

Hypoxemia and Hypoventilation Syndrome Improvement after Laparoscopic Bariatric Surgery in Patients with Morbid Obesity

FRANCO LUMACHI¹, BERNARDO MARZANO², GIOVANNI FANTI², STEFANO M.M. BASSO²,
FRANCESCO MAZZA³ and GIORDANO B. CHIARA²

¹Department of Surgical and Gastroenterological Sciences,
University of Padua, School of Medicine, 35128 Padova, Italy;

²U.O. Chirurgia I and ³U.O. Pneumologia, S.M. degli Angeli Hospital, 33170 Pordenone, Italy

Abstract. *Background:* The objective of this study was to evaluate the relationship between oxygen partial pressure (pO_2), awake oxymetric saturation (SpO_2), body mass index (BMI), and percentage of excess weight loss (EWL) in extremely severe obesity ($BMI > 50 \text{ kg m}^{-2}$) and hypoxemia, before and after laparoscopic Roux-en-Y gastric bypass. *Patients and Methods:* A group of 11 obese patients aged 41.2 ± 10.2 years (4 men, 7 women, median $BMI = 52.3 \text{ kg/m}^2$, range 50.2-57.1) were prospectively enrolled in the study. BMI, arterial blood gas measurements, and spirometry were obtained before and after (6 and 12 months) surgery. *Results:* The main preoperative parameters were $SpO_2 = 88.3 \pm 3.9\%$, predicted forced vital capacity (FVC) = $84.5 \pm 8.3\%$, predicted forced expiratory volume exhaled in one second (FEV1) = $79.9 \pm 10.1\%$. No relationship ($p > 0.01$) was found between BMI, SpO_2 , and FEV1. A significant correlation between SpO_2 and both paO_2 ($R = 0.74$, $p = 0.009$) and EWL ($R = -0.75$, $p = 0.008$) was found. Three, 6, and 12 months after surgery EWL was 18.9%, 26.4%, and 39.6% ($p < 0.001$), respectively. At one-year follow-up SpO_2 , FVC, and FEV1 were $96.2 \pm 3.2\%$ ($p < 0.001$), $112.3 \pm 9.9\%$ ($p < 0.001$), and $101.6 \pm 18.8\%$ ($p = 0.003$), respectively. *Conclusion:* In patients with extremely severe obesity, bariatric surgery may improve significantly both SpO_2 and spirometric parameters, and EWL represents the factor that impacted the results.

Correspondence to: Professor Franco Lumachi, Department of Surgical and Gastroenterological Sciences, University of Padua, School of Medicine, 35128 Padova, Italy. Tel: +39 0498211812, Fax: +39 0498214394, e-mail: flumachi@unipd.it

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Obesity is a disorder of body composition, defined by a relative or absolute excess of body fat (1).

The prevalence of obesity has increased dramatically in the last decades, occurring in about 25% of adult men and 30% of adult women in the USA (1, 2). Respiratory comorbidity (*i.e.* obesity-hypoventilation syndrome (OHS)) represents a common complication in morbidly obese patients, and when patients lose weight a rise in oxygen partial pressure (pO_2), and a fall in dioxide partial pressure (pCO_2) are usually observed.

The aim of this study was to evaluate the relationship between baseline and postoperative pO_2 , awake pulse oxymetric saturation (SpO_2), body mass index (BMI), and percentage of excess weight loss (EWL) in patients with extremely severe obesity and hypoxemia ($pO_2 < 75 \text{ mm Hg}$), before and after bariatric surgery.

Patients and Methods

Eleven extremely obese patients ($BMI > 50$) undergoing laparoscopic Roux-en-Y gastric bypass were enrolled in the study. The gastric bypass combines the creation of a small gastric pouch with bypassing a portion of the upper small intestine (3). Additional modifications resulted in the Roux-en-Y gastric bypass, a now common operation that involves stapling the upper stomach into a 30-mL pouch and creating an outlet to the downstream small intestine (4).

There were 4 men and 7 women (median $BMI = 52.3 \text{ kg/m}^2$, range 50.2-57.1) aged 41.2 ± 10.2 years. In all patients, preoperative and postoperative measurements of BMI, SpO_2 , predicted forced vital capacity (FVC), and predicted forced expiratory volume exhaled in one second (FEV1) were performed. Informed consent was obtained from all participants, in accordance with institutional review board approval.

The reported data are expressed as mean \pm standard deviation (SD). The Pearson's correlation coefficient (R) calculation was used to evaluate the linear relationship between pairs of variables. Comparisons between groups were performed using the Mann-

Whitney *U*-test and the Student's *t*-test. A *p*-value <0.01 was considered statistically significant.

Results

The main preoperative parameters were the following: SpO₂=88.3±3.9%, FVC=84.5±8.3%, FEV1=79.9±10.1%. No relationship was found between BMI and SpO₂ (R=0.06, *p*=0.86), FVC (R=0.26, *p*=0.43), or FEV1 (R=0.18, *p*=0.59). A significant correlation between age, SpO₂ (R=0.75, *p*=0.007), FVC (R=-0.95, *p*<0.001), and FEV1 (R=-0.96, *p*<0.001), and between SpO₂ and both pO₂ (R=0.74, *p*=0.009) and EWL (R=-0.75, *p*=0.008) was found.

Three, 6, and 12 months after surgery, EWL was 18.9%, 26.4%, and 39.6% (*p*<0.001), respectively. At one-year follow-up SpO₂, FVC, and FEV1 were 96.2±3.2% (*p*<0.001), 112.3±9.9% (*p*<0.001), and 101.6±18.8% (*p*=0.003), respectively.

Discussion

OHS describes the hypoventilation observed in severe obese patients when other causes (*i.e.* chronic lung or respiratory muscle diseases) are absent (5). It represents a heterogeneous group of disorders with differing clinical manifestations, such as obstructive sleep apnea, and chronic daytime hypoventilation.

Ventilatory responsiveness is attenuated in patients with OHS (6, 7). Respiratory muscle strength is reduced in obese patients with OHS, and following significant weight loss, both respiratory muscle performance and lung volumes improve, along with normalization of hypoxemia (3, 8). Patients with OHS should have daytime hypoxia (PO₂<70 mm Hg) and hypercapnia (PCO₂>45 mm Hg) (9). Moreover, the severity of obesity and the associated changes in lung function play an important role in the pathogenesis of pulmonary hypertension (10, 11).

The mechanism of hypoxemia and hypercapnia in obese patients with OHS are shown in Figure 1. The role of leptin is unclear. Leptin is a hormone produced in adipose cells that suppresses appetite through interaction with its receptor in hypothalamus (12). Hypoxic suppression of leptin production, and a central nervous system leptin resistance in patients with OHS have been suggested, but leptin is more likely a respiratory modulator or an epiphenomenon of obesity (13-15).

About 50% of obese patients scheduled for bariatric surgery have associated respiratory comorbidity, such as obstructive sleep apnea syndrome, OHS, and overlapping chronic obstructive pulmonary disease (16-18). In our series, all patients had hypoxemia, and reduced FVC and FEV1. At one year follow-up, SpO₂, FEV and FEV1 improved significantly (*p*<0.01), and a relationship between EWL and

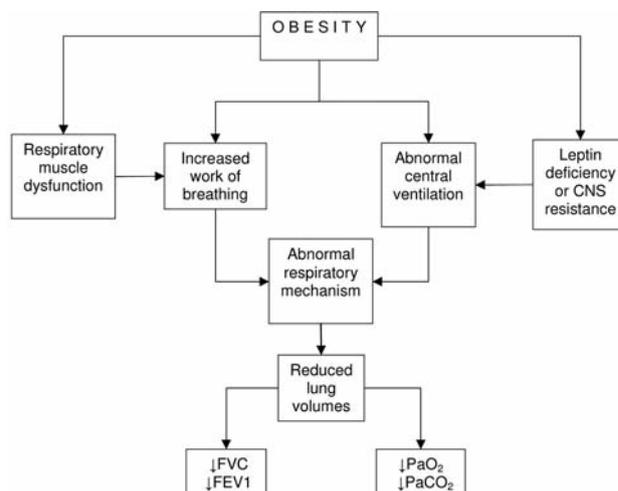


Figure 1. Mechanism of hypoxemia and hypercapnia in obese patients with obesity hypoventilation syndrome. CNS=Central nervous system, FVC=predicted forced vital capacity, FEV1=predicted forced expiratory volume exhaled in one second, PaO₂=arterial oxygen partial pressure, PaCO₂=arterial carbon dioxide partial pressure.

both SpO₂ and pO₂ was found. In several studies, after Roux-en-Y gastric bypass patients presented significant weight loss, and improvement of hypoxemia, hypercarbia and in spirometric results (18-21). However, neither number of weight loss attempts nor the maximal preoperative weight loss correlate with the percentage of excess weight loss after laparoscopic Roux-en-Y gastric bypass surgery (22).

In conclusion, in patients with extremely severe obesity, bariatric surgery may improve significantly both SpO₂ and spirometric parameters, and EWL represents the factor that impacted results.

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References

- Hellerstein MK and Parks ET: Obesity and Overweight. *In*: Basic and clinical Endocrinology. Greenspan FS and Gardner DG (eds.). New York, NY, Lange Medical Books/McGraw-Hill: pp. 745-761, 2001
- Flegal KM, Carroll MD, Ogden CL and Johnson CL: Prevalence and trends in obesity among us adults, 1999-2000. *JAMA* 288: 1723-1727, 2001.
- Piper AJ and Grunstein RR: Current perspectives on the obesity hypoventilation syndrome. *Curr Op Pulm Med* 13: 490-496, 2007.

- 4 Mason EE and Ito C: Gastric bypass in obesity. *Surg Clin North Am* 47: 1345-1351, 1967.
- 5 Maggard MA, Shugarman LR, Suttorp M, Maglione M, Sugerma HJ, Livingstone EH, Nguruyen NT, Li Z, Mojica WA, Hilton L, Rhodes S, Morton SC and Schkelle PG: Meta-analysis: surgical treatment of obesity. *Ann Intern Med* 142: 547-559, 2005.
- 6 Berger KI, Ayappa I, Chatr-Amontri B, Marfatia A, Sorkin IB, Rappaport DM and Goldring RM: Obesity hypoventilation syndrome as a spectrum of respiratory disturbances during sleep. *Chest* 120: 1231-1238, 2001.
- 7 Chouri-Pontarollo N, Borel J-C, Tamisier R, Wuyam B, Levy P and Pèpin JL: Impaired objective daytime vigilance in obesity-hypoventilation syndrome. *Chest* 131: 148-155, 2007.
- 8 Weiner P, Waizman J, Weiner M, Rabner M, Magadle R and Zamir D: Influence of excessive weight loss after gastroplasty for morbid obesity on respiratory muscle performance. *Thorax* 53: 39-42, 1998.
- 9 Kessler R, Chaouat A, Schinkewitch P, Faller M, Casel S, Krieger J and Weitzenblum E: The obesity-hypoventilation syndrome revisited: a prospective study of 34 consecutive cases. *Chest* 120: 369-376, 2001.
- 10 Bady E, Achkar A, Pascal S, Orvoen-Frija E and Laaban JP: Pulmonary arterial hypertension in patients with sleep apnoea syndrome. *Thorax* 55: 934-939, 2000.
- 11 Valencia-Flores M, Orea A, Herrera M, Santiago V, Rebollar V, Castano VA, Oseguera J, Pedroza J, Sumano J, Resendiz M and Garcia-Ramos G: Effect of bariatric surgery on obstructive sleep apnea and hypopnea syndrome, electrocardiogram, and pulmonary arterial pressure. *Obes Surg* 14: 755-762, 2004.
- 12 Styne D: Puberty. In: Basic and Clinical Endocrinology. Greenspan FS and Gardner DG (eds.). Lange Medical Books/McGraw-Hill. New York, NY, pp. 547-574, 2002.
- 13 Caro JF, Kolaczynski JW, Nyce MR, Ohannesian JP, Opentanova I, Goldman WH, Lynn RB, Zhang PL, Sinha MK and Considine RV: Decreased cerebrospinal-fluid/serum leptin ratio in obesity: a possible mechanism for leptin resistance. *Lancet* 348: 159-161, 1996.
- 14 Redolfi S, Corda L, La Piana G, Spandrio S, Prometti P and Tantucci C: Long-term noninvasive ventilation increases chemosensitivity and leptin in obesity-hypoventilation syndrome. *Respir Med* 101: 1191-1195, 2007.
- 15 Powers MA: The obesity hypoventilatory syndrome. *Respir Care* 53: 1723-1730, 2008.
- 16 Waldhron RE: Nocturnal nasal intermittent positive pressure ventilation with bi-level positive airway pressure (bipap) in respiratory failure. *Chest* 101: 516-521, 1992.
- 17 Collighan NT and Bellamy C: Anhaesthesia for the obese patients. *Curr Anaest Crit Care* 12: 261-266, 2001.
- 18 Martí-Valeri C, Sabaté A, Nasdevall C and Dalmau A: Improvement of associated respiratory problems in morbidly obese patients after open roux-en-y gastric bypass. *Obes Surg* 17: 1102-1110, 2007.
- 19 Balsiger BM, Kennedy FP, Abu-Lebdeh HS, Collazo-Clavell M, Jensen MD, O'Brien T, Hensrud DD, Dinneen SF, Thompson GB, Que FG, Williams DE, Clark MM, Grant JE, Frick MS, Mueller RA and Sarr MG: Prospective evaluation of roux-en-y gastric bypass as primary operation for medically complicated obesity. *Mayo Clin Proc* 75: 74-79, 2000.
- 20 Dhabuwala A, Cannan RJ and Stubbs RS: Improvement in comorbidities following weight loss from gastric bypass surgery. *Obes Surg* 10: 428-435, 2000.
- 21 Guardiano SA, Scott JA, Catesby Ware J and Schechner SA: The long-term results of gastric bypass on indexes of sleep apnea. *Chest* 124: 1615-1619, 2003.
- 22 Jantz EJ, Larson CJ, Mathiason MA, Kallies KJ and Kothari SN: Number of weight loss attempts and maximum weight loss before roux-en-y laparoscopic gastric bypass surgery are not predictive of postoperative weight loss. *Surg Obes Relat Dis* 5: 208-211, 2009.

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