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A single-center observational study

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Luigi La Via*, Gaetano Palumbo**, Gloria Guccione**, Carlotta Continella***, Giulia Cirica***, Simona Tutino***, Tiziana Nicosia**, Marinella Astuto*/*, Filippo Sanfilippo****

**Department of Anesthesia and Intensive Care, A.O.U. "Policlinico-San Marco", Catania, Italy*
***School of Specialization in Anesthesia and Intensive Care, University of Catania, Catania, Italy*
****School of Specialization in Anesthesia and Intensive Care, University "Magna Graecia", Catanzaro, Italy*
*****Department of General Surgery and Medical-Surgical Specialties, Section of Anesthesia and Intensive Care, University of Catania, Catania, Italy*

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BACKGROUND AND OBJECTIVES: *The induction of pneumoperitoneum (PP) during laparoscopy may cause hemodynamic alterations, especially in patients with unknown cardiovascular diseases. While invasive arterial monitoring could be considered excessive, continuous noninvasive arterial pressure (CNAP) monitoring may allow careful evaluation of hemodynamic variations during laparoscopy.*
MATERIALS AND METHODS: *The objective of this single center observational study was to evaluate hemodynamic changes after insufflation and after deflation of PP with CNAP monitoring. Patients included where adults undergoing elective laparoscopic cholecystectomy (American Society of Anesthesiologists physical status classification II and III). The Hemodynamic data (blood-pressure, cardiac-index, heart-rate, stroke-volume index, stoke-volume variation and arterial-elastance) were collected 30 seconds before pneumoperitoneum (t_1), and compared to values at 2 (t_2), 10 (t_3) and 20 (t_4) minutes after pneumoperitoneum insufflation. We also compared data 30 seconds before and 2 minutes after release of pneumoperitoneum.*
RESULTS: *65 patients were included. Compared with reference values at t_1, blood-pressure values increased at all time-points (t_2-t_3-t_4); cardiac-index augmented at t_3 and t_4 ($p<0.05$); heart-rate increased at t_3 ($p<0.005$); stroke-volume index decreased at t_2 ($p<0.005$) and was higher at t_4 ($p<0.005$). While stoke-volume variation remained always stable after pneumoperitoneum induction, arterial-elastance increased significantly at all time-points (t_2-t_3-t_4). The only difference at pneumoperitoneum deflation was a reduction in stoke-volume variation ($p<0.05$).*
CONCLUSIONS: *In patients undergoing elective cholecystectomy, CNAP monitoring showed significant hemodynamic changes that would have been underappreciated with standard non-invasive monitoring with increase in arterial elastance under stable preload conditions. Whether this effect is due to unknown cardiovascular diseases facilitating ventriculo-arterial decoupling remains to be determined.*

KEY WORDS: Arterial Elastance, Cardiac Outp, Pneumoperitoneum, Stroke Volume, Stroke Volume Variation

Introduction

Nowadays, an increasing number of surgical procedures is performed under laparoscopy, with significant clinical advantages and a reduced hospital stay [1-4]. The induction of pneumoperitoneum (PP) during such procedures causes an increase of the intra-abdominal pressure (IAP) which in turn affects both cardiovascular dynamics and

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Correspondence to: Luigi La Via, Department of Anesthesia and Intensive Care, A.O.U. "Policlinico-San Marco", Via Santa Sofia 78, 95123 Catania, Italy, (e-mail: luigilavia7@gmail.com)

respiratory function. Patients with known cardiorespiratory pathologies may be at particular risk [5] and in these patients an invasive arterial monitoring can be considered.

Most of the studies describing the hemodynamic effects of PP with invasive cardiovascular monitoring [6-8] have shown a decrease in cardiac index (CI) despite a simultaneous increase in mean arterial pressure (MAP) values [8-14]; nonetheless, these findings have been challenged by other studies [15-17]. In this context it should be noted that most of these studies have been conducted over a decade ago with obsolete techniques and degrees of PP, including highly variable populations (from low- to high-risk patients) and studying hemodynamic changes with variable monitoring devices. On top of these differences, the study sample size was very restricted in most of these studies, therefore exposing them to the risk of being underpowered.

When cardiovascular disease has not been diagnosed, an invasive cardiovascular monitoring device may be considered disproportionate. However, one should keep in mind that the figures of undiagnosed left ventricular systolic dysfunction in the community is estimated between 6% and 9%[18-21], and moderate to severe diastolic dysfunction in over 7% [18,22-24]. Therefore, several patients may undergo surgery with the erroneous belief of a normal cardiac function and may be exposed to cardiovascular impairments at the induction of PP. In this context, continuous noninvasive arterial pressure (CNAP) monitoring may offer to the treating anesthesiologist the opportunity to carefully evaluate hemodynamic variations not fully appreciable by non-invasive blood pressure (NIBP) monitoring. Importantly, it is increasingly recognized the importance of even single episodes of hypotension on patient's outcome, both in the perioperative period and long-term [25-28].

The use of CNAP has been described in patients with cardiovascular comorbidities such as those undergoing carotid endarterectomy conducted either under general [29] or regional [30] anesthesia. Although not always providing inter-changeable hemodynamic values, CNAP have also shown good trending capabilities in the setting of laparoscopic surgery [31-33], and could be considered a good alternative monitoring approach in a selected population of patients [34].

We performed a single center observational study describing the hemodynamic changes produced by the insufflation and deflation of PP in a population of patients undergoing elective laparoscopic cholecystectomy. We had no prespecified hypothesis.

Materials and Methods

This single center observational study was conducted at Azienda Ospedaliero-Universitaria "Policlinico-Vittorio Emanuele", Catania ("G. Rodolico" site).

Ethical approval for the study (n. 137/2018/PO of the Ethical Committee registry) was provided by the Ethical Committee "Catania 1" of "Policlinico-Vittorio Emanuele" University Hospital, Via S. Sofia n. 78, 95123, Catania, Italy (Chairperson Prof. F. Drago) on 16th July 2018. The study is reported according to the checklist of the STROBE Statement [35].

We included adult patients (≥18 years old) undergoing elective laparoscopic cholecystectomy under general anesthesia and stratified according to the American Society of Anesthesiologists (ASA) classification as physical status II or III. We excluded cases of non-elective surgery, those with previous history of myocardial infarction or stroke, known atrial fibrillation or frequent ectopic beats, known peripheral vascular disease, and patients not consenting for the study.

Patients were monitored intra-operatively with ECG, SaO2 and non-invasive blood pressure (NIBP) monitoring cuff. In the contralateral arm to the NIBP monitoring, a 20- or 18-gauge cannula was inserted and the CNAP monitoring with *Clearsight®* (Edwards Lifescience, Irvine, CA) was applied at operating room entrance. Residents of the School of Anesthesia and Intensive Care of the University of Catania collected the following data: patient's demographics and comorbidities, ASA classification, and hemodynamic values provided by the CNAP monitoring collected at seven time-points (Table I). In particular, we collected heart rate (HR), systolic arterial pressure (SAP), diastolic arterial pressure (DAP), MAP, CI, stroke volume index (SVI), stroke volume variation (SVV). With the limitations of assessing the arterial elastance (Ea) with a CNAP according to the formula MAP/SV [36,37].

Induction of anesthesia was performed with propofol bolus (1.5-2 mg Kg^{-1} or with target controlled infusion [TCI] with plasmatic target 2-4 μg ml^{-1}), remifentanil (continuous infusion 0.1-0.5 μg Kg^{-1} min^{-1} or TCI target 2-4 ng ml^{-1}) and rocuronium (bolus 0.6 mg Kg^{-1}). After induction, maintenance of anesthesia was performed with propofol infusion (6-8 mg Kg^{-1} h^{-1} or TCI with target 2-4 μg ml^{-1}) or sevoflurane (target MAC 1.00), remifentanil (0.1-0.5 μg Kg^{-1} min^{-1} or TCI target 2-4 ng ml^{-1}) and rocuronium boluses (0.3 mg Kg^{-1} each 20 minutes). The PP was induced with a target

TABLE I - *Time points of data collection.*

Time point	
t0	Baseline
t1	30 seconds before PP
t2	2 minutes after PP
t3	10 minutes after PP
t4	20 minutes after PP
t5	30 seconds before release of PP
t6	2 minutes after release of PP

PP= Pneumoperitoneum

TABLE II - *Demographic and clinical patients' characteristics. Continuous variables are expressed as mean and standard deviation, categorical variables are reported as number and percentage.*

Variables	
Males	21/65 (33.3%)
Age (years)	52 ± 14
Height (cm)	165 ± 8
Weight (Kg)	71±15
Active smokers	23/65 (35.9%)
ASA class	
II	51/65 (78.5%)
III	14/65 (21.5%)
Known heart disease	17/65 (26%)
Known lung disease	4/65 (6.1%)

ASA= American Society of Anesthesiologists.

TABLE III - *Number of patients showing a negative variation (-) of at least 15% in cardiac index (CI) as compared with pre-insufflation of pneumoperitoneum (t1).*

	t_2 vs. t_1	t_3 vs. t_1	t_4 vs. t_1
- CI 15%	n=10/65 15%	n=6/65 9%	n=7/65 11%

t2 =2 minutes after pneumoperitoneum; t3 =10 minutes after pneumoperitoneum; t4 =20 minutes after pneumoperitoneum

IAP of 12 mmHg (range 10-13 mmHg). Patients were ventilated with a tidal volume of 8-10 ml/kg of ideal body weight and with a positive end-expiratory pressure of 5 cmH_2O. Attending anesthesiologists were aware of the study and, because of the observational design of the study, we left at their discretion to consider or not the results of CNAP monitoring during their anesthetic management, including fluid administration and use of vasoactive agents.

STATISTICAL ANALYSIS

We did not perform a formal sample size calculation, but considered to perform a larger study as compared to the ones available in the literature. Considering that the largest study included 48 patients and included a population with similar or rather lower anaesthesiological risk (American Society of Anesthesiologists physical status classification I-III) [38], we fixed our target to 70 patients, accounting for up to 15% of the population with incomplete data or with conversion to laparotomy, and thus excluded.

Statistical analyses were performed using IBM® SPSS® Statistics 17 for windows. The Kolmogorov-Smirnoff test was performed to test for the normality assumption of continuous variables. Continuous variables are presented as mean and standard deviation or median and 95% confidence interval (95% CI), and categorical variables as number and percentage (%). A two-sided t-student test for paired samples was performed to detected differences among couple of samples between time-points of interest. All tests were two-sided and a result of $p<0.05$ was considered statistically significant.

We also explored the subgroup of patients showing a drop in CI of at least 15% ($\Delta CI \leq 15\%$) before and 2 minutes after the induction of PP (t_2). Indeed changes of at least 15% are the most commonly adopted cut-off indicating a positive response to fluid administration in fluid-responsiveness studies, and CI is one of the most commonly referenced parameter in such studies [39,40]. Therefore, we assumed that a drop in CI of at least 15% following PP inflation could indicate a shift towards a fluid-responsiveness status (possibly due to variation in preload condition) and/or worsening myocardial performance[41] (especially in absence of changes in SVV). However, due to the observational nature of the study the true responsiveness to intravenous volume loading was not assessed and decision to administer intravenous fluids as always left to the treating anesthesiologist.

Results

We collected data on 70 consecutive patients undergoing elective laparoscopic cholecystectomy, and 5 were excluded due to missing critical information (n=3) or conversion to laparotomy (n=2). Demographic and clinical characteristics are described in Table II. The average duration of PP was 65 ± 27 minutes.

We report in Figure the trend of CI, SVI and HR (Fig. 1A, 1B, 1C respectively), SAP, MAP and DAP (Fig. 2), and SVV and Ea (Fig. 3A, 3B, respectively). The mean values of hemodynamic parameters at each time-point are reported as Supplementary Digital Content.

Several changes were seen in the periods subsequent to the insufflation of PP, while the only statistical significant difference seen at deflation of PP was a reduction in SVV. As compared with values before PP induction (t_1), CI increased 10 (t_3) and 20 (t_4) minutes after induction of PP. HR increased after PP induction but this change was statistically significant only at 10 minutes (t_3); the SVI decreased significantly 2 minutes after PP induction (t_2) and then gradually increased to higher values that reached statistical significance at 20 minutes (t_4). The SVV did not change after induction of PP; on the contrary, the Ea increased significantly at 2, 10 and 20 minutes after induction of PP with the highest values recorded at t_2.

Although on average the CI increased significantly 10 and 20 minutes after the induction of PP (t_3 and t_4 respectively), in our subgroup analysis we found a relatively large number of patients with a drop of CI (ΔCI)

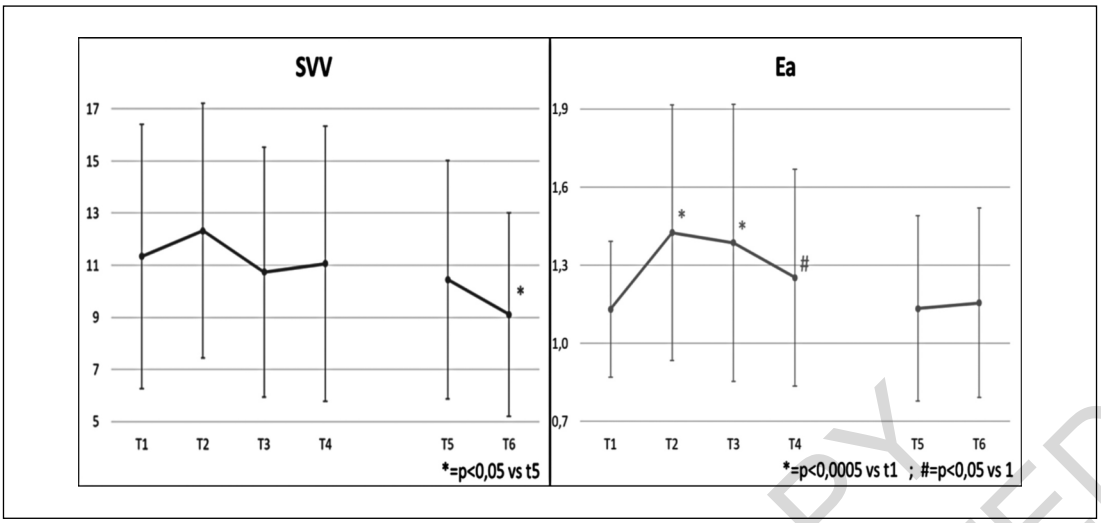

Fig. 1: Heart Rate (HR, Fig 1a), Cardiac Index (CI, Fig 1b) and Stroke Volume Index (SVI, Fig 1c) trend at time-points t1-t4 and t5-t6. The p values with statistically significant differences are shown on the right side of the figure.

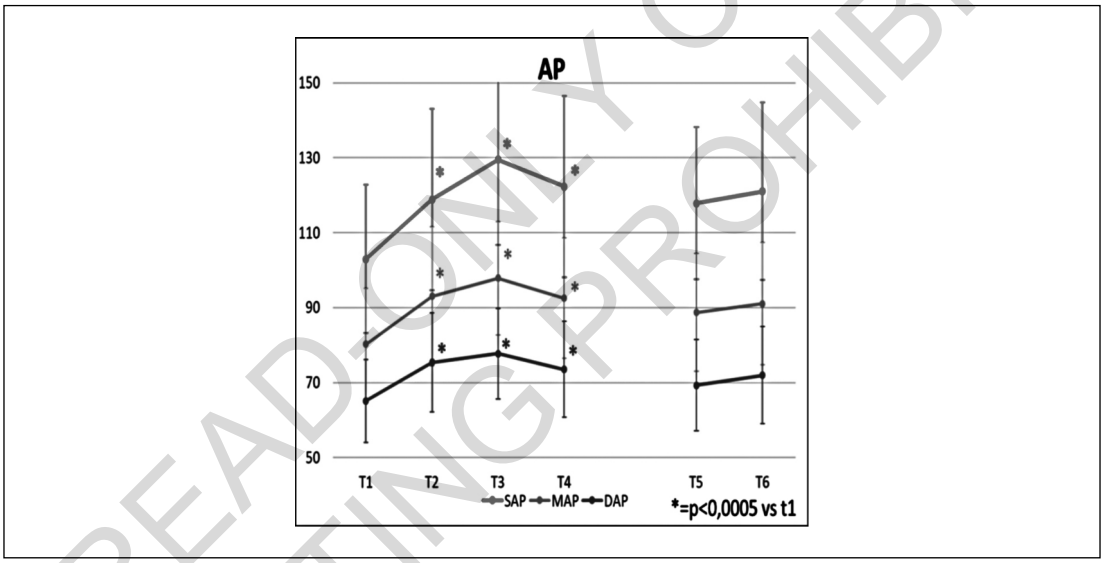

Fig. 2: Systolic Arterial Pressure (SAP), Mean Arterial Pressure (MAP) and Diastolic Arterial Pressure (DAP) trend at time-points t1-t4 and t5-t6. The p values with statistically significant differences are shown on the right side of the figure.

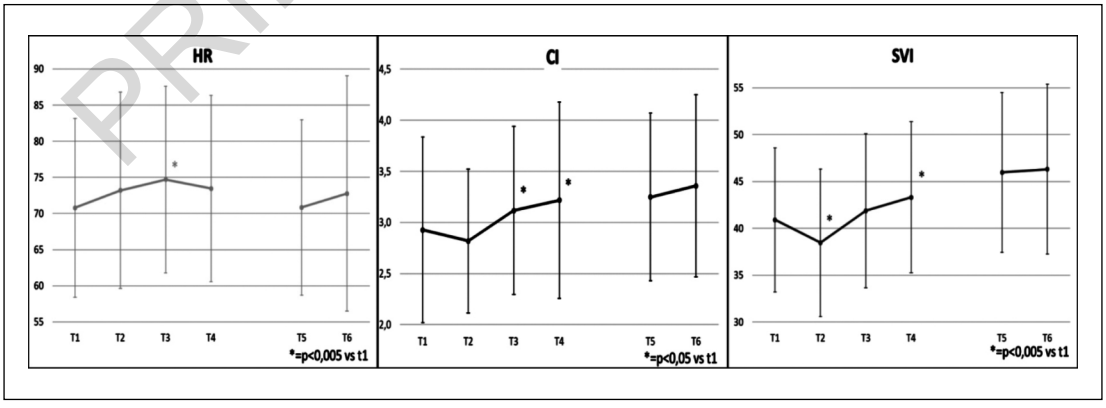

Fig. 3: Stroke volume variation (SVV, Fig 3a) and Arterial Elastance (Ea, Fig 3b) trend at time-points t1-t4 and t5-t6. The p values with statistically significant differences are shown on the right side of the figure.

≤15% measured 2 minutes after PP induction (n=10/65, 15%); (Table III).

In this subgroup of the patients with a ΔCI≤15% at t_2, the SVV remained within the same range of fluid-responsiveness (or unresponsiveness) in n=9/10 of cases, and only two patients showed an increased SVV at t_2 as compared with t_1 (with absolute increase of 6% and 7%). On the contrary, the Ea in patients with a ΔCI≤15% at t_2 showed higher absolute values as compared to the remaining patients (1.58 vs. 1.40).

Discussion

In the present study, we aimed at describing hemodynamic changes produced by the insufflation of PP with non-invasive assessment by CNAP in patients with ASA physical status II-III and without significant cardiovascular history. Since the hemodynamic impact of PP cannot be described only in terms of blood pressure, we evaluated changes in myocardial performance (CI and SVI) in relation to changes of preload (SVV) and afterload (blood pressure and Ea). Such evaluation was conducted in 65 patients undergoing elective cholecystectomy, and in a subgroup of patients showing a greater impact of PP inflation as demonstrated by a sudden decrease of CI≤15%. Importantly, in term of sample size, the number of patients enrolled in our study represents by far the largest one on the hemodynamic monitoring of patients undergoing laparoscopic cholecystectomy and the first one studying the PP effects with CNAP.

We found that CI remained stable shortly after induction of PP (t_2 - 2 minutes after induction) and eventually increased significantly after 10 and 20 minutes if compared with values before PP induction. The CI remained also stable at deflation of PP. The maintenance of stable CI seen on average in our study at the first time point after the induction of PP (2 minutes) seems the balance between a tendency towards decreased SVI and increased HR. On the other hand, we saw a progressive increase of SVI at t_3 and t_4 that contributed to the overall increased CI at these two time-points.

We reported a significant increase in blood pressure values, which is an already well-known effect of the induction of PP [8-13,15-17,38]. This increase of systemic vascular resistances is probably mediated by both mechanical and neuro-hormonal factors[42]. Catecholamines, renin-angiotensin system and vasopressin contributes to the increase in afterload [14,43] and possibly HR during PP induction. For instance, the mechanical stimulation of peritoneal receptors increases the release of vasopressin [44,45]. The trend of increased blood pressure during the induction of PP may lead to underestimation of changes in myocardial performance that are unapparent if patients are monitored only by NIBP. In this context, our study is the first one describing the hypertensive response after PP induction in terms of fluid responsiveness and Ea. The maintenance of similar values of SVV on one side and the increase of Ea support the idea of some impact of PP induction on worsening ventriculo-arterial coupling. It is important to note that Ea progressively returned to the values recorded before PP induction. While ventriculo-arterial uncoupling has been associated with worse prognosis in patients with septic shock [46,47], it is difficult to hypothesize that a transient increase of Ea after PP induction in relatively healthy patients correlates with prognosis. More studies are needed in this context and probably they should include patients with impaired cardiovascular reserve first.

Regarding the deflation of PP we found that only SVV changed significantly, but this change was far from being clinically meaningful since it decreased from 10.4% to 9.1%, thus remaining in the range of fluid unresponsiveness (good filling status, flat part of the Starling curve and patient unlikely to be fluid responsive). All the other hemodynamic parameters did not significantly change.

Another interesting aspect of our study is the finding in that a non-negligible amount of patients showed a decrease in CI≥15% after the induction of PNP. This drop appeared more frequent immediately after the induction of PP (15% of patients) rather than subsequently (t_3 or t_4, incidence around 10%). While the SVV did not change in most of these patients, the mean Ea at t_2 was higher in patients with sudden decrease of CI≤15% (1.58 vs. 1.40 in the remaining ones). Although it remains speculative, patients showing a sudden decrease of CI below 15% after PP induction may be those with under-appreciated cardiac comorbidities, and these figures are not far from the reported levels of systolic (6%-9%) dysfunction as well as moderate to severe diastolic impairment in the community [18,19].

Whether the drop in CI truly depends on the presence of under-recognized cardiovascular comorbidities cannot be determined by the present study. Nonetheless, the increase of Ea in absence of variation in the fluid-responsiveness status (stable SVV, and average values below 13% [48]) indicates that some degree of "ventriculo-arterial uncoupling" is probable and the heart suffers from inflation of PP, although the Ea gradually returns to pre-induction levels.

Most of the hemodynamic changes described in this observational study would have not been appreciated with standard NIBP monitoring, and we documented a non-trivial number of patients show worsening myocardial performance during stable preload conditions at PP induction.

Strengths and Limitations

Our investigation can be considered as a small pilot observational study describing hemodynamic changes evaluated via noninvasive finger cuff method. The main strength of our study is the enrollment of a large sam-

ple size in a homogeneous population of patients undergoing laparoscopic cholecystectomy with ASA risk II or III. Other studies investigating hemodynamic changes in patients undergoing laparoscopic cholecystectomy included a population ranging from 11 to 48 patients [13,38]. Moreover, such studies have been published between 10 and 20 years ago, and several of them evaluated hemodynamics changes with approaches that would not be justified nowadays, as the use of pulmonary artery catheter [10,11,15,17] or trans-esophageal echocardiography [16].
However, our study has several limitations. First of all, it is based on the assumption of under-recognized cardiac comorbidities and increased susceptibility to the hemodynamic effects of PP in some patients with ASA classification II and III.
We hypothesized that effects of PP induction could be very different according to myocardial function, which is difficult to assess without preoperative echocardiography screening. For this reason, we performed an exploratory subgroup analysis for patient with a clinically significant drop in CI as defined by a reduction of at least 15%. Secondly, as we performed an observational study, we did not couple the advanced CO monitoring evaluated via finger cuff method with values from an arterial cannulation, thus offering invasive hemodynamic monitoring.
This limitation does not allow calculating the accuracy and precision of the finger cuff method in this population of patients, although the trend ability of the method seems good as shown in other studies [30,31,49]. A third limitation is that our study was not blinded and the attending anesthesiologists may have integrated the information provided by the hemodynamic monitoring in their anaesthesiological management, thus possibly modulating hemodynamic changes. Indeed, the SVV on average was always below 13% and it was relatively stable, ranging between 10.7% and 12.3% after induction of PP, maintaining the patient in the flat part of the Starling curve.
On the other hand, this allowed the evaluation of the changes in afterload and ventriculo-arterial coupling in presence of relatively stable preload conditions.

Conclusions

In patients undergoing elective cholecystectomy we found that continuous noninvasive arterial pressure monitoring with finger cuff method demonstrated hemodynamic changes that would have been not appreciated with standard blood pressure monitoring.
Importantly, the induction of PP determined an increased arterial elastance in presence of stable preload conditions. This effect was more pronounced in patients with greater reduction of CI. Whether this effect is due to under-recognized cardiovascular diseases remains to be determined.

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Riassunto

Background ed Obiettivi: L'induzione dello pneumoperitoneo (PP) durante la laparoscopia può causare alterazioni emodinamiche, soprattutto in pazienti con patologie cardiovascolari non note.
Mentre il monitoraggio arterioso invasivo può essere considerato eccessivo, un monitoraggio continuo non invasivo della pressione arteriosa (Continuous noninvasive arterial pressure - CNAP) potrebbe permettere una attenta valutazione delle variazioni emodinamiche durante la laparoscopia.
Materiali e Metodi: L'obiettivo di questo studio osservazionale monocentrico è quello di valutare i cambiamenti emodinamici dopo insufflazione e dopo deflazione dello pneumoperitoneo con un monitoraggio arterioso continuo non invasivo. I pazienti inclusi sono soggetti adulti candidati ad intervento di colecistectomia laparoscopica in elezione (American Society of Anesthesiologists physical status classification II and III). I dati emodinamici (pressione arteriosa, gittata cardiaca indicizzata, frequenza cardiaca, gittata sistolica indicizzata, stroke-volume variation ed elastanza arteriosa) sono stati raccolti 30 secondi prima dello pneumoperitoneo (t1) e comparati con i valori a 2 (t2), 10 (t3) e 20 (t4) minuti dopo l'insufflazione dello pneumoperitoneo. Abbiamo inoltre comparato i dati 30 secondi prima e 2 minuti dopo deflazione dello pneumoperitoneo.
Risultati: Sono stati inclusi 65 pazienti. Rispetto ai valori di riferimento al tempo t1, i valori di pressione arteriosa sono aumentati in tutti i tempi (t2, t3, t4); la gittata cardiaca indicizzata è aumentata al tempo t3 e t4 ($p<0.05$); la frequenza cardiaca è aumentata al tempo t3 ($p<0.05$); la gittata sistolica indicizzata risulta ridotta al tempo t2 ($p<0.05$) ed aumentata al t4 ($p<0.05$). Mentre le variazioni dello della gittata sistolica sono rimaste per lo più stabili dopo l'induzione dello pneumoperitoneo, l'elastanza arteriosa è aumentata significativamente in tutte le misurazioni t2, t3, t4. L'unica differenza alla deflazione dello pneumoperitoneo è stata una riduzione dello stroke volume variation ($p<0.05$).
Conclusioni: In pazienti da sottoporre ad intervento elettivo di colecistectomia, il monitoraggio arterioso non invasivo ha mostrato significativi cambiamenti emodinamici che sarebbero stati sottostimati con un sistema di monitoraggio non invasivo standard con aumento nella elastanza arteriosa in condizioni di precarico stabile. Resta da determinare se questo effetto sia dovuto a malattie cardiovascolari sconosciute che facilitino il disaccoppiamento ventricolo-arterioso.

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