Pattern of Oxygenation during Intestinal Bypass for Morbid Obesity

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In the awake, supine, obese individual, the closing volume of the lung is often less than the functional residual capacity.[1] In an anesthetized, paralyzed and mechanically ventilated subject, the diaphragm shifts cephalad, further decreasing functional residual capacity, and ventilation redistributes preferentially to the nondependent parts of the lung.[2,3] Thus, a greater A-aD_{O_2} occurs in the anesthetized obese subject when ventilation is controlled.

This study documents a transient arterial blood oxygen desaturation during jejunoileal bypass for morbid obesity. The decrease in Pa_{O_2} occurred after the abdominal wall was retracted. In some cases, the Pa_{O_2} approached hypoxic levels.

MATERIALS AND METHODS

Twenty-three consecutive patients who underwent jejunoileal bypass for morbid obesity were studied. The physical characteristics of the five male and 18 female patients are presented in table 1. The criteria for selection of the patients for operation were those of Payne and DeWind[4]: 1) weight more than 45.2 kg above ideal weight; 2) failure of medical and psychiatric therapy to alleviate obesity; 3) absence of correctable endocrinopathy; 4) willingness of patients to participate in the study and cooperate for prolonged follow-up visits; 5) complications of obesity.

TABLE 1. Physical Characteristics of 23 Patients (5 Male, 18 Female) Studied during Jejunoileal Bypass

	Mean ± SE	Range
Age (years)	32.5 ± 0.7	22–49
Height (cm)	167.0 ± 1.9	151.8–182.9
Weight (kg)	143.8 ± 7.5	107.3–250.0
Excess weight/ideal weight (per cent)	143.3 ± 7.9	94.3–221.4

All our patients were ambulatory and in no acute distress. However, ten patients had elevated systolic blood pressures; in two systolic pressures were above 180 torr. Five patients had elevated diastolic pressures. Eight patients were hyperglycemic, and two of the eight were being treated with orally administered hypoglycemic drugs. Two patients had thrombophlebitis of the legs and one patient had degenerative arthritis of the lumbar spine.

The patients were premedicated with combinations of morphine sulfate 8–12 mg, pentobarbital 100–150 mg, and atropine or scopolamine, 0.4–0.6 mg, administered im 1 to 1½ hours prior to induction of anesthesia. Anesthesia was induced and maintained with intravenously administered thiopental and narcotics, and a 50 per cent mixture of nitrous oxide and oxygen. Endotracheal intubation was facilitated with succinylcholine, 100–120 mg, iv. The electrocardiogram and body temperature were monitored continuously. Muscle relaxation during the operation was provided by *d*-tubocurarine or pancuronium bromide. Following endotracheal intubation, the patient was connected to a mechanical ventilator set to deliver a tidal volume of 15 ml/kg ideal body weight. The ventilator frequency was adjusted to obtain a Pa_{CO_2} of approximately 35 torr. Tidal volume was monitored with a Wright respirometer. Inspiratory time of the ventilator was usually set at maximum.

A radial artery was cannulated in every patient for arterial blood sampling and pres-

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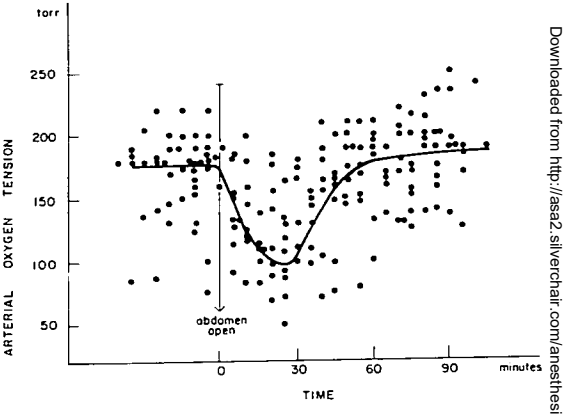

FIG. 1. The line was drawn visually through the mean values of Pa_{O_2}. A decrease in Pa_{O_2} occurred when the abdominal wall was retracted.

sure monitoring. Arterial blood samples were obtained every 10 minutes and analyzed for Pa_{O_2}, Pa_{CO_2}, and pH.

Results

One patient who had thrombophlebitis of the leg had severe wheezing after induction of anesthesia and suffered multiple pulmonary emboli in the postoperative period. This patient was excluded from the review.

In 17 of the 22 patients (77 per cent), Pa_{O_2}'s transiently decreased after the abdomen was opened and the Balfour retractor was applied. The Pa_{O_2}'s (FI_{O_2}, 0.5) before application of the retractor ranged from 104 to 220 torr, with a mean of 173 ± 8 torr. The lowest Pa_{O_2}'s occurred 25 ± 6 minutes after the abdomen was opened. These ranged from 52 to 165 torr, with a mean of 112 ± 8 torr ($P < 0.01$). The duration of desaturation was 63 ± 19 minutes, and Pa_{O_2} returned to 187 ± 6 torr (fig. 1). The mean weight of the patients in this group was 131.5 ± 4.7 kg, and the mean excess weight over the ideal body weight 131.1 ± 6.3 per cent.

In five patients, opening the abdomen or application of the retractor did not influence Pa_{O_2}. Of these, the two heaviest patients had the lowest Pa_{O_2}'s. The initial Pa_{O_2} of 155 torr in one patient weighing 230.9 kg (213.6 per cent overweight) decreased steadily over a period of 50 minutes and stabilized at 91 torr. The initial Pa_{O_2} of 75 torr in the second patient, weighing 250.0 kg (226.6 per cent overweight), did not change throughout the procedure. The last three patients, weighing 143.1 ± 4.8 kg (171.0 ± 12.4 per cent overweight) had a mean Pa_{O_2} of 155 ± 29 torr.

Discussion

The change in the functional residual capacity may explain the transient decreases in Pa_{O_2} that occurred in our patients when the abdomen was opened and the retractor inserted. The position of the diaphragm is determined by the balance of forces across the diaphragm. In anesthetized, paralyzed and mechanically ventilated subjects, the forces acting cephalad are intra-abdominal pressure and elastic recoil of the lung, and the force acting caudad is airway pressure. In supine obese subjects, intra-abdominal pressure is increased, presumably secondary to the weight of the thick abdominal wall, and the functional residual capacity is decreased. It seems reasonable to assume that when the high intra-abdominal pressure is relieved by retracting the abdominal wall, the functional residual capacities of morbidly obese subjects increase to that of non-obese subjects. Such

expansion of the lung volume can be accompanied by opening of the silent alveolar–capillary units, thus creating temporarily hypoventilated but perfused alveoli or actual venous admixture. Although this could account for the transient nature of the observed Pa_{O_2} change in 17 patients, we are unable to explain why five of our significantly heavier patients did not show the same Pa_{O_2} pattern.

The changes in monitored circulatory variables were minimal and variable when the abdomen was opened. The low-cardiac-output state, however, could not be ruled out as a cause for the Pa_{O_2} change, as cardiac output was not measured. Similarly, transient pulmonary congestion remains a possibility.

The average Pa_{O_2}'s in individual patients varied widely. Our finding, the transient decrease of Pa_{O_2}, further indicates that the monitoring of arterial blood gases is essential for adequate oxygenation of morbidly obese patients undergoing laparotomy.

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Prevention of Spinal Hypotension Associated with Cesarean Section

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Maternal hypotension during spinal anesthesia for cesarean section is a persistent problem. Without preventive measures, the incidence of hypotension (defined as a decrease in systolic pressure to less than 100 torr) can rise as high as 82 per cent,[1] and there is evidence that fetal deterioration can result.[2,3]

Various methods are used in the treatment and prevention of spinal hypotension. Administration of a vasopressor before[1,2] or after administration of spinal anesthesia has been utilized. Greiss and Crandell[4] have demonstrated that intravenous fluid administration improves uterine blood flow. Phenylephrine, although effective in returning maternal pressure to normal, is ineffective in improving uterine blood flow. The pre-spinal infusion of a liter of lactated Ringer's solution has been shown to prevent a decrease in maternal mean blood pressure.[5] Finally, several devices and methods have been advocated to displace the uterus off the inferior vena cava (Colon-Morales,[6] Kennedy,[7] Finley,[8] Eckstein and Marx,[9] etc.[10–12]) to reduce hypotension by improving venous return.

We have studied the effects of fluid loading and left uterine displacement in the prevention of hypotension under three conditions: 1) no fluid loading or left uterine displacement prior to administering spinal anesthesia; 2) fluid loading only; 3) both fluid loading and left uterine displacement by means of a "Sluder" (sustained left uterine displacer[7]).

Materials and Methods

ASA class I patients who were at or near term and had agreed to the use of spinal anesthesia for cesarean section were studied, indications being either repeat cesarean sec-

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