ELEVATED PULMONARY ARTERY SYSTOLIC STORAGE IN MORBID OBESITY

Charles Her MD*, Mosses Bairamian MD, Thomas Cerabona MD and Yuqun Pan MD
Department of Anesthesiology, New York Medical College, Valhalla, NY, United State

Introduction: The possibility that the increased pulmonary blood volume (PBV) due to the increased total body blood volume in morbidly obese patients can elevate pulmonary artery (PA) systolic storage (SS) was examined. SS is fraction of stroke volume stored in PA during systole. The elevated SS can augment the diastolic pulmonary capillary blood flow (PCBF), which can then increase the capillary blood volume participating gas diffusion. The increased PBV contributes to the pulmonary overperfusion with recruitment of capillaries, and then to the distension of PA. Since the distension of PA accounts for SS, the increased PBV in obesity should elevate SS. We examined this possibility by measuring SS at two different PBV in obese patients. Changes in PBV should correlate with changes in SS. If so, the elevated SS in morbid obesity may counteract the adverse effects of increased PBV on the distribution of ventilation-to-perfusion.

Method: We used N_2O-airway-pneumotachographic method, to measure the instantaneous PCBF from which SS was measured. During end-expiratory pause or short apneic period after breaths of either air (or oxygen) or N_2O, the pulmonary capillary pulsation produces a pulsatile gas flow, which can be measured with a pneumotachograph. With inhalation of N_2O, the uptake of N_2O by the pulmonary capillary blood attenuates the pulsatile gas flow. Subtraction of gas flow tracing during apnea after inhalation of N_2O from that after air or oxygen produces a pulsatile waveform of PCBF pulse. Since SS is the same as the flow volume of PCBF during diastole, SS was determined from the flow volume during diastole divided by the flow volume during a whole cardiac cycle. A phonocardiogram was used to define end-systole and -diastole. Included were 12 morbidly obese patients undergoing laparoscopic or open gastric bypass surgery under general anesthesia, with the approval of IRB. The baseline measurement was done right after induction before hydration was begun. When the peritoneum was open or pneumoperitoneum was established, N_2O was discontinued. The measurement was repeated when the peritoneum was closed and N_2O was restarted. By this time, patients were adequately hydrated as assessed by pulmonary capillary wedge pressure (PCWP) and urine output. For the gas flow measurements, the side arm spirometry tubing was redirected from the spirometer monitor to pneumotachograph without disconnecting any part of airway or breathing circuit. Cardiac output was determined by thermodilution technique.

Results: The mean baseline SS was .53 ± .075 at the mean PCWP of 12 ± 2 mmHg and stroke volume index of 45 ± 10 ml. In all cases, SS was elevated at the higher PBV. The mean SS was increased to .67 ± .068 at the mean PCWP of 17 ± 2 +mmHg and stroke volume index of 49 ± 4 ml. Mean body mass index was 53 ± 7.7.

Conclusion: Our data indicate that the high PBV in morbidly obese patients is associated with the elevated SS. Since an increase in SS is associated with improved overall ratio of ventilation-to-perfusion, the elevated SS in obesity should counteract adverse effects of the high PBV on distribution of ventilation-to-perfusion.