous return from the lower extremities.[12] The cephalad shift of the diaphragm due to abdominal distention will increase pleural pressure, which will, in turn, be partially transmitted to the cardiac chambers causing an increase in cardiac filling pressures

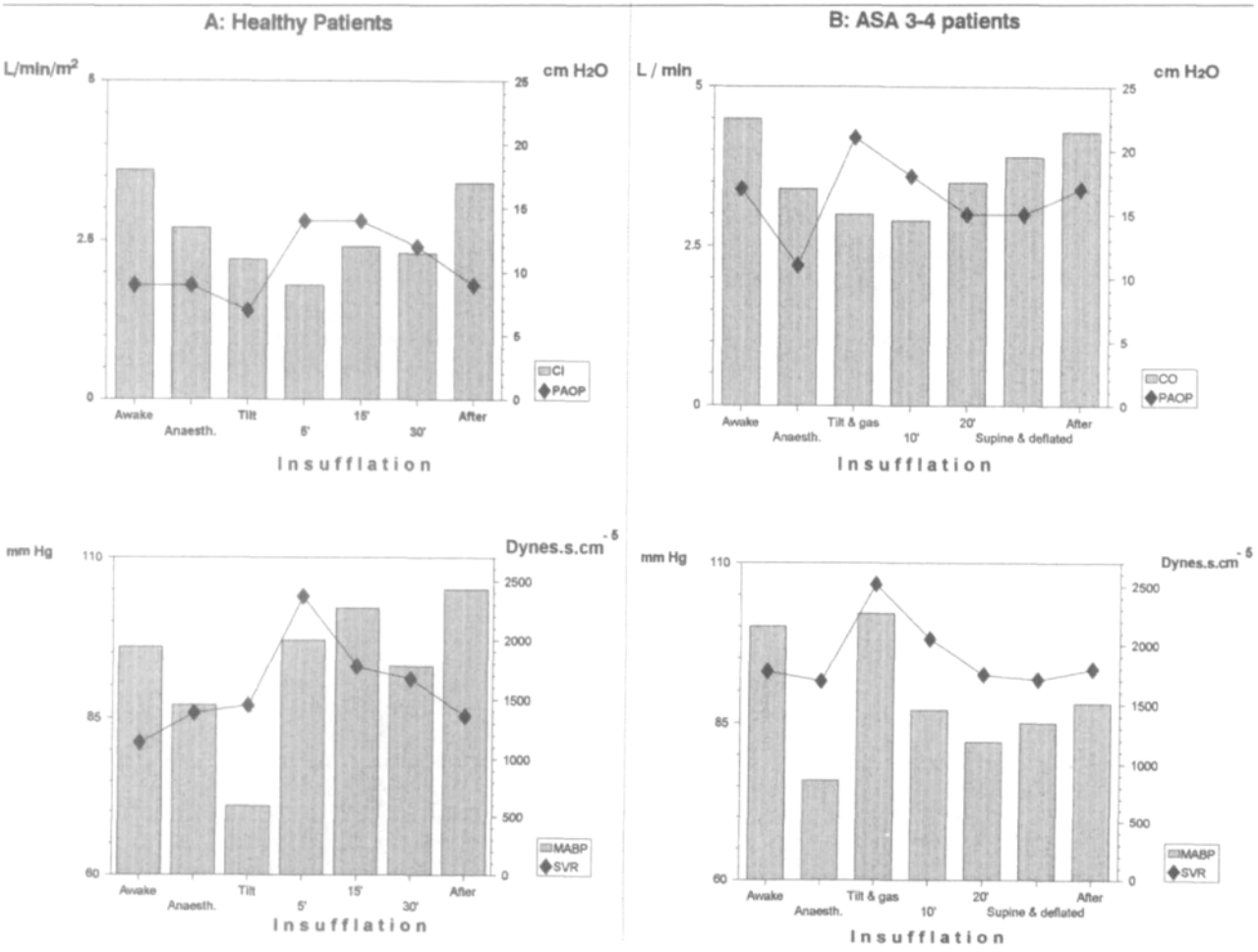

FIGURE 1 Cardiovascular function during CO_2 pneumoperitoneum. The mean values of CI (or CO), MABP, PAOP and SVR during the procedure are shown. Note the marked changes coincident with the start of insufflation and the subsequent partial restoration. Sources: healthy[1]; ASA III–IV.[9]

(CVP, PAOP). The increased PAOP and reduced CI do not represent cardiac dilatation since ventricular volumes measured by transoesophageal echography (TEE) in healthy patients[4] are not increased. However, in a preliminary report, oesophageal pressure, which is a good estimate of pleural pressure (Ppl), increased by 6 mmHg in healthy patients during the initial 15 min[13] with CO_2 insufflation at 14 mmHg pressure. There is no information available on the increase in Ppl or on ventricular volume change determined by TEE in ASA III–IV patients. In addition to its effect on venous return, the pneumoperitoneum will directly compress the abdominal arterial tree to a certain extent. This increase in afterload will adversely affect CI. The increased SVR during that phase supports this. As can be seen in Figure 1, the changes in CI are closely linked chronologically to the changes in SVR. This may be partially explained by the mathematical coupling which exists between these two measurements:

$$SVR = \frac{MABP - CVP}{CO} \times 80 \text{ dynes} \cdot \text{cm} \cdot \text{sec}^{-5}$$

However, one must invoke potent neuro-humoral responses to adequately explain CI recovery and changes in MABP and SVR that occur in all patients during prolonged CO_2 insufflation.

NEURO-HUMORAL RESPONSE

During LC, the release of catecholamines and other humoral factors increases shortly after the start of the pneumoperitoneum. The plasma concentration of dopamine,[14] vasopressin,[15,16] adrenaline,[16] noradrenaline,[14–16] renin,[16] and cortisol[16,17] increases considerably. Of particular importance is the time course of vasopressin and norepinephrine. The plasma concentration – time course profile parallels that of the changes in CI, MABP, and SVR,[14–16] suggesting a probable cause – effect relationship. Recovery of CI occurs in spite of the negative in-

otropic effect of increased inspired concentration of inhalational agents during this period.[1] Three of the reports[14-16] are preliminary and do not have control (open cholecystectomy) groups for adequate comparison, but the information contributes to our understanding. The 24 hr urinary excretion of vanyillylmandelic acid (VMA) following open cholecystectomy was 1.5 times that following laparoscopic cholecystectomy.[17]

ABSORBED CO_2

During prolonged CO_2 insufflation, considerable systemic absorption occurs and hypercarbia probably plays an important role in the recovery of CI and in minimizing the adverse haemodynamic effects of increased intra-abdominal pressure. The pulmonary elimination of carbon dioxide during LC is biphasic. The pattern is characterized by an initial brisk increase in CO_2 elimination (27–37 ml^{-min},[18] 30%[19]) which starts shortly after insufflation begins, followed by a less brisk rate. The initial steep increase reflects the initial rapid peritoneal absorption of CO_2 followed by reduced absorption due to the stretch of the peritoneal surface with compression of the peritoneal vessels.[18,19] Pulmonary elimination of CO_2 with increasing insufflation pressures in an experimental model was also biphasic.[20]

Intentional hypercarbia increases cardiac output,[21,22] mean arterial blood pressure, plasma epinephrine (E) and norepinephrine (NE) concentrations.[21] The SVR decreases,[21,22] reflecting the direct vasodilatatory effects of CO_2 when that is not countered by activation of the sympathetic nervous system with vasoconstriction of venous capacitance vessels. The net effect of the pneumoperitoneum and hypercarbia during LC will usually include increases in SVR, CO, CVP[21] and PAOP.[22] Thus, the alterations in cardiovascular dynamics during LC are probably influenced by increased CO_2, either directly (vasodilatation) or indirectly by stimulating the sympathetic nervous system.

A distinct difference in the haemodynamic effects of CO_2 and N_2O insufflation at graded pressures (10, 15 and 10 mmHg) performed with the patients lying supine has been reported.[23] Nitrous oxide caused a slightly larger reduction in CI (30 vs 23%). The magnitude with both gases was directly related to the insufflation pressure. The most marked difference between the two gases was the effect on MABP. Hypotension only occurred with N_2O. After positioning, CI and MABP were considerably less with N_2O. Some of the observed differences between the two groups may be attributable to the effects of absorbed CO_2, but the issue is confounded by the anaesthetic effects of absorbed N_2O. The results of that study do not support the choice of N_2O as the insufflating gas. One study examined the use of helium as an alternate gas.[24] Serial measurements of $PaCO_2$ during helium pneumoperitoneum were markedly less than those during CO_2 insufflation (37 ± 1 vs 50 ± 3 mmHg).[24] Cardiac output may be higher during CO_2 pneumoperitoneum (5.6 vs 4.5 L^{-min}) but the observed difference was not statistically significant. The data presented suggest that CO_2 probably plays a role in the restoration phase.

TABLE II Cardiopulmonary complications

Intraoperative
Bradycardia
Myocardial infarction
Postoperative
Cardiac
- Arrhythmias
- Ischaemia
- Infarction
Circulatory
- stroke
- Aortofemoral occlusion
- Deep vein thrombosis
Pulmonary
- Atelectasis
- Pneumonia
- Ventilatory arrest
- Pulmonary embolism
- Pneumothorax

Clinical implications

The reported cardiopulmonary complications during and after laparoscopic cholecystectomy are listed in Table II. Overall, this procedure is usually well tolerated with few perioperative complications. The deleterious effects of positioning in rT and the creation of a CO_2 pneumoperitoneum are well tolerated in healthy patients but can lead to serious morbidity and mortality in patients with limited cardiopulmonary reserve. A thorough understanding of the adverse physiological effects of laparoscopic cholecystectomy is, therefore, required.

Considerable CO_2 absorption can occur during laparoscopic cholecystectomy. The absorbed CO_2 can have deleterious effects in patients with preexisting cerebrovascular, pulmonary and cardiac diseases. Hypercarbia is a major concern in patients with increased intracranial pressure. During LC in healthy patients, the blood flow velocity through the middle cerebral artery (an index of CBF) can increase by as much as 50%, probably due to the increase in $PaCO_2$.[25] Hypercarbia can also cause cardiac arrhythmias and direct vasoconstriction of pulmonary vessels. One can speculate that in patients with preexisting pulmonary hypertension or right ventricular ischaemia/infarction, pulmonary vasoconstriction may result in right ventricular failure. This complication has not been reported.

TABLE III Factors increasing PAOP measurements during laparoscopic cholecystectomy

Cause of increased PAOP	*Mechanism*	*Comments*
Myocardial dysfunction	Myocardial ischaemia	Most episodes of ischaemia are *not* detected by the PAC
	Increased myocardial wall tension during CO_2 insufflation	
Increased venous return	Volume overload	Poor correlation between ventricular filling volume and PAOP in patients with coronary artery disease or with ejection fraction <50%
	Vasoconstriction with light anaesthesia	
	Hypercarbia-induced contraction of venous capacitance vessels	
Increased pleural pressure	Increased abdominal pressure with insufflation	
Increased transmission of pleural pressure	Altered lung compliance	e.g., pulmonary oedema

PAC: pulmonary artery catheter.

The surgical position and the compression of the inferior vena cava during LC impede venous return. The resulting venous stasis increases the risk of deep vein thrombosis and pulmonary embolism.

Patients with cardiovascular disease are at risk of developing myocardial ischaemia during LC due to the increase in myocardial wall tension caused by the increased MABP and SVR. The problem will be accentuated if tachycardia also occurs. The decrease in CI observed in ASA III–IV patients can result in insufficient tissue O_2 delivery and a reduction in mixed venous oxygen saturation.[10] One must then consider proceeding to an open cholecystectomy or adding inotropic agents.[27]

The clinical management of ASA III–IV cardiac patients should include continuous monitoring of ST segment changes and invasive haemodynamic monitoring. Computerized vectorcardiography (V-ECG) is a non-invasive, sensitive detector of ischaemic changes. The changes in the pattern during LC in healthy patients are similar to the changes noted during ischaemia.[28] These changes may, in effect, be due to alterations in the shape or position of the heart secondary to the cephalad displacement of the diaphragm during LC. At the moment, the most promising method is automated, two lead (II and V_5 or modified V_5) instantaneous ST segment analysis. Properly positioned TEE gives important information on regional wall motion abnormalities, filling volumes and ejection fractions.[4] However, the TEE probe is usually inserted during anaesthesia, and, therefore, can only compare values after the induction of general anaesthesia. Also, TEE is not available in all hospitals and requires particular expertise by a dedicated interpreter. Arterial cannulation will give beat-to-beat information on MABP and permit arterial blood gas sampling when required. The PAOP does not accurately reflect ventricular filling volume in these patients during CO_2 insufflation but measurements of oesophageal pressure may indicate the extent to which PAOP has been affected by increased intrapleural pressure. Moreover, an increase in PAOP or CVP during constant CO_2 insufflation pressure may be indicative of myocardial ischaemia or dysfunction. However, it is important to recognize the limitations of PAOP measurements.[29] Many other factors (Table III) will affect CVP and PAOP, including changes in the depth of anaesthesia which will be associated with vasodilatation or vasoconstriction. Changes in pulmonary compliance will also dictate the extent to which pleural pressure will be transmitted to the CVP and PAOP. Therefore, invasive monitoring by pulmonary artery catheterization will be most useful if serial $S\bar{v}O_2$ and/or CI monitoring are planned.[29]

Patients with coronary artery disease maintain satisfactory haemodynamic function with isoflurane and narcotic supplementation,[27] but may require vasoactive or inotropic drugs. The use of N_2O has been questioned[30] due to the possibility that it may cause bowel distention and hamper surgery though this does not occur in relatively short procedures. Adequate muscle relaxation is recommended to minimize abdominal CO_2 insufflation pressure.

Considering the adverse haemodynamic effects of LC, particular attention must be paid to the inflation pressure and the degree of tilt, especially in ASA III and IV patients. The magnitude of decrease in CI is directly related to insufflation pressure.[23] We also recommend slow and gradual abdominal insufflation to a maximum pressure of 10 mmHg followed by a limited 10° head up tilt which has been associated with cardiovascular stability in elderly ASA III–IV patients.[31] Considering that large amounts of CO_2 may be absorbed during LC, an increase in minute ventilation is required.

Intraoperative lung function and gas exchange

Laparoscopic upper abdominal surgery is performed in the reverse Trendelenburg position. Therefore, the effects of anaesthesia, posture and insufflation will be considered separately. This section will deal with functional residual capacity (FRC), compliance (CTOT), oxygenation and CO_2 homeostasis.

During general anaesthesia, FRC and CTOT are re-

duced by about 20% with an increased airways resistance (Raw) determined by the magnitude of FRC reduction.[32] The latter is directly related to body build, and can be reduced by 50% in the obese.[32] Cephalad displacement of the diaphragm following induction of anaesthesia causes the reduction in FRC and the increase in ventilation to perfusion mismatching, which results in the increase in the alveolar-arterial oxygen tension difference ($P_A - aO_2$).[32] Placing the anaesthetized patient in the rT position will increase FRC and, presumably, compliance, but will not necessarily improve oxygenation.[33] This is probably due to the reduction in cardiac output secondary to the head-up tilt nullifying any beneficial effects of position on the distribution of ventilation. We can assume that the cephalad displacement of the diaphragm by the insufflated gas will reduce FRC and CTOT.

Functional residual capacity and compliance

There is no information available on the changes in FRC during LC, but there are preliminary reports of the changes in CTOT determined non-invasively by side-arm spirometry in healthy patients[34-37] and in patients with systemic diseases.[9] Insufflation with the patient supine immediately decreased CTOT by 43%[34] and no further change occurred upon positioning.[34] The distention will prevent the expected passive caudad displacement of the diaphragm that results from rT positioning.[33] When insufflation was performed with the patient already in rT, mean CTOT was reduced by 32%[35] to 48%.[36] The data indicate that insufflation alone leads to a marked reduction in compliance and that subsequent tilting does not further affect compliance.[34,37] No further change occurred with time.[35] The data do not indicate the extent to which lung and chest wall compliances are individually affected. In ASA III–IV patients, CTOT (determined after insufflation and tilting) was reduced by 40%.[9] Thus, the pattern is similar to that in healthy patients and FRC must also be adversely affected by the insufflation.

The initial sharp decrease in CTOT is due to the initial cephalad shift of the diaphragm caused by the insufflated gas and the lack of change thereafter reflects the balance between the addition and loss (uptake and escape) of gas during the procedure. Airway pressure (Paw) increases during insufflation in healthy[1,35] and in ASA III–IV patients.[9,38] The increase in Paw cannot be used to estimate alterations in compliance because the increase is also due to the augmentation in minute ventilation required to decrease $P_{ET}CO_2$. The "driving pressure" (the difference between airway and abdominal pressures (Paw – Pabd)), which increases from 8.7 (1.0) to 10.4 (1.1) cm H_2O during insufflation,[39] also cannot be used for the same reason. A reasonable guide to changes in CTOT is the plateau pressure (P plateau) at end-inspiration, particularly if the ventilator is equipped with an inspiratory hold. The increase in inspiratory plateau pressure ranged from 39% to 69% during insufflation.[36,1] Sudden changes in P plateau indicate the occurrence of serious complications such as pneumothorax[40] during surgery involving the diaphragm. Total compliance was reported to decrease abruptly by 16 to 33% during a pneumothorax.[40]

Oxygenation

Arterial oxygenation, which is influenced by changes in pulmonary and cardiovascular function, has not been extensively studied. During LC in healthy patients, PaO_2 was stable[1] in spite of the changes in compliance and cardiac output mentioned above. Oxygen delivery, however, was much less due to the decrease in CI, with lactic acidosis.[13] The values of PaO_2 in ASA class II–III patients before and during insufflation surprisingly were not analyzed.[41] However, marked mixed venous desaturation in ASA III–IV patients has been reported.[10]

CO_2 homeostasis

Carbon dioxide homeostasis is a major consideration during LC. Hypercarbia occurs because of CO_2 absorption and because compliance is reduced, which impedes adequate pulmonary gas exchange. During the initial 30 min, 27 ± 2.5 L CO_2 may be insufflated.[39] The delivery of CO_2 to the lungs can increase by 30% during the first 30 min,[19] increasing delivery from 166 ± 24 ml^{-min} to 202 ± 32 ml^{-min}.[42] Appropriate increases (12–16%) in minute volume in healthy patients could, in most instances, maintain $PaCO_2$ within acceptable limits,[1,39] but did not invariably normalize $PaCO_2$.[35,39] Even with normal $P_{ET}CO_2$ (39 ± 1 mmHg) in ASA III–IV patients, achieved by increasing minute volume from 5.5 ± 0.4 to 9.9 ± 0.9 L^{-min}, $PaCO_2$ was high (50 ± 1 mmHg).[38] The normal gradient of 3–5 mmHg between $PaCO_2$ and $P_{ET}CO_2$ was markedly increased during LC in ASA III–IV patients[35-38] (Figure 2). Factors which may influence $P_{ET}CO_2$ during LC are summarized in Table IV. Although under normal circumstances $P_{ET}CO_2$ can be used as an estimate of $PaCO_2$, during LC $P_{ET}CO_2$ is not a reliable index of $PaCO_2$ in healthy patients,[39] and is more inaccurate in ASA III–IV patients.[35,38] This marked divergence between $PaCO_2$ and $P_{ET}CO_2$ has been reported in patients undergoing major operations without an exogenous CO_2 load.[43] The increased gradient can be caused by reduced cardiac output, increased ventilation:perfusion mismatching, or both.

Occasionally, during the course of laparoscopic surgery, the gradient may be decreased or even negative,[39] in which case $P_{ET}CO_2$ overestimates $PaCO_2$. This can occur under several conditions. A time lag between incremental changes in tidal volume and the resulting

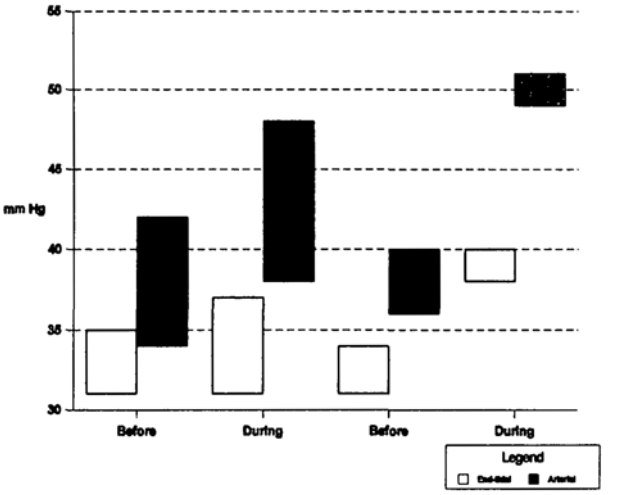

FIGURE 2 Pa and $PETCO_2$ during laparoscopy. The rectangles enclose the scatter (mean ± SD) of the two measurements before and during the insufflation. In both studies of ASA patients, the Pa-$PETCO_2$ increased during insufflation.[34,58]

TABLE IV Factors influencing $PETCO_2$ during laparoscopy

Increased $PETCO_2$	*Decreased $PETCO_2$*
Cardiovascular	
Increasing cardiac output	Decreasing cardiac output
	Hypovolaemia
	Pulmonary embolism
Ventilatory	
Hypoventilation	Hyperventilation
Bronchial intubation causing hypoventilation	
Carbon dioxide	
Increased delivery	
Small CO_2 venous embolis	Massive CO_2 under pressure

$PETCO_2$ decrement exists. Increasing tidal volume will open hitherto closed alveoli, whose CO_2 will now be "seen" as an increase in the slope of phase 3 of the capnograph and $PETCO_2$ will be close to or exceed the $PaCO_2$ line (Figure 3). A negative Pa-$PETCO_2$ occurs if FRC and compliance are reduced and airways resistance is increased markedly,[44] e.g., during Caesarean section under general anaesthesia,[44] or by an increase in CO_2 delivery. These pitfalls in $PETCO_2$ monitoring must be kept in mind during laparoscopic surgery. Thus, although serial determinations $PaCO_2$ in healthy patients may not be necessary, they are suggested when caring for sicker patients. Factors which indicate increased risk for hypercarbia with acidosis (pH < 7.35) during LC are low preoperative values of first second force expiratory volume (FEV_1) and vital capacity (VC), and high ASA

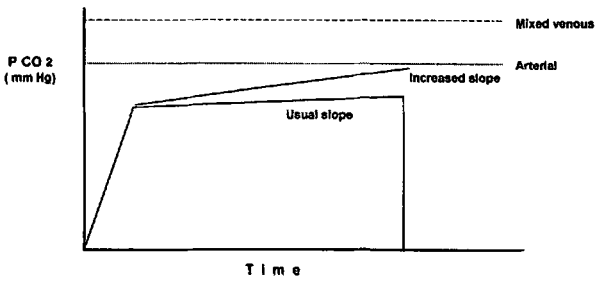

FIGURE 3 Expiratory capnograph. In most cases the usual slope will be observed. In some instances an increase in the slope of phase 3 can be noted and can approach or exceed the $PaCO_2$ line. For discussion, see text.

(III–IV scores).[45] Age alone does not predispose to respiratory acidosis during LC.[45] The concern about hypercarbia in ASA III–IV patients during CO_2 pneumoperitoneum has prompted a search for alternatives. In the porcine[46] and canine[47] animal models, $PaCO_2$ increased very considerably during CO_2 insufflation but not with helium. In humans, helium insufflation was not accompanied by increased $PaCO_2$ when compared with CO_2 insufflation (37 ± 1 vs 50 ± 3 mmHg).[25] The lack of hypercarbia during helium pneumoperitoneum suggests that absorption of CO_2 rather than the mechanical effects of the pneumoperitoneum is responsible. Although helium pneumoperitoneum may avoid hypercarbia, helium gas emboli would be catastrophic. Nitrous oxide pneumoperitoneum did not cause marked increases in $PETCO_2$[23] but N_2O supports combustion. Recently a mechanical device which lifts the abdominal wall, thus allowing gasless endoscopic surgery, has been described.[48]

In summary, maintaining normocarbia, particularly in ASA III–IV patients, may be difficult. Since CO_2 absorption depends on insufflating pressure, we recommend limiting the pressure to 10 mmHg and the rT tilt to 10°. Because $PETCO_2$ is a poor guide to $PaCO_2$ during LC in sick patients, arterial blood sampling is recommended. Monitoring compliance is desirable in patients with chronic airways disease and during surgery around the diaphragm.

Postoperative lung function

A major advantage of laparoscopic surgery is believed to be the avoidance of the pulmonary consequences of an abdominal incision. Lung function following open UAS is typically restrictive, with more rapid and shallower breathing, reduced (VC) and FRC with hypoxaemia.[32] A shift from abdominal to rib-cage breathing occurs and is due to the loss of diaphragmatic contribution to tidal volume.[32] There is also a considerable alteration in the pulmonary defense mechanism. The direction of muco-

TABLE V Postoperative FVC, FEV_1, and FRC

Ref.	*Function examined*	*Experimental design*	*Patient grouping*	*Results and observations*
50	FVC 7 FEV_1	Open vs closed	16 vs 20 Healthy	Open: −48%, closed: −27%* on day 1.
51		Historical comparison	48 Healthy adults	−21% on day 1.
52		Open vs closed	15 Healthy in each gp	Open: −40%, closed: −15%* on day 2.
53		Open vs closed	10 Healthy in each gp	Open: −72%, closed: −41%*
		Closed: epidural vs systemic analgesia	10 Healthy per group	(a) Systemic: −41%, epidural: −26% (n.s.) (b) Better VAS score in epidural group during 1st 8 hr.
17		Open vs closed	11 and 10 Healthy	Open: −1.71 L, closed: −0.7 L* on day 1.
54		"Preemptive" analgesia with 60 mg ketorolac on lung function	29 Healthy controls vs 31 healthy treatment group	Significant reductions in both, but less so in the treatment group in the immediate postoperative period. No difference on the following day.
55		Closed vs open	20 ASA 1–2 per group	Expiratory volumes decreased less in LC group*.
	$FEF_{25-75\%}$ atelectasis			70% of control with LC & 50% with open on 2nd day, More frequent and severe in open group.
56	(a) FVC, FEV_1 and PaO_2	Open vs closed	10 Healthy in each group	Significantly reduced in both groups. A more significant reduction in open group. Near normal lung function at 72 hrs in the closed group, with normal PaO_2 on day 1.
	(b) FRC			Transient, early reduction (−20%) in closed group. In open group, more marked (−34%) and longer lasting.
57	FVC, FRC and PaO_2	Closed vs historical	31 Reasonably healthy	FVC: −13%; PaO_2: −7 mmHg; FRC: −7% (sd = 17%) Risk factors: age, obesity, smoking.
58	FRC	Closed only	12 Healthy	−8%* at 24 hr.

*Statistically significant; (ns): not significant; sd = standard deviation.
FVC: forced vital capacity; FEV1: forced expiratory volume in 1 sec; $FEF_{25-75\%}$: forced expiratory flow during mid portion of VC; FRC: functional residual capacity; PaO_2: partial pressure of oxygen in arterial blood; VAS: visual analogue scale.

ciliary flow is reversed, particularly in the lung zones immediately above the operative site, with retention of secretions.[32] Two major factors determining the magnitude of these changes are incisional pain and reflex inhibition of diaphragmatic function due to afferent nociceptive signals from the chest wall and/or viscera. Adequate subjective pain relief by epidural analgesia brings about a small increase in FRC and a transient restoration of about 30% of the loss in VC.[49] The diaphragm and the rib cage function as a mechanical couple and their relative contributions to tidal breathing can be assessed by pressure measurements. That of the diaphragm can be assessed by measuring gastric pressure (Pga) and that of the rib cage by measuring pleural pressure (Ppl) during breathing. The alteration in diaphragmatic function depends on the extent of surgery. Following "open" abdominal surgery, Pga is maximally reduced for two days with gradual restoration over a week. During the entire period, Ppl does not change.[32] These changes parallel the changes in FRC. Direct stimulation of the phrenic nerves restores normal diaphragmatic function – supporting the belief that the dysfunction is reflex.[32] Thus, in examining lung function after LC, one must consider the changes in VC, FRC, oxygenation and analgesic requirements.

The studies reported so far have dealt with healthy patients. The earliest studies dealt with the changes in expiratory lung volumes and later reports dealt with FRC. The pertinent features of these studies[17,50–58] are summarized in Table V. Most have examined the reduction in expiratory lung volumes and leave no doubt that the magnitude of reduced VC and FEV_1 following LC, although quite marked (−20 to −40), was much less than that following "open" cholecystectomy (−40 to −70%). The reduction in VC and FEV_1 indicates a small diminution in the inspiratory function of the diaphragm. Reduced PaO_2 was minimal and transient.[56,57] Two aspects of two recent reports[56,57] are noteworthy. First, the return to normal function was noted to be twice as fast following LC (5 vs 10–12 days).[56] Secondly, the determination of VC and FEV_1 is effort-dependent whereas forced expiratory flow during 25–75% of the VC breath ($FEF_{25-75\%}$) is not, and is, therefore, a better measurement. The reduction in $FEF_{25-75\%}$ following open cholecystectomy (−50%) was almost double that after LC (−25%) on day 2.[57] Although the rate of recovery was more rapid in

the case of LC, the values were only 80% of control on the third postoperative day in healthy patients,[57] suggesting residual pulmonary dysfunction at 72 hr.

The FRC is normally reduced de novo shortly but not immediately after surgery.[32] On the day of surgery, a transient 20% reduction in FRC following LC was reported. This compares very favourably with the 34% reduction after open cholecystectomy.[56] Other authors reported a 7% reduction[57] with a wide scatter (17%). An important aspect of that report is that the more marked reductions in FRC occurred in the older and more obese patients and in smokers. Reduced FRC is associated with the development of atelectasis. In fact, patients who develop postoperative atelectasis or pneumonia also have larger reductions in FRC.[32] Two reports[55,57] deal specifically with the development of atelectasis following LC. Subradiological microatelectasis following surgery occurs normally, detectable only by computerized tomographic (CT) scanning, in 90% of patients one hour after surgery and can be found in 50% after 24 hr, without clinical evidence of atelectasis[59] or reduced FRC. Radiological atelectasis of varying degrees (micro to lobar) occurred in 40% of patients following LC, compared with 90% of patients after open cholecystectomy.[55] The incidence of the more clinically important local and segmental atelectasis was much less following LC.[55-57] Thus, a percentage (10–40%) of healthy patients will develop radiological segmental atelectasis after LC. The probability increases with age and obesity.[55] Atelectasis, per se, does not necessarily have clinical importance in healthy patients but it may be important in patients at risk for developing postoperative pneumonia.

The restrictive breathing pattern (rapid and shallow) following UAS is due partly to reflex inhibition and partly to incisional pain. Thus, avoiding the former by laparoscopic surgery is attractive. The question then becomes whether avoiding reflex inhibition can be achieved by LC. During the initial 90 min following LC, the diaphragmatic contribution to tidal volume was found to be unchanged.[60] However, three hours after surgery, breathing was more rapid and shallow with an increased $P_{ET}CO_2$.[61] The data are given graphically in Figure 4 which shows mean standardized tidal volume ($L \cdot m^{-2}$) and mean frequency ($Fb \cdot min^{-1}$) before and after LC with the 0.38 $L \cdot min^{-1} \cdot m^{-2}$ isopleth to indicate no change in minute volume. The range (mean ± scatter) of $P_{ET}CO_2$ is shown in the right hand panel. The relative contributions of the abdomen (diaphragm) and rib cage to tidal volume following LC (Figure 5) range with a marked reduction in diaphragmatic breathing, with gradual restoration.[62] The directly recorded diaphragmatic EMG also indicates diaphragmatic dysfunction in the early postoperative phase.[62] Clearly, the breathing pattern

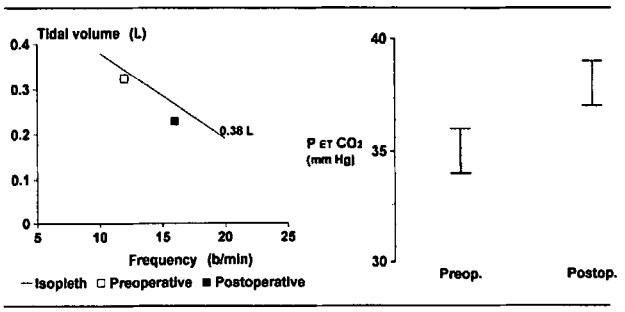

FIGURE 4 Breathing patterns and $PETCO_2$ after laparoscopy. Discussion in text.[61]

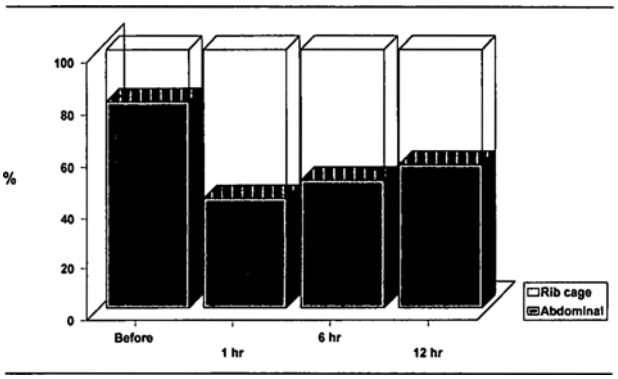

FIGURE 5 Partitioning of tidal breaths. Note the marked reduction in the abdominal (diaphragmatic) contribution to tidal breathing in the early phase, followed by gradual restoration.[60]

after LC is qualitatively similar to that after open cholecystectomy. Sixteen to eighteen hours after surgery, the abdominal contribution was still reduced with a slight elevation of the right hemi-diaphragm.[60] Equally important is the observation that, although the pressure developed by the diaphragm was 50% less (range −17 to −79%), the contractility is not affected. These data suggest that reflex inhibition does occur after laparoscopic UAS. We suspect that the degree of dysfunction will be greater following longer and more extensive operations and in patients at risk for developing postoperative pneumonia such as the elderly, the obese and those with chronic airway disease. Another advantage of the avoidance of a surgical incision is reduced analgesic requirements. This has been reported by several authors[17,54,55,63] but several points must be considered. Epidural analgesia provided adequate subjective pain relief but did not lead to improvement in lung function.[53] Preemptive analgesia with ketorolac *im* caused a transient improvement in VC in the immediate postoperative period only.[54] Patient-controlled analgesia (PCA) requirements were less after LC only in the immediate postoperative period.[64]

In summary, the data confirm the belief that LC leads to better postoperative lung function than OC. However,

the patterns of alteration are qualitatively similar but of a lesser degree following LC in healthy patients. Patients >50 yr recover more slowly than younger ones.[65]

General summary and conclusions

The rapidly increasing number of laparoscopic upper abdominal procedures being performed necessitates, on the part of the anaesthetist, a thorough understanding of the physiological changes that occur. Based on this understanding, s/he must design a system for monitoring and maintaining the well-being of sicker as well as healthy patients.

The cardiovascular changes follow a pattern of progressive depression of cardiac index and increase of SVR following induction of general anaesthesia, insufflation of the abdomen with CO_2 and institution of the rT position. Partial recovery occurs over the next 15–30 min. These changes may be of sufficient magnitude to affect adversely patients with pre-existing cardiovascular disease. We recommend careful attention to cardiovascular measurements in ASA III–IV patients undergoing these procedures. For these patients, invasive monitoring should be considered with the knowledge that CI and mixed venous oxygen saturation measurements will be more useful than measurements of filling pressures alone. In these sicker patients we also recommend limiting rT to 10° head up, and limiting insufflation pressure to 10 cm H_2O in order to attenuate the decrease in CI.

Intraoperative pulmonary changes are due to decreased pulmonary compliance secondary to upward movement of the diaphragm during insufflation, and to changes in CO_2 homeostasis secondary to absorption of insufflated CO_2 from the peritoneum. Measuring inspiratory plateau pressure and serial $PaCO_2$ in ASA III–IV patients is desirable. In the postoperative period, the pulmonary changes are qualitatively similar to, but less than those of "open" upper abdominal surgery, with restrictive breathing and decreases in VC and FRC. Atelectasis occurs in 10–40% of patients. There have been no prospective studies of changes in pulmonary function in ASA III–IV patients after laparoscopic upper abdominal surgery.

In conclusion, laparoscopic surgery of the upper abdomen has become a frequently performed procedure due to the perception that it is a "smaller" operation than "open" surgery, and that postoperative recovery is faster. Nevertheless, the procedure results in considerable alterations of intraoperative cardiopulmonary physiology.

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