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Pathophysiological and clinical aspects of the CO_2 pneumoperitoneum (CO_2-PP)

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Abstract. Experimental studies demonstrated a severe cardiac load of the CO_2 pneumoperitoneum caused by an accelerated after- and a decreased preload. Patients displaying cardiovascular risks are therefore often rejected from laparoscopic surgery. Hence, the pathophysiological changes and the intraoperative risk of the CO_2 pneumoperitoneum in high-risk cardiopulmonary patients (NYHA II–III, $n = 15$) undergoing laparoscopic cholecystectomy are described. The changes in cardiac after- and preload seem to be due to the elevated intraabdominal pressure rather than transperitoneally resorbed CO_2 and are reversible by desufflation. In one patient conversion to open operation had to be performed because of a severe drop in cardiac output and right ventricle ejection fraction. Mixed oxygen saturation was predicting intraoperative worsening in this case. The described pathophysiological changes may seem to be well tolerated even in high-risk cardiac patients. Monitoring of hemodynamics should include an arterial catheter line and blood gas analyses. Pharmacologic interventions or pressureless laparoscopic procedures might not be necessary as long as laparoscopic cholecystectomy is performed.

Key words: Laparoscopic surgery — CO_2 pneumoperitoneum — Pathophysiology — Intraoperative risk — Monitoring

The reduction in pain and pulmonary dysfunction achieved via laparoscopic surgery decreases the potential for postoperative (p.op.) complications and prolonged recovery. Patients displaying cardiovascular risks or patients undergoing lengthy operations should profit most from these benefits, since preexisting diseases are not exacerbated as after laparotomy. However, the scientific evaluation of the pathophysiological changes caused by CO_2 pneumoperitoneum (CO_2-PP) and elevated intraabdominal pressure (IAP) has lagged behind the clinical use of this technique. The intraoperative stress may be greater than under laparotomy since experimental studies have demonstrated severe changes in heart rate, systemic vascular resistance, cardiac output, and pulmonary function [5, 6, 16–18]. The increased intraabdominal pressure led to a reduction of venous reflux from the periphery and squeezed the venous reservoir within the abdominal cavity. Cardiac afterload parameters, such as the systemic vascular resistance, increased significantly. Establishing the pneumoperitoneum caused a 40% reduction in cardiac output, raising concerns about the clinical use of this method [1, 5].

However, only few clinical studies have been undertaken to evaluate the pathophysiological changes since an invasive monitoring is required to investigate the hemodynamic alterations observed in experimental studies. A reason for this might be that, so far, the majority of laparoscopic operations have been performed in young and healthy patients not allowing a substantial, invasive hemodynamic monitoring because of ethical and moral problems [1, 8, 9, 14]. Only a few studies have been done to evaluate the indications and perioperative risks of laparoscopic surgery in high-risk cardiac patients.

Despite the significant benefits, patients with cardiopulmonary risk are therefore often rejected from laparoscopic surgery. For further evaluation of the pathophysiology and the safety of this method we investigated in a prospective, clinical study the pathophysiological changes caused by CO_2 pneumoperitoneum that occur in patients with underlying heart and/or pulmonary diseases who undergo laparoscopic cholecystectomy using an intensive, invasive hemodynamic monitoring throughout the operative procedures.

Materials and methods

Fifteen patients with cardiopulmonary insufficiency stage II ($n = 9$) and stage III ($n = 6$), according to the classification of the New York Heart

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Table. 1. Patient characteristics[a]

Age	Sex	Operation time (min)	Cardiac disease	NYHA	Others
47	F	47	Aortic stenosis, P.myoc.inf.	II	
53	F	71	I.H.D., C.C.P.	III	Diabetes mellitus II
67	M	88	Mitral stenosis, heart block, artrial fibrillation	III	Peripheral vascular disease, diabetes mellitus II
56	F	56	Idio.h.suba.st., left ventricular hypertrophy	II	
75	F	91	I.H.D., stable angina	II	Hypertension
81	F	57	P.myoc.inf., artrial fibrillation	II	
83	M	64	P.myoc.inf., C.C.P.	II	
46	F	74	Tricuspidal insuff., P.myoc.inf.	III	Hypertension, diabetes mellitus II
73	F	58	I.H.D., P.myoc.inf.	II	
84	F	69	I.H.D., exertional angina	II	Hypertension
51	F	74	Cor pulmonale, stable angina	II	COLD
66	M	60	ACVB, heart block	III	Hypertension
78	M	61	I.H.D., C.C.P.	III	
63	F	68	Cor pulmonale, I.H.D.	III	COLD, diabetes mellitus II
78	M	88	P.myoc.inf., I.H.D.	II	

[a] P.myoc.inf., postmyocardial infarction; I.H.D., ischemic heart disease, C.C.P., congestive cardiomyopathy; Idio.h.suba.st., idiopathic hypertrophic subaortic stenosis.

Association, were scheduled for laparoscopic cholecystectomy and considered as candidates for the study. Four of these patients suffered from a valvular heart disease, eight an ischemic heart disease, and two from a cor pulmonale as a result of pulmonary hypertension. Informed consent was obtained from all participants. The characteristics of the patients and the duration of the operation are presented in Table 1. The mean duration of the CO_2-PP was 68 ± 12 min at an intraabdominal pressure (IAP) of 14 mmHg, the mean age of the patients 66.7 ± 14.3 years.

General anesthesia was induced with etomidate (0.2 mg/kg body weight i.v.), fentanyl (0.1 mg/kg body weight i.v.), succinylcholine (1–1.5 mg/kg body weight i.v.) and maintained with isoflurane (0.4–1 vol.%), N_2O/O_2 2:1, sufentanyl and vecuronium bromide (0.1 mg/kg body weight). Mechanical ventilation (Draeger Cicero, Draeger Co., FRG) was performed with the respiratory rate adjusted to achieve normal arterial (30–35 mmHg) and end-tidal pCO_2 values. The invasive hemodynamic monitoring included a pulmonary artery Swan-Ganz Thermodilution catheter (Model 744 H-7.5 F, Baxter Int. Coop., USA) and a radial arterial catheter line (1.0, Argyle Mediport 2, Sherwood Int. Co., USA) for measurement of central-venous pressure (CVP), pulmonary arterial wedge pressure (PAWP), cardiac output (CO), right ventricle ejection fraction (RVEF), and mean arterial blood pressure (MAP) using on-line hemodynamic monitoring (Sirecust 401-1 and 961, Siemens Corp., FRG). For an indirect measurement of the intrathoracal pressure changes, a catheter line was placed in the esophagus (IEP) at the level of the right ventricle (Sirecust 401-1, Siemens Corp., FRG). Calculated values like the systemic vascular resistance (SVR) were obtained from CVP, CO, and MAP. By subtracting the IEP from the CVP the transmural right atrial pressure (TMP) was obtained.

After induction of anesthesia hemodynamic and ventilatory parameters were normalized to achieve SVR of less than 1,800 dyn × s/cm^5, CO of more than 3.9 l/min, and PCWP between 8 and 12 mmHg. The abdominal insufflation of the CO_2-PP at an intraabdominal pressure of 14 mmHg was then performed. Directly before and 2 min after establishing CO_2-PP a set of data was obtained. Additional values under the pneumoperitoneum and after desufflation of it were obtained every 5 min. A final set of data were collected 90 min after desufflation of the abdomen.

In addition the end-tidal carbon dioxide concentrations ($VEXP_{CO2}$) were measured and the positive end-expiratory pressure (PEEP), peak inspiratory pressure (PEAK), as well as the inspiratory plateau phase (PLAT) and tidal volume (V_t) were protocolled by the respiratory system (Draeger Cicero, Draeger Co., FRG). Arterial blood samples were analyzed with a radiometer ABL2 blood-gas analyser which was calibrated every hour for measurement of arterial pCO_2 ($PaCO_2$) and O_2 (PaO_2).

Statistical analysis was performed with a two-way analysis of variance for repeated measures, and p values < 0.05 were regarded as significant.

Results

Significant hemodynamic but not ventilatory alterations were observed in all patients. Already at 2 min after establishing the CO_2 pneumoperitoneum we noticed a significant increase in the CVP from 15.5 ± 2.9 mmHg to 22.4 ± 3.4 mmHg. This was followed by a decrease to 18.4 mmHg ± 2.4 mmHg during the elevated intraabdominal pressure caused by the CO_2-PP. However, immediately after induction of the elevated IAP the intraesophageal (IEP) and the intrathoracal pressure increased even more strongly. As a result the TMP (Fig. 1), a calculated value (CVP-IEP) giving the actual information about precardial load, showed a constant decrease throughout CO_2-PP from 15.2 ± 3.4 mmMHg to 3 ± 2.8 mmHg (45 min CO_2-PP). Only after release of the pneumoperitoneum did these parameters of cardiac preload returned to baseline, preinsufflation values (15 min after desufflation).

The MAP increased initially (5 min CO_2-PP) after establishing the CO_2-PP from 86 ± 12 mmHg to 111 ± 18 mmHg. PAP also increased from 22.1 ± 4 to 32.2 ± 5 mmHg after 5 min. The MAP- and PAP-baseline values were reached before desufflation (45 min). Other afterload parameters such as SVR (Fig. 2) rose from 1,770 ± 224 to 2,415 ± 221 dyn × s/cm^5 immediately after induction of CO_2-PP raised the elevated IAP, before any reasonable amount of carbon dioxide was resorbed transperitoneally. These afterload parameters returned to almost normal values of 1,615 ± 241 dyn × s/cm^5 after about 60 min of CO_2-PP.

As a consequence of these changes in cardiac after- and preload, CO (Fig. 3) dropped after induction of CO_2-PP from 3.7 ± 0.6 to 2.8 ± 0.4 l/min. This was followed by an increase of up to 4.2 ± 0.6 (30 min CO_2-PP). After desufflation a peak increase of 5.04 ± 0.78 l/min occurred. The increase in CO was accompanied by an increase in heart rate. Cardiac stroke volume decreased constantly through-

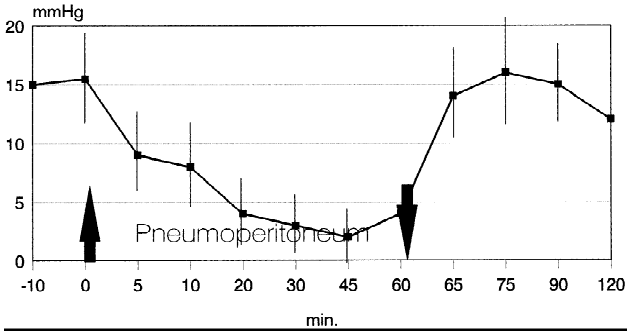

Fig. 1. Transmural right atrial pressure (TMP) in high-risk cardiac patients (NYHA II–III, $n = 15$) before (−10 min, 0 min), during (0–60 min), and after (60–120 min) CO_2-pneumoperitoneum at an intraabdominal pressure of 14 mmHg.

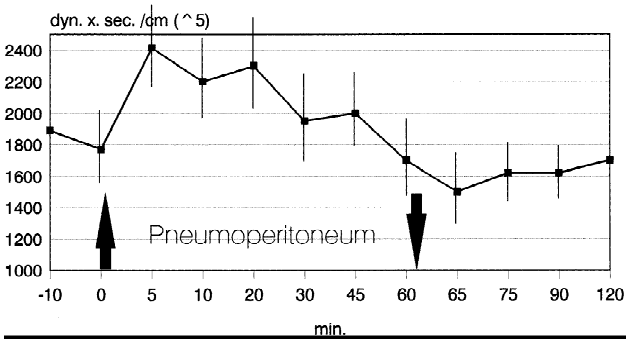

Fig. 2. Systemic vascular resistance in high-risk cardiac patients (NYHA II–III, $n = 15$) before (−10 min, 0 min), during (0–60 min), and after (60–120 min) CO_2 pneumoperitoneum at an intraabdominal pressure of 14 mmHg.

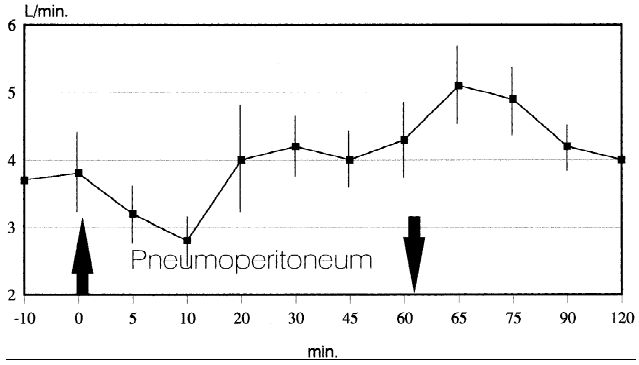

Fig. 3. Cardiac output in high-risk cardiac patients (NYHA II–III, $n = 15$) before (−10 min, 0 min), during (0–60 min), and after (60–120 min) CO_2 pneumoperitoneum at an intraabdominal pressure of 14 mmHg.

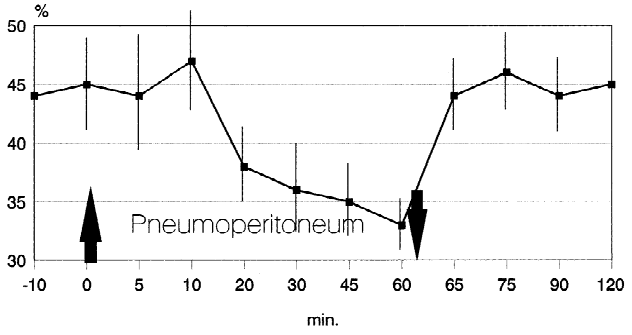

Fig. 4. Right ventricle ejection fraction in high-risk cardiac patients (NYHA II–III, $n = 15$) before (−10 min, 0 min), during (0–60 min), and after (60–120 min) CO_2 pneumoperitoneum at an intraabdominal pressure of 14 mmHg.

out the CO_2-PP. The decrease in cardiac pre- and the increase in afterload led to a constant drop of the right ventricle ejection fraction (RVEF) from 45 ± 3.4% to 33 ± 2.1% after 60 min of CO_2-PP (Fig. 4). In one patient the ejection fraction dropped to 23% after 32 min of CO_2-PP, demanding conversion to open operation. This patient began the operation with a low CO of 2.98 l/min and a high SVR of 2,254 dyn × s/cm^5. During the operation the mixed oxygen saturation decreased significantly, reflecting inadequate pulmonary perfusion. After desufflation these parameters returned to preinsufflation values within 4 min and the patient did well during the postoperative course.

$Vexp_{CO2}$ increased from preinsufflation values of 238 ± 14 ml/min to 298 ± 28 ml/min (30 min after insufflation) since minute ventilation was adapted to maintain normal $PaCO_2$ between 30 and 35 mmHg. PEEP values did not show any significant changes. After establishing the CO_2-PP PLAT values increased significantly from 20 ± 3.4 mbar to 27.5 ± 2.7 mbar and PEAK values from 26.8 ± 4.5 to 33.7 ± 2.4 mbar. The changes in PEAK values were not regarded as significant.

Discussion

Experimental pull-through manometries via V. femoralis up to V. cava superior under CO_2-PP in the sheep have already demonstrated a reduction zone in the elevated IAP beginning at the diaphragma along the basal parts of the thorax up to the level of the right atrium [5]. Only at that level the intravasal pressure within V. cava superior was like the IEP changes plus the CVP. The compression of the basal parts of the thorax might be responsible for the increase in PLAT and PEAK. This compression and the diaphragmatic elevation decrease pulmonar functional residual capacity, anatomical deadspace, and size of airways [5, 14]. They might also be responsible for the augmented pulmonar resistance under CO_2-PP. However, there seem to be no significant changes in PEAK, PEEP, PaO_2, and $PaCO_2$ during laparoscopic cholecystectomy as long as an adapted, controlled mechanical ventilation is performed.

The results of the pull-through manometries via V. femoralis superior to V. cava superior and the alterations in TMP (Fig. 2) indicate that CVP alone is not an appropriate indicator of cardiac preload, since the observed increase is caused by the elevated IAP carrying off into the basal parts of the thorax, altering CVP. As shown by the TMP, there is actually a constant decrease in cardiac preload during the pneumoperitoneum. This has to be kept in mind during laparoscopic procedures under CO_2-PP.

Other groups described a 40% reduction of the flow within intraabdominal V. cava and V. mesenterica as well as a stasis and dilatation within common V. femoralis [2, 8, 12]. These findings and the changes in TMP indicate a shift of blood volume from the center to the periphery with the elevated IAP reducing blood flow within V. cava abdominalis. This augments the decrease of cardiac preload and

might be, in contrast to the findings of Safran and Ho et al., one of the main causes of the observed changes in cardiac output [6, 16]. The venous stasis in the periphery might also promote venous thrombosis of the lower extremities. Intermittent sequential pneumatic compression, as suggested by Millard et al., reversed venous stasis within common femoral vein, returning peak velocity to normal [12]. This might be of special interest in preventing thrombosis in high-risk patients or in the case of longstanding laparoscopic operations.

The parameters of cardiac afterload, as there is the SVR and PAR, increased up to 35% also immediately after induction of the CO_2-PP before any reasonable amount of CO_2 was resorbed. During the time investigated, these parameters returned to baseline values 60 min after insufflation. The initial onset of hemodynamic alterations, the reverse effects after desufflation, and the results of the pull-through manometry as well as the TMP, indicate that the observed hemodynamic changes are caused by direct effects of the elevated IAP rather than systemic effects of transperitoneally resorbed CO_2 as suggested by Ho et al. [6]. Kashtan et al. and Leighton et al. described similar changes after hydroperitoneum, helium or nitrous oxide pneumoperitoneum in the dog, further suggesting that the elevated IAP is the main cause [7, 10]. The CO_2-PP, and the elevated IAP, also lead to an initial release of epinephrine, norepinephrine, and vasopressin as soon as 5 min after insufflation. The elevated catecholamine levels returned to baseline values only after desufflation [11, 13]. The observed changes in cardiac afterload, which are also described by Koksoy et al. [9], are therefore not only due to direct effects of the elevated IAP on the vascular system but also to increased catecholamine levels augmenting systemic vascular resistance.

The changes in cardiac pre- and afterload caused the observed alterations in CO (Fig. 3) and RVEF (Fig. 4). These might be critical for the organism, since cardiac wall tension and oxygen demand increase. Simultaneously, coronary perfusion is reduced because of a reduced stroke volume and shortened diastolic filling time during tachycardia. Hypovolemia, aggravating these hemodynamic changes, should therefore be avoided as long as CO_2-PP is used for laparoscopic procedures.

However, these data show that these changes are tolerated, and most of the patients with myocardial insufficiency respond well to CO_2-PP. Only in one patient did conversion to open operation have to be performed because of a severe drop in CO and RVEF. In this case a reduced mixed oxygen saturation and an increase in SVR was predicting intraoperative worsening. The effects were easily reversible by taking away the elevated IAP by removing the CO_2-PP.

Despite the described pathophysiological alterations of the CO_2-PP, laparoscopic surgery might be safely performed even in high-risk cardiac patients as long as careful perioperative monitoring and controlled mechanical ventilation are performed. The intraoperative monitoring should include an arterial catheter line, in-line capnography for $VEXP_{CO2}$, and a close look at the mixed oxygen saturation predicting intraoperative worsening. Safran et al. recommended the use of a pulmonary arterial catheter line [16]. We do not regard this as mandatory any more since the mixed oxygen saturation reflected inadequate perfusion and the increase in systemic vascular resistance as well as the drop in CO and RVEF. Extraperitoneal carbon dioxide insufflation, pharmacologic interventions, or so-called pressureless laparoscopic procedures, as described by Feigh and Chin et al., Rademaker et al. as well as Paolucci and Gutt, might not be necessary as long as laparoscopic cholecystectomy is performed [3, 4, 15, 18].

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