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Effect of prone position on hepato-splanchnic hemodynamics in acute lung injury

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Abstract *Objective:* To evaluate the effects of prone position on hepato-splanchnic hemodynamics, metabolism and gut mucosal energy balance. *Design:* Prospective clinical study. *Setting:* Medical intensive care unit in a university hospital. *Patients:* Eleven hemodynamically stable patients with acute lung injury (ALI) requiring mechanical ventilation. *Intervention:* Patients were studied in the supine position, after 90 min in the prone position and after 90 min of supine repositioning. *Measurements and results:* In addition to global hemodynamics we measured intra-abdominal pressure (IAP, bladder), hepato-splanchnic blood flow (HSBF, steady state indocyanine green technique using a hepatic vein catheter) and gastric mucosal-arterial PCO_2 gap (PCO_2 gap, automated air tonometry). Systemic hemodynamics did not change during the whole study. Prone positioning did not significantly affect IAP. HSBF as well as splanchnic oxygen consumption remained unaltered, too. Similarly, neither liver lactate uptake nor indocyanine green extraction were influenced by positional changes. Finally, stable regional hemodynamics were accompanied by an unchanged PCO_2 gap. *Conclusion:* We conclude that if IAP and systemic hemodynamics remain unaffected, the prone position in ALI patients compromises neither hepato-splanchnic perfusion nor gastric mucosal energy balance.

Keywords Splanchnic blood flow · Prone position · Intra-abdominal pressure · Gastric tonometry · Acute lung injury

Introduction

In the era of "lung protective strategies", turning mechanically ventilated patients from the supine to prone position is often used in the management of acute lung injury (ALI). The potential of prone ventilation in limiting or preventing ventilator-induced lung injury includes reduction of oxygen toxicity and inspiratory pressures due to decreased right-to-left shunt and increased blood flow to lung areas with a normal ventilation-perfusion relationship [1]. Although prone position is generally considered a safe intervention and improved gas exchange and lung function are primary goals, its use might theoretically be associated with relevant extrapulmonary side effects. Prone position could interfere with the circulatory functions due to a decrease in venous return as a result of increased intra-abdominal pressure (IAP), and decreased left ventricular volume in the prone position [2, 3]. By contrast, no significant alterations of indices of systemic hemodynamics (cardiac output, mean arterial pressure (MAP), pulmonary artery wedge pressure) were demonstrated during prone position in patients with ALI/ARDS [4, 5, 6].

It has to be stressed, however, that stable systemic hemodynamics do not implicate adequate regional blood flow. In addition, increased IAP, commonly seen in critically ill patients, might further be elevated during prone position [7, 8]. This, in turn, could have profound effects

Table 1 Patient characteristics (*M* male, *F* female, *BSA* body surface area, *ALI* acute lung injury, *APACHE II* APACHE II score on admission, *SOFA* SOFA score on the day of the study, *NE* norepinephrine)

Patient	Age (years)	Sex	BSA (m^2)	Cause of ALI	APACHE II	SOFA	Catecholamine ($\mu g\ kg^{-1}\ min^{-1}$)
1	69	F	1.73	Pneumonia	33	11	0
2	57	M	2.07	Pneumonia	32	13	NE (0.04)
3	46	M	2.13	Intoxication	31	6	0
4	57	F	2.06	Aspiration	25	11	NE (0.13)
5	55	M	1.93	Pneumonia	19	7	0
6	68	F	1.64	Hemoptysis	17	8	NE (0.04)
7	58	M	1.77	Pneumonia	16	7	0
8	41	M	1.85	Sepsis	22	9	NE (0.02)
9	48	M	1.9	Pneumonia	27	8	0
10	52	M	1.95	Pneumonia	24	8	0
11	79	F	1.45	Sepsis	22	7	NE (0.18)

on regional organ perfusion and function [7], in particular on the hepato-splanchnic organs (i.e. liver and gastrointestinal tract) [7, 9, 10]. Given the postulated role of the hepato-splanchnic region in the pathogenesis of septic shock and multiple organ failure [11], the important question remains to be clarified whether the prone position may alter hepato-splanchnic perfusion and oxygen exchange. We therefore studied the effects of prone position on hepato-splanchnic hemodynamics, metabolism and gut mucosal energy balance in critically ill patients with ALI.

Methods

The study was approved by the local university hospital ethics committee and written informed consent was obtained from the next of kin. The study was conducted according to the principles established in the Helsinki Declaration.

Patients

Eleven patients fulfilling the following criteria were studied: (1) ALI requiring mechanical ventilation [12]; (2) prone position indicated if PEEP more than 10 cmH_2O and FIO_2 more than 55%. The clinical characteristics of patients are listed in Table 1. Each patient was ventilated in the volume-controlled mode with tidal volumes between 6 and 8 ml kg^{-1}. Ventilator settings were unchanged throughout the study. All patients were deeply sedated with midazolam and fentanyl infused at a constant rate in order to avoid circulatory changes caused by a stress-induced increase of endogenous catecholamines. Only for the purpose of the study, each patient was paralyzed with pancuronium.

Study procedures, measurements and calculations

Routine invasive hemodynamic monitoring included arterial and pulmonary artery thermodilution catheters. An angiography catheter was placed via the right jugular vein into a hepatic vein under ultrasound guidance [13]. A gastric tonometry tube (TRIP Catheter, Tonometrics, USA) was inserted, and its position was confirmed radiologically.

Hepato-splanchnic blood flow (HSBF) was estimated using a hepatic vein catheter and primed continuous indocyanine green (ICG, Pulsion, Munich, Germany) infusion. This technique has been validated for critically ill patients and described in detail [14, 15]. Briefly, after a priming bolus of 12 mg of ICG, a constant infusion of 0.5 mg min^{-1} was continued for 30 min. Arterial and hepatic vein indocyanine concentrations, obtained after 20, 25 and 30 min of infusion, were measured by spectrophotometry. HSBF was calculated according to the Fick principle [14, 15]. The coefficient of variation for consecutive blood flow measurements at 20, 25 and 30 min during each infusion of ICG was 5.7±3.5% (mean ± SD). The ICG extraction exceeded the limit of 10% in each single measurement (50±15%, mean ± SD). None of the patients had markedly elevated plasma levels of bilirubin (16±13 μmol l^{-1}, mean ± SD).

Arterial, mixed venous and hepatic vein blood gases were measured using a blood gas analyzer (ABL 520, Radiometer, Copenhagen, Denmark) and hemoglobin oxygen saturation was determined by a co-oximeter (OSM-3, Radiometer, Copenhagen, Denmark). Systemic oxygen delivery (DO_{2sys}) and systemic oxygen consumption (VO_{2sys}) were calculated from the standard formula. Hepato-splanchnic DO_2 (DO_{2spl}) was calculated as the product of HSBF and arterial oxygen content (CaO_2), and hepato-splanchnic VO_2 (VO_{2spl}) as the product of HSBF and the difference between CaO_2 and hepatic vein oxygen content. The arterial and hepatic venous lactate was measured enzymatically (Hitachi 717, Boehringer-Mannheim, Germany). Hepato-splanchnic lactate uptake rate was calculated as the product of HSBF and the arterial-hepatic venous lactate difference.

Gastric intramucosal PCO_2 ($PiCO_2$) was measured semi-continuously (time equilibration 10 min) by automated air tonometry (Tonocap, Datex-Ohmeda, Helsinky, Finland). Each patient was given the proton pump-inhibitor omeprazol (80 mg day^{-1} i.v.) to increase gastric juice pH to a value of greater than 4, which improves the accuracy of $PiCO_2$ measurement [16].

Intra-abdominal pressure (IAP) was measured indirectly as an intra-vesicular pressure using the revised Kron's technique [17]. After being zeroed at the level of the midaxillary line, the IAP was measured at end-expiration. The urine output was measured at the end of each study period just before the bladder was filled with 50 ml of saline. After the IAP measurement 50 ml of saline was aspirated and completely removed from the bladder. The patency of the Foley catheter was checked shortly after each positional change.

Protocol

All measurements were made in the supine position (SP1), after 90 min of prone position (PP) and after 90 min of supine repositioning (SP2). All patients were placed on air-cushioned beds (Nimbus II, UK) and in prone position (swimmer's position), except for the oro-facial area, shoulder and pelvis were not supported in any way. During the whole protocol each patient was hemodynamically stable, the doses of norepinephrine (if needed) and

Table 2 Systemic hemodynamics in the supine and prone positions. Data are median and 25th and 75th percentiles (*SP1* supine position before prone position, *PP* prone position, *SP2* supine position after PP, *MAP* mean arterial pressure, *MPAP* mean pulmonary artery pressure, *HR* heart rate, *CI* cardiac index, *PAOP* pulmonary artery occlusion pressure, *CVP* central venous pressure, *A-lactate* arterial lactate)

	SP1	PP	SP2
MAP (mmHg)	85 (81; 99)	86 (84; 107)	86 (82; 106)
MPAP (mmHg)	30 (26; 33)	29 (26; 33)	27 (25; 32)
HR (beats min^{-1})	76 (69; 85)	72 (69; 83)	70 (65; 77)
CI ($l\ min^{-1}\ m^{-2}$)	3.5 (3.0; 3.6)	3.4 (3.1; 3.6)	3.2 (2.7; 3.6)
PAOP (mmHg)	15 (15; 16)	15 (15; 16)	16 (15; 16)
CVP (mmHg)	15 (13; 16)	14 (14; 15)	14 (13; 15)
A-lactate (mmol l^{-1})	1.5 (1.0; 1.7)	1.5 (0.9; 1.9)	1.3 (1.0; 1.8)

Table 3 Basic ventilatory parameters in the supine and prone positions. Data are median and 25th and 75th percentiles (*SP1* supine position before prone position, *PP* prone position, *SP2* supine position after PP, PaO_2/FIO_2 ratio between arterial oxygen tension and inspired oxygen concentration, *Vt* tidal volume, *PEEP* positive end-expiratory pressure, *MawP* mean airway pressure, *PawP* peak airway pressure, $PaCO_2$ arterial carbon dioxide partial pressure)

	SP1	PP	SP2
PaO_2/FIO_2	163 (149; 200)	183 (172; 214)[a]	165 (148; 197)[b]
FIO_2 (%)	60 (55; 65)	60 (55; 65)	60 (55; 65)
Vt (ml)	565 (520; 620)	560 (500; 620)	555 (530; 630)
Respiration ($breaths\ min^{-1}$)	18 (16; 22)	18 (16; 22)	18 (16; 22)
PEEP (cmH_2O)	12 (10; 13)	12 (10; 13)	12 (10; 13)
MawP (cmH_2O)	16 (16; 19)	17 (17; 20)	16 (16; 18)
PawP (cmH_2O)	32 (31; 35)	33 (32; 36)	31 (30; 35)
$PaCO_2$ (kPa)	5.6 (5.1; 6.0)	5.2 (5.1; 5.9)	5.5 (5.0; 5.9)

[a] $p<0.05$ versus SP1 [b] $p<0.05$ versus PP

fluid infusion rates were kept constant, no red blood cells were given and no other therapeutic interventions were performed.

Statistical analysis

All values shown are medians and interquartile range. After exclusion of normal distribution, the differences between the periods were analyzed by the Friedman rank sign analysis of variance and a subsequent Dunn's test for multiple comparisons. The differences in regional blood flow between patients with and without norepinephrine were tested by the Mann-Whitney rank sum test. Statistical significance was considered at p less than 0.05.

Results

Systemic hemodynamics and gas exchange parameters over the course of the study are summarized in Table 2. Prone position did not change any of the variables related to systemic and pulmonary hemodynamics. Similarly, mean airway pressure and peak airway pressure did not change between the supine and prone positions (Table 3). Turning a patient to the prone position resulted in a significant increase in PaO_2/FIO_2 ratio, which decreased back to SP1 values after supine repositioning (Table 3). The IAP was not significantly affected by the prone position (Fig. 1). Evolution of IAP was: before prone 10 (9;11), during prone 11 (10;13) and after prone 11 (10;11) mmHg. Concomitantly, the abdominal perfusion pressure (APP), defined as the difference between MAP and IAP [18], remained unchanged through the whole study period (Table 4). Taking 15 mmHg as a cutoff point for intra-abdominal hypertension [19], none of the

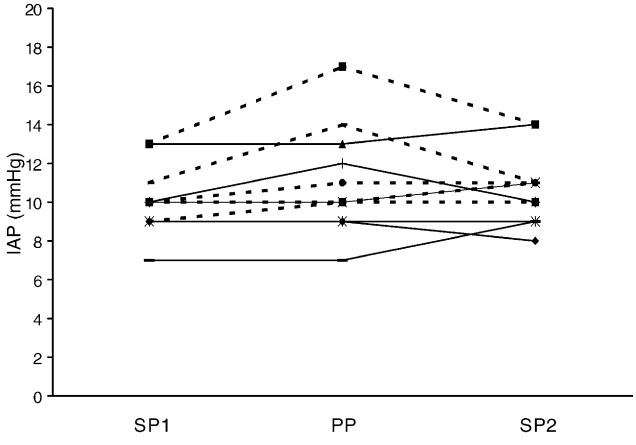

Fig. 1 Individual changes in intra-abdominal pressure (IAP) in the supine and prone positions (n=11). *Dashed line*: patient receiving norepinephrine

Table 4 Hepato-splanchnic hemodynamics and metabolism in the supine and prone positions. Data are median and 25th and 75th percentiles (*SP1* supine position before prone position, *PP* prone position, *SP2* supine position after PP, DO_{2spl} hepato-splanchnic oxygen delivery, VO_2spl hepato-splanchnic oxygen uptake, SvO_2-ShO_2 gradient between mixed venous and hepatic venous O_2 saturation, *HVP* hepatic venous pressure, PCO_2 *gap* gastric mucosal-arterial PCO_2 gradient, *Lact upt* hepato-splanchnic lactate uptake, *ICG extraction* indocyanine green liver extraction, *APP* abdominal perfusion pressure)

	SP1	PP	SP2
DO_{2spl} (ml min^{-1} m^{-2})	149 (125; 184)	154 (119; 211)	141 (107; 191)
VO_2spl (ml min^{-1} m^{-2})	63 (50; 70)	54 (52; 63)	59 (48; 75)
SvO_2-ShO_2 (%)	17 (8; 20)	16 (12; 22)	19 (12; 28)
HVP (mmHg)	15 (13; 16)	15 (14; 16)	14 (14; 15)
PCO_2 gap (mmHg)	6.8 (3.6; 8.6)	7.9 (3.8; 9.0)	6.8 (3.8; 11.0)
Lact upt (mmol min^{-1} m^{-2})	0.5 (0.2; 0.7)	0.5 (0.1; 0.8)	0.4 (0.3; 0.8)
ICG extraction (%)	45 (34; 61)	50 (39; 53)	51 (35; 56)
APP (mmHg)	76 (69; 90)	76 (73; 101)	78 (71; 100)
Urine output (ml/h)	130 (68; 149)	130 (110; 215)	140 (68; 198)

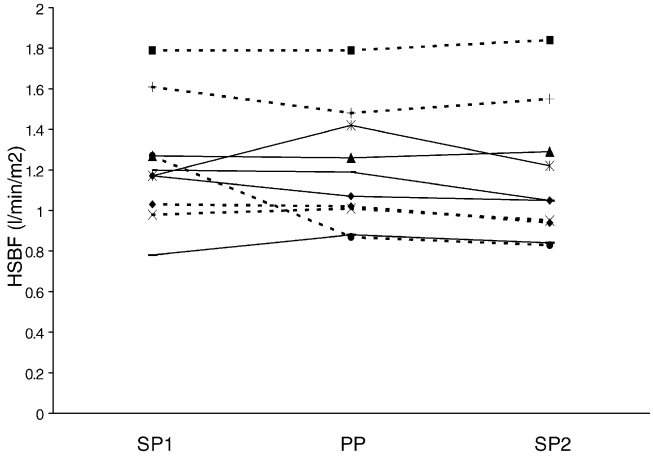

Fig. 2 Individual changes in hepato-splanchnic blood flow (HSBF) in the supine and prone positions (n=10). *Dashed line*: patient receiving norepinephrine

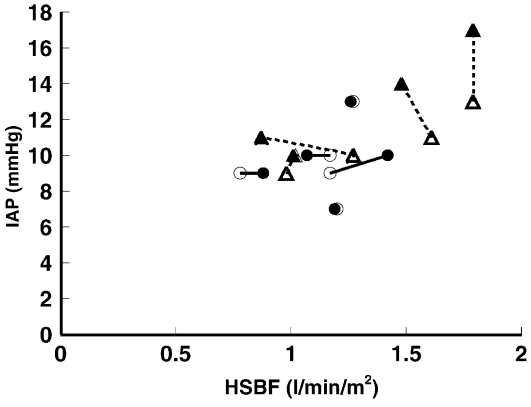

Fig. 3 Individual changes in hepato-splanchnic blood flow and intra-abdominal pressure and their relationship after turning to the prone position (n=10). *Filled line and circle*: patient without vasopressor support. *Dashed line and triangle*: patient receiving norepinephrine. *Open symbols*: supine position (SP1), *filled symbols*: prone position

patients had IAP of 15 mmHg at baseline. A significant increase of IAP (a change >3 mmHg [8]) during the prone position was observed in only two cases (Fig. 1).

Figure 2 depicts individual responses in the regional hepato-splanchnic blood flow to the prone position. One patient had to be excluded from the analysis of HSBF for technical reasons. No significant differences between the supine and prone positions were observed (HSBF in SP1: 1.2 (1.0;1.3), PP: 1.1 (1.0;1.4), SP2: 1.1 (0.9;1.2) l min^{-1} m^{-2}). Variable and small individual changes were not related to changes in IAP (Fig. 3). In five patients receiving norepinephrine (dashed lines in individual figures) neither the baseline hepato-splanchnic blood flow nor its response to positional changes (SP1 vs PP) differed from the group of patients without the vaso-pressor support (p=0.42; p=0.55, respectively). There were no changes in hepato-splanchnic oxygen delivery (DO_{2spl}) or oxygen consumption (VO_{2spl}) (Table 4). No marked variations in DO_{2spl} and VO_{2spl} were observed in the two patients with reported increase in IAP during the prone position. Stable regional hemodynamics and oxygen transport were accompanied by an unchanged gastric mucosal-arterial PCO_2 gap (Table 4). The PCO_2 gap also remained unaffected in the two patients with increase in IAP while prone. Furthermore, neither liver lactate uptake nor ICG extraction were affected by positional changes (Table 4). The urine output did not change either (Table 4).

Discussion

Our clinical study demonstrates the effects of prone position on hepato-splanchnic hemodynamics and oxygen transport in mechanically ventilated patients with ALI. The principal findings were that: (1) in patients without significant intra-abdominal hypertension, the IAP remains unaffected during the time they are in the prone position. (2) In this situation, turning to the prone position compromises neither regional hepato-splanchnic circulation, as assessed by total hepato-splanchnic blood

flow and oxygen transport, nor the gut mucosal energy balance, as indicated by unchanged gastric mucosal-arterial PCO_2 gap. (3) Mechanical ventilation in prone position does not adversely affect indices of hepato-splanchnic metabolism and liver function, as documented by stable regional lactate uptake and ICG extraction.

Our results, in agreement with other human studies [4, 5, 6], confirm that mechanical ventilation of stable patients in the prone position is not associated with adverse effects on systemic and/or pulmonary hemodynamics. It must be stressed, however, that the response of the regional tissue perfusion may differ substantially from that of global hemodynamics [20]. With this in mind, the blood flow to the gut and liver is of particular interest, as splanchnic hypoperfusion may play a central role in the pathogenesis of multiple organ failure during sepsis [10, 11, 20]. Since up to now little human data have been available, the main issue to resolve in our study was determination of the physiologic effects of turning critically ill patients with ALI from the supine to the prone position with regard to the hepato-splanchnic perfusion and metabolism. In this context, the major concern about prone positioning is that it may lead to increased IAP, which, if acutely elevated, disturbs organ perfusion and function [7]. In our study shifting a patient from the supine to the prone position did not result in significantly increased IAP, which is in keeping with the findings of Pelosi et al. [21].

Interestingly, Kiefer et al. recently demonstrated that although the mean intragastric pressure was not significantly changed in the prone position, nearly 50% of patients exhibited an increased IAP by more than 3 mmHg [8]. At first glance, this observation is in contrast to the present study. Our findings did not reveal the magnitude of changes in the IAP seen by Kiefer et al. Only two of our patients showed a tendency to increased IAP while prone (the differences between SP1 and PP were 3 and 4 mmHg, respectively). However, in contrast to our medical patient population, the majority of patients in Kiefer's study had abdominal sepsis (P. Kiefer, personal communication). Other authors [22] reported similar inconsistent inter-individual responses of the IAP. Hence, different groups of patients (medical versus surgical), different underlying diseases, body composition (obese versus non-obese) and abdominal wall compliance as well as various methods of positioning may assume importance in this context. In this respect, particular care has been paid to allow free abdominal movement using supported chest and pelvis [21, 23]. Hering et al. recently demonstrated that no special support to minimize restriction of the abdomen is necessary in patients without intra-abdominal hypertension [22, 24], which is also supported by our findings.

The key finding of the present study indicates that neither total hepato-splanchnic blood flow, regional oxygen transport nor gastric mucosal-arterial PCO_2 gap appear to be influenced when placing the patients prone, at least in the time interval studied. There have been only two clinical studies so far that have attempted to elucidate the impact of the prone position on the hepatic capacity to eliminate ICG [24] and gastric mucosal-arterial PCO_2 gap [8, 24] in mechanically ventilated patients with ALI. In a very recent study from Hering et al. the plasma disappearance rate of ICG was not altered despite moderately increased IAP during the time in the prone position [24]. ICG clearance may, however, be very variable in critically ill patients depending on both liver blood flow and liver dye extraction, so that liver blood flow or oxygen kinetics cannot be accurately inferred from the plasma disappearance rate of ICG [14]. In this context, our study provides additional information on the effects of prone position on the hepato-splanchnic blood flow and oxygen transport, as we measured the regional blood flow and oxygen kinetics using more rigorous methodology, i.e. the continuous ICG infusion combined with hepatic vein catheterization [14, 15].

In addition, our study also showed that the functional and metabolic markers such as hepato-splanchnic oxygen uptake, lactate clearance and ICG extraction remained unchanged, further indicating the lack of prone position-induced changes. Moreover, while Hering et al. studied only hemodynamically stable patients not requiring vasopressors, five of our patients received norepinephrine. Although patients treated with adrenergic agents may be more at risk for hepato-splanchnic hemodynamic alterations [25], the positional changes did not adversely influence different markers of regional perfusion and function in these five patients. It should be noted, however, that none of our patients exhibited significant intra-abdominal hypertension at any time point of the study. Thus, we cannot exclude a different response pattern of the regional hepato-splanchnic hemodynamics to the prone position in the case of marked intra-abdominal hypertension. Even though we could not evaluate the effects of an acute increase in IAP on regional perfusion, the clinically important implication of our study is that the hepato-splanchnic perfusion is not influenced by the prone position as long as cardiac output remains stable and no major changes in IAP occur.

Regarding the gastric tonometry, the study by Kiefer et al. [8] showed that the prone position, provided that the IAP increases, might be associated with an increased gastric mucosal-arterial PCO_2 gradient. Because the changes in gastric mucosal-arterial PCO_2 gap were modest, the question arises as to whether the statistical significance of elevated PCO_2 gap in their study had any clinical significance, as also pointed out by the authors. Since, in the above-mentioned study, the PCO_2 gradient increased as late as after 2 h in the prone position, one could argue that a longer study period would be required before the identification of significant physiological changes. However, in contrast to gastric tonometry measurements, the effect of increased IAP on regional blood flow is obvi-

ously not time-dependent since, in an animal experiment, a reduction of liver blood flow induced by an acute increase in IAP was evident within 15 min [26].

The kidney is another organ, whose function may be affected by positional changes and intra-abdominal hypertension. Although renal functions were not specifically studied in our study, turning prone had no effect on urine output, which agrees with a recent report showing that prone positioning did not impair renal functions, even in the presence of a small increase in IAP [22].

In conclusion, the present study gives further evidence that mechanical ventilation of ALI patients in the prone position does not exert any detrimental effects on the regional hepato-splanchnic perfusion, oxygen transport and gut mucosal energy balance provided that no major increase in IAP occurs and cardiac output remains stable. Additional studies are required to evaluate the regional extrapulmonary responses to the prone position in patients with intra-abdominal hypertension.

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