Anesthesiology
V 45 No 1, July 1976

Cardiovascular and Respiratory Changes in Response to Change of Posture in the Very Obese

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Circulatory and respiratory effects of change of posture from sitting to supine were studied in 11 obese patients scheduled for gastric bypass operations. Patients were studied on the morning of operation before any medication was given. Average weight was 138.8 kg, 108.6 per cent above ideal weight. Change of posture from sitting to supine was associated with 11 per cent increase in oxygen consumption ($\dot{V}_{O_2}$), 35.5 per cent increase in cardiac output (CO), 35.8 per cent increase in cardiac index, 17.83 per cent decrease in arteriovenous oxygen difference, 31 per cent increase in mean pulmonary arterial pressure, 44 per cent increase in pulmonary-artery wedge pressure, 21.5 per cent decrease in peripheral resistance (PR), 6 per cent decrease in heart rate, and 17.7 per cent increase in venous admixture ($\dot{Q}_s/\dot{Q}_t$). All these changes were significant. There was no change in mean arterial pressure, alveolo-arterial O_2 difference and respiratory rate. The increase in CO was attributed to reduction in PR and to increased $\dot{V}_{O_2}$ probably due to increased work of breathing. (Key words: Ventilation, obesity; Heart, cardiac output, obesity; Complications, obesity; Position, ventilatory effects.)

HEMODYNAMIC EFFECTS of change of posture from sitting or standing to supine in normal man have been the subject of several studies.[1–3] Other studies[4–6] have dealt with the effects of severe obesity on cardiovascular dynamics. However, no detailed study of hemodynamic effects of change of posture in the very obese has been reported.

The effects of morbid obesity on various aspects of pulmonary function have been extensively studied.[7–14] Few data on the effects of changes in posture on pulmonary closing volume (CV) in relationship to functional residual capacity (FRC) in the very obese are available, however.

The present study measured the effects of change of posture from sitting to supine on hemodynamics and pulmonary function in the very obese.

Methods

Patients scheduled for gastric bypass procedures were studied on the morning of the operation. Informed consent was obtained from each patient the previous night. Every patient weighed at least 45 kg (100 lb) more than ideal body weight. No sedative or other drug was used before the study.

ABBREVIATIONS

$A\text{-}aD_{O_2}$ = alveolo-arterial oxygen difference ($FI_{O_2} = 1$)
Ca_{O_2} = oxygen content in arterial blood
$Ca_{O_2} - C\bar{v}_{O_2}$ = arteriovenous oxygen difference
Cc_{O_2} = oxygen content in pulmonary capillary blood
CI = cardiac index
CO = cardiac output
$C\bar{v}_{O_2}$ = oxygen content in mixed venous blood
Hb = hemoglobin content
HR = heart rate
MAP = mean arterial pressure
PAP = mean pulmonary arterial pressure
PAWP = pulmonary-artery wedge pressure
P_B = barometric pressure
PR = peripheral resistance
PVR = pulmonary vascular resistance
$\dot{Q}_s/\dot{Q}_t$ = percentage venous admixture
RR = respiratory rate
$\dot{V}_{O_2}$ = oxygen consumption (STPD)

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Received from the Department of Anesthesia (Intensive Care Unit), College of Medicine, University of Iowa, Iowa City, Iowa. Accepted for publication March 9, 1976.
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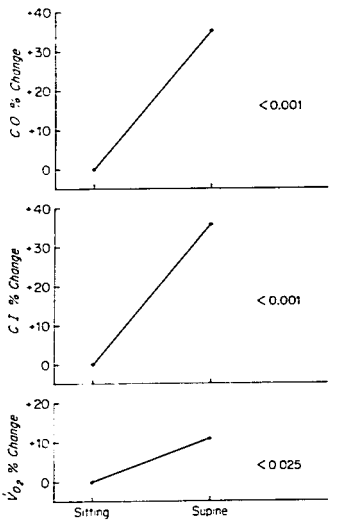

FIG. 1. Average percentage changes in cardiac output (CO), cardiac index (CI) and oxygen consumption ($\dot{V}_{O_2}$) with change of posture from sitting to supine. *P* value indicated to the right.

Percutaneous cannulation of brachial or radial arteries was performed using 18-gauge Teflon catheters. A vein was surgically exposed in the antecubital fossa and a triple-lumen Swan-Ganz catheter, size 7 French (Edwards Laboratories, Inc., Santa Ana, California), using pressure waveforms, was floated into position so that the distal orifice was in a branch of the pulmonary artery and the proximal orifice (20 cm from the tip of the catheter) was in the right ventricle. All samples of mixed venous blood were collected from the right ventricle. All catheters were connected to Statham transducers and pressure tracings were displayed and recorded on a multichannel recorder. Pulmonary-artery wedge pressures were obtained by inflating the Swan-Ganz balloon with 0.6–1 ml of air. Care was exercised to align accurately the transducers with the right atrium in the supine and sitting positions.

Cardiac output and $\dot{Q}_s/\dot{Q}_t$ were calculated from measurements made with each patient in the supine and sitting positions. The sequence of positions was reversed in alternate cases. A minimum of 20 minutes was allowed for stabilization following change of posture before measurements were made. Cardiac output was measured by Fick principle using a previously described method.[15] Measurement of $\dot{Q}_s/\dot{Q}_t$ was done as follows: a mask was firmly applied to the patient's face using standard straps and patient breathed 100 per cent oxygen through a Sierra valve nonrebreathing system for a minimum of 20 minutes to ensure denitrogenation. Cardiac output and $\dot{Q}_s/\dot{Q}_t$ were calculated from the formulas shown in the Appendix. Arterial and mixed venous blood samples used for both measurements were drawn in duplicate into heparinized chilled 5-ml glass syringes, which were kept on ice until samples were processed. Oxygen and carbon dioxide tensions were measured using IL 313 *p*H and blood-gas analysis equip-

TABLE 1. Effects of Change of Posture on Hemodynamic and Pulmonary Variables (Mean ± SE)

	Sitting	Supine	*P*
$\dot{V}_{O_2}$ (ml/min)	282.2 ± 19.5	312.6 ± 23.6	<.025
CO (l/min)	7.12 ± .65	9.55 ± .86	<.001
CI (l/min/m²)	3.04 ± .270	4.06 ± .32	<.001
$Ca_{O_2} - C\bar{v}_{O_2}$ (ml/100 ml)	4.2 ± .3	3.4 ± .2	<.001
PAP (torr)	15.7 ± 1.8	19.4 ± 1.1	<.01
PAWP (torr)	9.2 ± 1.5	13.4 ± 1.1	<.005
PR (units)	14.5 ± 1.2	11.3 ± 1.1	<.001
PVR (units)	1.0 ± .2	0.7 ± .1	N.S.
HR (beats/min)	81.8 ± 5.0	76.9 ± 4.8	<.0025
$\dot{Q}_s/\dot{Q}_t$	26.7% ± 1.8%	30.6% ± 1.5%	<.01
A-aD_{O_2} (torr)	314.4 ± 20.3	301.9 ± 14.6	N.S.
RR (breaths/min)	13.1 ± 1.3	14.6 ± 1.8	N.S.

ment.§ Hemoglobin content and oxygen saturation were measured using an IL CO-oximeter.§ Peripheral resistance and PVR were calculated according to formulas shown in the Appendix.

After blood samples had been obtained, patients were given appropriate preoperative medications and were then taken to the operating room.

Statistical analysis of results was done using Student's t test and correlation test.[16]

Results

Eleven patients, eight women and three men, were studied. Average age was 41.2 years, range 26 years to 46 years. Average weight was 138.84 kg. This was 108.6 per cent in excess of average ideal weight based on age and height. Only two patients weighed less than 100 per cent more than ideal weight (63 and 66 per cent). Average height was 165.3 cm, with a range of 137 to 190 cm. Average body surface area was 2.37 m^2. Average baseline blood-gas values, FI_{O_2} 0.21, were Pa_{O_2} 72 torr (range 59–85 torr) and Pa_{CO_2} 31 torr (range 27–44 torr). Measurements were made in the sitting position followed by the supine position in five patients and in reverse sequence in six patients. Although the former group was significantly older than the latter (50 years and 33.8 years, respectively), there was no difference in weights or percentage excesses over ideal weights between the two groups. Mean values for Pa_{O_2} were 258 and 267 torr during CO measurements and 358 and 346 torr during $\dot{Q}_s/\dot{Q}_t$ measurements in the supine and sitting positions, respectively. The lowest Pa_{O_2} measured was 115 torr. There was no difference between Pa_{CO_2} values obtained in the sitting and in the supine postures (31.7 and 32.2 torr, respectively).

§ Instrumentation Laboratory, Inc., Lexington, Massachusetts.

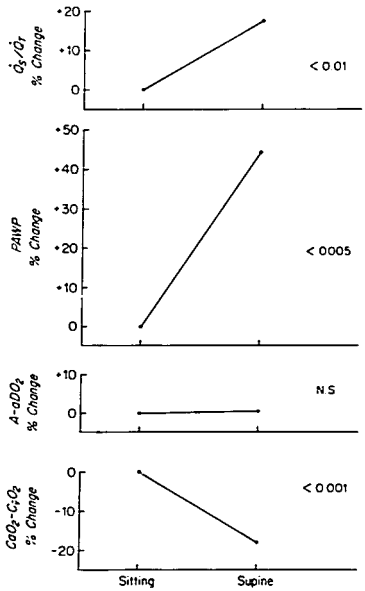

FIG. 2. Average percentage changes in venous admixture ($\dot{Q}_s/\dot{Q}_t$), pulmonary-artery wedge pressure (PAWP), alveolar-arterial oxygen difference (A-aD_{O_2}) and arteriovenous oxygen difference ($Ca_{O_2} - C\bar{v}_{O_2}$) with change of posture from sitting to supine. *P* value indicated to the right.

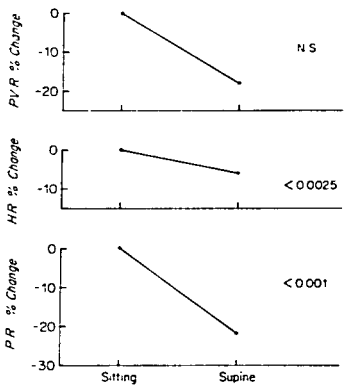

FIG. 3. Average percentage changes in pulmonary vascular resistance (PVR), heart rate (HR), and peripheral resistance (PR) with change of posture from sitting to supine. *P* value indicated to the right.

Table 1 shows mean values obtained in the two postures. Figures 1, 2 and 3 show means of percentage changes in measured and calculated variables following a change from the sitting to the supine posture. There was no difference in percent changes of these variables between patients who sat up first compared with those who were supine first.

Changes in $\dot{V}_{O_2}$, CO, $\dot{Q}_s/\dot{Q}_t$, PAP, PAWP, and PR did not correlate with weights or weight excess over ideal. Changes in $\dot{Q}_s/\dot{Q}_t$ did not correlate with changes in $\dot{V}_{O_2}$, CO, PAP or PAWP. There was no difference in MAP between supine and sitting positions (95.5 and 92.2 torr, respectively).

Discussion

In normal subjects, cardiac output is less in the upright than in the supine position.[2] This is unaccompanied by changes in $\dot{V}_{O_2}$, but $Ca_{O_2} - C\bar{v}_{O_2}$ increases significantly in the upright posture. In the present study, changes in CO were in the same direction. However, unlike normal subjects, obese patients showed significant increases in $\dot{V}_{O_2}$ in the supine position. Decreases in CO in healthy subjects in the upright position are associated with marked reduction in blood flow to the lower limbs[2] and increased vascular tone of resistance vessels.[3] It would seem reasonable to assume that the same mechanisms were operative in obese patients in this study. The magnitude of reduction in PR in supine subjects in this study was less than the magnitude of increase in CO. One would assume that increased $\dot{V}_{O_2}$ in the supine posture contributed to the increase in CO beyond the change that could be attributed to reduction in PR. Pulmonary vascular resistance decreased somewhat in the supine position but the change was not significant. This resulted in a significant increase in PAP.

Increased blood volume in the heart and lungs in the supine position in healthy subjects has been reported, although changes in PAWP or left ventricular filling pressure with change in posture have not, to our knowledge, been documented. In the present study there was a significant increase in PAWP when the supine position was assumed to a mean value of 13.37 torr (approximately 18.2 cm H_2O), a value not appreciably lower than figures obtained in patients with pulmonary congestion.

Changes in $\dot{Q}_s/\dot{Q}_t$ obtained in this study were somewhat expectable on the basis that changes in pulmonary closing volume result in small-airway closure in the supine position.[11] Don *et al.*[11] also found that as the ratio between weight and height increased, CV became a greater proportion of FRC in obese subjects in the supine position. This relationship was not evident in the sitting position. Holley *et al.*[13] also reported abnormalities in regional distribution of pulmonary ventilation and perfusion when expiratory reserve volume (ERV) in obese subjects was less than 0.4 liters in the sitting position. They predicted that subjects who had ERV's of more than 0.4 liters in the sitting position could show similar abnormalities in ventilation distribution if they acquired the supine position, a maneuver known to reduce ERV.

Despite significant increases in intrapulmonary shunting in the supine position, patients in this study did not show corresponding changes in $A\text{-}aD_{O_2}$ or in Pa_{O_2} values. The simultaneous increase in CO was apparently adequate to mask the effects of increased $\dot{Q}_s/\dot{Q}_t$; thus, there was no difference in $A\text{-}aD_{O_2}$ or Pa_{O_2} values obtained in the supine and upright positions.

Increased $\dot{V}_{O_2}$ occurring in the supine position could be attributed to increased work of breathing due to small-airway closure and increased congestion of the lung, resulting in increased airway resistance and decreased pulmonary compliance. It should be realized that significant cardiopulmonary changes found in this study while the patients were supine were superimposed on marked deviations from normal simply due to severe obesity.[17] Cardiac output and total blood volume are significantly increased in the obese,[4,5] the increase in CO being correlated with body weight in excess of ideal weight. Elastic mechanical work of breathing is also 40–50 per cent greater in obese subjects than in normal individuals.[18] Furthermore, in obese patients the oxygen cost of breathing increases more than mechanical

work of breathing,[8,18,19] denoting decrease in the calculated efficiency of the respiratory muscles. Respiratory system compliance is also reduced in obese subjects, with significant negative correlation between compliance and weight.[20]

We believe that the key change in the supine posture was the ability of the heart to respond to the increased preload and increased work of breathing by increasing output. This response minimized the rise in filling pressure and resulting pulmonary congestion and also essentially masked the effects of increasing intrapulmonary shunting on arterial oxygenation.[21] It would seem reasonable to postulate that in patients in whom cardiac reserve was inadequate to meet the increased work of breathing or the shift of blood to central locations, appropriate increases in cardiac output might not occur, with clinical evidence of pulmonary congestioin and hypoxemia. Hypoxemia would in turn lead to reduced responsiveness of the central respiratory control mechanisms, leading to hypoventilation, hypercarbia and acidosis. These changes would subsequently increase pulmonary vascular resistance, increase extravascular water, decrease compliance, and further increase work of breathing, resulting in a vicious circle.

Several clinical implications can be drawn from this study. Don *et al.*[22] reported that FRC was reduced in spontaneously ventilating patients during anesthesia. This was associated with increases in airway closure and volume of trapped gas. Intra-abdominal operations were found to result in further reduction in FRC, probably due to limitation of diaphragmatic movements by packs, manipulations, and retraction.[23] One would assume that such changes could result in more small-airway closure and further increase in the work of breathing in supine obese patients undergoing major abdominal operations. Mechanical ventilation using appropriate ventilatory patterns[24] could minimize work of breathing, improve oxygenation through improving the CV-to-FRC ratio, and probably reduce the tendency towards pulmonary congestion found in this study when the patients were supine. In the postoperative period, pain from the surgical incision, especially in upper abdominal procedures, was reported to lead to further reduction in FRC and to cause hypoxemia, probably due to small-airway closure and alveolar collapse.[25] It would be expected that obese patients could also benefit from mechanical assistance of ventilation during the immediate postoperative period.

The authors thank Drs. Edward Mason and Kenneth Printen, Department of Surgery, University of Iowa, College of Medicine, for permission to study their patients.

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APPENDIX

1. $$CO = \frac{\dot{V}_{O_2}(STPD)}{(Ca_{O_2} - C\bar{v}_{O_2})} \times \frac{1}{10}$$

2. $$\dot{Q}_S/\dot{Q}_T = \frac{Cc_{O_2} - Ca_{O_2}}{Cc_{O_2} - C\bar{v}_{O_2}}$$

where

$$Ca_{O_2} = [(Pa_{O_2} \times 0.0031) + (Hb \times 1.38 \times Sa_{O_2})]$$

$$C\bar{v}_{O_2} = [(P\bar{v}_{O_2} \times 0.0031) + (Hb \times 1.38 \times S\bar{v}_{O_2})]$$

$$Cc_{O_2} = [(PA_{O_2} \times 0.0031) + (Hb \times 1.38 \times 1)]$$

assuming that:

a. PA_{O_2} = O_2 tension in pulmonary capillary blood

$= P_B - (Pa_{CO_2} + PA_{H_2O})$

b. 1.38 = O_2 content in ml per gram Hb at full saturation

0.0031 = O_2 solubility coefficient in 100 ml blood

3. $$PR = \frac{MAP}{CO}$$

4. $$PVR = \frac{PAP - PAWP}{CO}$$