Cardio-respiratory Changes with Increased Intra-bladder Pressure in Prone Position during Anesthesia

Yasuhiko HIRAGA and Masayoshi HYODO

Twenty non-obese patients, 13–29 years of age, operated on for scoliosis were examined for cardio-respiratory changes that occur during positioning on bolsters, and the effect on the cardio-respiratory system of raised intra-abdominal pressure was evaluated. Hemodynamic and respiratory responses were measured when the position was changed from supine to prone and back to supine during anesthesia. We measured the intra-bladder pressure using a transurethral catheter (IBP) as an index of the intra-abdominal pressure. When the position was changed from supine to prone, the cardiac index (CI) decreased by 10–13% and the systemic vascular resistance index increased by 8–14%. IBP rose significantly ($P < 0.001$), but it remained below 5 mmHg. These changes continued to be mild until the patient was returned to the supine position. Pa_{O_2}, A-aD_{O_2} and Qs/Qt remained unchanged. CI decreased significantly ($P < 0.001$) when IBP was increased to 10 mmHg by abdominal compression, but was not affected when IBP was increased to only 5 mmHg. It was concluded that mild abdominal compression in the prone position during anesthesia has little effect on the cardio-respiratory system in lean young subjects. (Key words: hemodynamics, prone position, intra-bladder pressure)

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During surgery and anesthesia, the patient is usually positioned to provide optimal surgical conditions. Many surgical positions, however, can impose physiological burdens on the patient. A prone position may adversely affect both circulation and ventilation in anesthetized patients. Various frames have been designed for improved surgical access and reduction of inferior vena cava pressure in the prone position[1–3]. Alterations in position may cause cardiovascular changes because anesthetics blunt normal compensatory mechanisms. However, there have been few studies of the circulatory and ventilatory effects of postural change and the maintenance of the prone position for several hours[4]. It has been reported that there are few cardiovascular problems in the prone position unless the adbomen is compressed. However, there have been no studies on how extended compression of the abdomen influences the cardio-respiratory system in the prone position. The purpose of this study is to

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Table 1. Characteristics of the patients

	age (years)	weight (kg)	height (cm)
range	13–29	32–62	141–170
mean	17.7	44.4	156.4
SEM	1.0	1.7	1.8

n=20

Table 2. Characteristics of the patients whose IBP was 1.0 mmHg in the prone position

	age (years)	weight (kg)	height (cm)
range	14–29	38–57	149–166
mean	20.4	46.3	156.5
SEM	1.9	2.5	2.2

n=8

explore the cardio-respiratory effects of postural change and to determine the influence of intra-abdominal pressure on the cardio-respiratory system in prone patients under general anesthesia.

Materials and Methods

Twenty patients, 13–29 years of age, who were scheduled for elective corrective spinal fusion and instrumentation, were studied. None of the patients had any cardiac disease, and none were obese. They were classified as ASA Physical Status 1. Every patient was informed of the nature of the experiment in detail, and written consent was obtained before the start of the study. Table 1 lists the patients' data. Premedication consisted of hydroxyzine 50 mg and atropine 0.5 mg, injected intramuscularly one hour before induction of anesthesia. Anesthesia was induced with fentanyl 4 $\mu g \cdot kg^{-1}$, droperidol 0.15 $mg \cdot kg^{-1}$ and thiamylal 4 $mg \cdot kg^{-1}$. After endotracheal intubation with pancuronium 0.1 $mg \cdot kg^{-1}$, anesthesia was maintained with 0.3–1.0% enflurane and 67% nitrous oxide in oxygen supplemented by fentanyl and droperidol. Pancuronium was administered whenever it was required. Controlled ventilation was maintained to keep Pa_{CO_2} at 30–40 mmHg during the study. No positive end-expiratory pressure was applied. Immediately after induction of anesthesia, a 21-gauge teflon catheter was inserted into a radial artery for the measurement of blood pressure and the sampling of blood for gas analysis. A balloon-tipped thermodilution Swan-Ganz catheter was inserted into the pulmonary artery through the right internal jugular vein for the measurement of right atrial pressure (RAP), mean pulmonary artery pressure (PA), pulmonary capillary wedge pressure (PCWP) and cardiac output (CO) and for the sampling of mixed venous blood. After the patient's condition was stabilized, these hemodynamic and respiratory variables were measured with the patient supine, then 5, 15, 30 and 60 min after being slowly and gently turned to the prone position. After completion of the operation, they were measured again in the prone position, then 5 and 15 min after a return to the supine position. Instead of the intra-abdominal pressure, the intra-bladder pressure using a transurethral catheter (IBP) was measured by the method of Iberti et al.[5] (fig. 1). Hemodynamic responses were measured at the end-expiratory periods.

In another study, we examined the effect of compression of the abdomen on the hemodynamic system. In 8 subjects whose IBP was 1.0 mmHg in the prone position, pressure was applied to the abdomen. Table 2 lists the patients' data. IBP was increased to 3 mmHg, 5 mmHg and 10 mmHg, and heart rate (HR), mean arterial pressure (mAP), and CO were measured at each time interval.

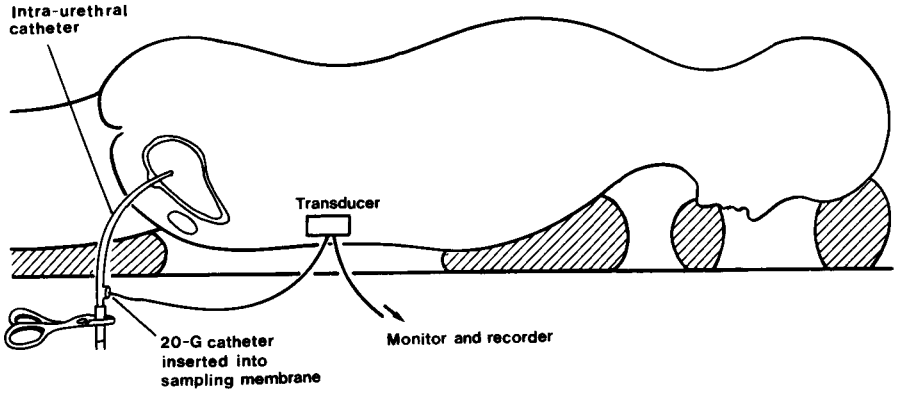

Fig. 1. Diagram of a patient in the prone position showing the intra-bladder pressure monitoring technique with a transurethral catheter. The transducer for the bladder catheter was set at zero at the level of the pubis.

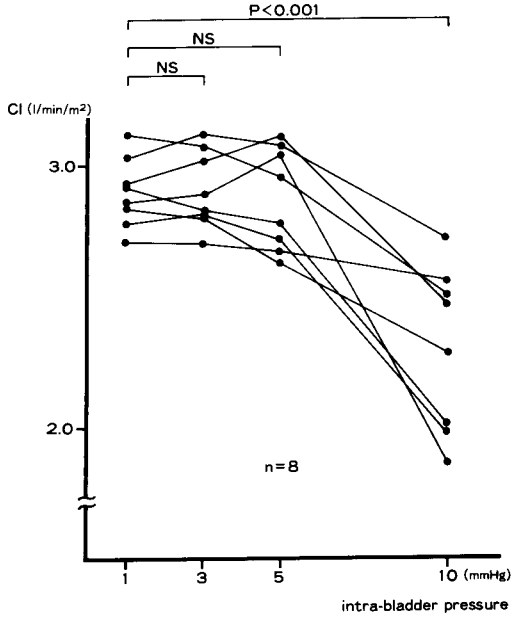

Fig. 2. Effect of increased intra-bladder pressure (IBP) on cardiac index (CI).

Eight anesthetized patients had IBP of 1 mmHg in the prone position. IBP and cardiac output were measured during abdominal compression. There was a significant difference in CI between IBP of 1 mmHg and IBP of 10 mmHg. There was no significant difference in CI when IBP was 1 mmHg, 3 mmHg or 5 mmHg.

NS: not significant.

Cardiac index (CI), systemic vascular resistance index (SVRI), pulmonary vascular resistance index (PVRI), shunt fraction (Qs/Qt), and alveolar arterial difference of partial pressure of oxygen ($A\text{-}aDo_2$) were calculated using the standard formulas. The measurement of arterial and mixed venous blood gases was performed with an ABL 2 analyzer (Radiometer, Copenhagen, Denmark). CO was measured with an RMC-1200 CO computer system (Nihon Kohden, Tokyo, Japan). The paired t-test was used for statistical analysis, and a P value of less than 0.05 was considered statistically significant.

When the patient was prone, the weight of the torso was borne by the upper thorax and pelvis, permitting the abdomen to protrude. Soft bolsters were placed under the left and right sides of the chest wall. The thickness of the required pads varied with the ventral protrusion of the abdomen relative to an imaginary plane between the manubrium of the sternum and the anterior superior iliac spines. The head was supported by pads under the chin, cheeks, and forehead: pressure on the eyes was avoided. The arms were

Table 3. Cardio-respiratory effects of change from supine to prone and prone to supine position during anesthesia

	supine	prone				prone	supine	
		5 min	15 min	30 min	60 min		5 min	15 min
mAP (mmHg)	73.3 ± 1.9	73.1 ± 1.9	73.5 ± 2.1	71.9 ± 1.8	72.7 ± 2.0	74.8 ± 3.6	75.2 ± 3.5	76.6 ± 3.7
HR (beats·min^{-1})	87.0 ± 2.4	85.4 ± 2.5	78.7 ± 2.8**	77.8 ± 2.1**	76.6 ± 2.1**	95.0 ± 3.2	96.2 ± 3.2	97.4 ± 3.4#
RAP (mmHg)	8.1 ± 0.4	7.9 ± 0.6	7.9 ± 0.6	8.4 ± 0.7	8.4 ± 0.6	7.2 ± 0.4	7.2 ± 0.5	7.3 ± 0.8
PA (mmHg)	14.0 ± 1.0	14.9 ± 1.0	15.2 ± 0.9	15.3 ± 0.9	15.3 ± 1.0	16.3 ± 1.4	16.4 ± 1.4	16.8 ± 1.6
PCWP (mmHg)	10.3 ± 0.9	10.6 ± 0.7	10.9 ± 0.7	11.2 ± 0.8	11.2 ± 0.9	12.0 ± 0.8	11.7 ± 0.7	11.8 ± 0.9
CI (l·min^{-1}m^{-2})	3.78 ± 0.14	3.59 ± 0.16	3.40 ± 0.17*	3.26 ± 0.14**	3.44 ± 0.12**	4.20 ± 0.31	4.38 ± 0.32	4.50 ± 0.30#
SVRI (dyne·sec·cm^{-5}·m^{-2})	1410 ± 59	1590 ± 89*	1582 ± 93**	1631 ± 90**	1532 ± 71**	1264 ± 88	1189 ± 75	1143 ± 80#
PVRI (dyne·sec·cm^{-5}·m^{-2})	78.5 ± 4.0	97.6 ± 5.8	98.1 ± 7.0*	101.7 ± 6.7**	97.7 ± 8.5*	80.8 ± 9.8	80.1 ± 9.6	78.6 ± 10.4
SI (ml·beat^{-1}·m^{-2})	43.5 ± 1.8	42.2 ± 1.7	42.0 ± 1.7	42.0 ± 1.9	44.5 ± 2.1	44.6 ± 2.2	45.7 ± 2.3	46.4 ± 2.0
Pa_{O_2} (mmHg)	183.4 ± 9.2	190.8 ± 9.0	190.3 ± 8.9	189.0 ± 8.8	191.6 ± 8.5	179.8 ± 9.0	182.1 ± 8.9	181.4 ± 9.3
Qs/Qt (%)	2.95 ± 0.28	2.96 ± 0.23	2.96 ± 0.24	2.95 ± 0.24	2.90 ± 0.24	3.60 ± 0.34	3.61 ± 0.30	3.40 ± 0.33
A-aDo_2 (mmHg)	54.2 ± 6.0	53.4 ± 3.9	54.0 ± 4.0	53.6 ± 3.8	53.9 ± 4.2	58.2 ± 3.9	59.8 ± 3.8	58.2 ± 4.0
IBP (mmHg)	0.8 ± 0.2	2.6 ± 0.4**	2.4 ± 0.4**	2.8 ± 0.5**	2.5 ± 0.4**	2.7 ± 0.4	0.9 ± 0.5 ##	0.8 ± 0.2##

*significant difference from supine, $P < 0.05$ #significant difference from prone, $P < 0.05$
**significant difference from supine, $P < 0.01$ ##significant difference from prone, $P < 0.01$
All values are given as mean ± SEM

secured to the patient's sides with a draw sheet to allow the surgeon better access to the operative site.

Results

Table 3 shows the cardio-respiratory effects of postural change. Systolic blood pressure, diastolic blood pressure and mAP did not change. HR decreased significantly 15 min after the patient was changed to the prone position. CI decreased significantly ($P < 0.05$ after 15 min, $P < 0.01$ after 30 min) and continued to be low until the supine position was resumed. In contrast, SVRI significantly increased approximately 10%. RAP, PA and PCWP showed no significant changes. Pa_{O_2}, A-aDo_2 and Qs/Qt remained unchanged.

IBP increased significantly ($P < 0.001$) in the prone position and re-

turned to almost its former level when the supine position was resumed. Figure 2 shows the effect on CI of increased IBP. CI remained unchanged when IBP was increased to 5 mmHg with abdominal compressions, but decreased significantly when IBP was raised to 10 mmHg. Neither mAP nor HR changed much even when IBP was increased to 10 mmHg.

Discussion

Many physiological mechanisms are involved in the cardiovascular changes occurring during postural change. As anesthetics and muscle relaxants decrease compensatory responses to postural changes, changes in body position mainly lead to changes in hydrostatic pressure which affect both systemic and pulmonary circulation, particularly the venous circulation[6]. These changes are maximal along the long axis of the body. They can usually be ignored when the patient is moved from the supine to the prone position. Because of this, there are few cardiovascular problems in the prone position if the patient is positoned so that no pressure is exerted on the inferior vena cava[7]. However, various hemodynamic changes in the prone position have been reported. Fukushi et al. reported a decrease in CO of 18% and in stroke volume of 23% in Mahomet's position during general anesthesia[8]. Hirakawa et al. noted a decrease in CI of 10–20% and a significant decrease of HR in prone patients supported by bolsters and a Mackay frame[4]. Brustowicz observed hemodynamic responses for 30 min after a change to the prone position in patients supported by a Hall frame, and all data remained unchanged[9]. Hirakawa found an increase in CO of 13% in conscious patients in the prone position without a frame and a decrease in CO of 29% in those on a Mackay frame[4]. BP remained unchanged in all these studies. It is difficult to generalize about the hemodynamics in the prone position from these studies, since patients' ages, position, mechanical ventilation, anesthetics, and techniques of supporting the body were not controlled. The effects of the prone position on the cardiovascular system differ not only with the depth of anesthesia and the concentration of anesthetics but also with the type of support of the prone patient. We observed a decrease in HR of 4–10%, a decrease in CI of 10–13% and an increase in SVRI of 8–14% when the position was changed from supine to prone.

Why did HR decrease in this study? In some studies HR remained unchanged[8,9] and in others HR decreased in the prone position[4,10]. They differ from diseases, anesthetics, positioning, age, circulatory volume, or length of time. One reason of a decrease in HR is that neuroleptoanesthesia tends to maintain autonomic mechanisms and reflexes. Fentanyl induces bradycardia by stimulating the central vagal nucleus[11]. Another is that the carotid sinus might be pressed when the head is positioned. As a result, baroreceptors might act to inhibit the sympathetic outflow to the cardiovascular system and to increase vagal cardiac nerve activity. Furthermore, when respiration is held constant, the primary effect of carotid chemoreceptor stimulation on the heart is a decrease in both HR and force of contraction. The minimum alveolar concentration of anesthetics that block adrenergic reflexes was higher than the anesthetic concentration in this study[12]. Therefore, autonomic function might be maintained and HR decreased in this study.

In this study, CO also remained low for over one hour, even though the legs were kept at the heart level. Increased abdominal pressure might lead to inferior vena cava obstruction and

CO might be decreased. However, the venous wall contains little smooth muscle tissue, so the venous system is very compliant, and venous volume is not necessarily proportional to venous pressure. Although Richardson and Trinkle reported[13] a close relationship between IVC pressure and intra-abdominal pressure, IVC flow was not proportional to intra-abdominal pressure. Compression of the abdomen is distributed because abdominal viscera are fluid-like. As a result, the IVC flow seems to remain unchanged and IVC pressure increases when the abdomen is compressed slightly. It is necessary to measure intra-abdominal pressure to assess the degree of compression of the abdomen when hemodynamics are measured in the prone position. However, it is clinically difficult to measure intra-abdominal pressure noninvasively. Therefore, in this study, we measured the intra-bladder pressure using a transurethral catheter (IBP) which is closely proportional to the intra-abdominal pressure. IBP increased to 4 mmHg at the most when the patient was moved to the prone position. Stroke index remained almost unchanged. CI decreased significantly when IBP was increased to 10 mmHg by abdominal compression, but CI remained unchanged when IBP was increased to 5 mmHg. Thus, abdominal pressure above 10 mmHg by the abdominal compression may reduce CO by decreasing venous return to the heart. Therefore, this result indicates that mild abdominal compression below 5 mmHg IBP appears to have little effect on IVC flow and CO.

Postural change also affect the respiratory system by the force of gravity. The changes of functional residual capacity (FRC) caused by postural change are proportional to the sine curve of the long axis of the body along the vertical axis. Therefore, little gravitational force affects the respiratory system in the prone position. In conscious healthy persons, however, the decrease in FRC in the prone position is less than that in the supine position if devices provide good support. This is due to the ventral protrusion of the abdominal wall and its effect on the diaphragm[14]. In the prone position, the dorsal parts of the lung are released from the effect of the abdominal contents and the abdominal hydrostatic pressure. Therefore, an increase in Pa_{O_2} was observed after conscious patients were turned to the prone position[15].

In the supine position, induction of anesthesia is consistently accompanied by a significant decrease in FRC, a change in respiratory patterns, airway constriction and abnormal distribution of inspired gasses[16]. The reduction in FRC correlates well with the increase in $A\text{-}aDo_2$ during anesthesia. In this study, both $A\text{-}aDo_2$ and Qs/Qt remained unchanged even when CO decreased in the prone position. The different effects of the abdominal weight on the diaphragm in the two positions may, in part, underly the differences in inspired gas distribution[17]. All subjects in this study were young and lean, so that their abdominal weights might hardly affect the diaphragm and its volume, even in the supine position, and the reduction in their FRC might be probably slight. Furthermore, closing volume increases with age, and in supine adults over 35 years of age it exceeds the FRC. It is well known that the distribution of ventilation is disturbed and gas exchange deteriorates when the closing capacity exceeds the FRC. They were all under 30 years of age, the decrease in closing capacity due to aging could be ignored.

The results of this study indicate that slight compression of the abdomen in the prone position has little effect on the cardio-respiratory system in young non-obese subjects un-

der general anesthesia. In obese patients, however, intra-abdominal pressure may be greatly increased and cardio-respiratory system may be depressed.

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