The Morbidly Obese Pregnant Woman

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Obesity in women of childbearing years is increasing exponentially. Obstetric and anesthesia management of morbidly obese pregnant women poses a great challenge to both obstetricians and anesthesiologists alike, because obesity is a significant risk factor for adverse perinatal outcome mediated through increased risk of hypertension, diabetes, coronary disease, respiratory dysfunction, and thromboembolic complications. Equally important is the effect of maternal overweight on the pregnancy outcome. Obesity increases antepartum stillbirths, especially at term. It is estimated that 34 million adults in the United States are overweight to a significant degree. These patients require communication and cooperation of the entire health care team to prevent catastrophic complication and improve outcome.

The purpose of this chapter is to define and quantify obesity, describe the pathologic alterations in obesity, outline the influence of physiologic changes of pregnancy on the pathologic alterations of obesity, and elucidate obstetric and anesthetic management of morbidly obese parturient.

Definition and Prevalence of Obesity

There is disagreement about a precise definition of obesity, but in general terms a person must be considered obese when the amount of fat tissue is increased beyond the point compatible with physical and mental health and normal life expectancy. The most important health risks of obesity are distinctly related to excessive weight, with a progressive and disproportionate increase as the patient’s weight rises. A 10% overweight carries an excess mortality of 33% and a 20% or more overweight a 50% excess mortality. At a weight of 60% above standard insurance tables, disease risk is doubled as compared with the general population. However, this excess mortality risk appears to be more pronounced in men compared to women. Given that there is a positive relationship between excess weight and morbidity, it becomes important to quantify obesity and its gradations. Some define morbid obesity as a doubling of the person’s predicted ideal weight. Ideally, an index of obesity should be independent of height, muscularity, and skeletal mass; it should reflect fatness only. Body mass index (BMI) is defined as weight (kg)/height$^2$(m). Normal values range between 22 and 28 in the United States. A BMI greater than 28 for women is considered obese and greater than 35 to 40 as morbidly obese. Weight greater than 300 pounds in a gravida at term is also considered to be morbidly obese. There are other ways of defining morbid obesity. Bendexin stated that a morbidly obese individual is one who weighs twice the predicted weight for sex, age, and height as prescribed in the Metropolitan Life Insurance Company table. Garbaciak et al. thought that the definition should be reserved for parturients who were 150% above ideal body weight. A simple method of identifying this group of women is to state every pregnant patient who is 100 pounds overweight is morbidly obese. Mason et al. described obese pregnant women who exceed 225% of ideal body weight as super obese.

The prevalence of obesity varies depending on the definition, the criteria used, and the cultural and economic area studied. In the United States, a National Institute of Health consensus defined obesity as 20% above relative weight; by this criterion, 30% to 40% of women are obese.

Pathophysiology

Cardiovascular Changes

Nonpregnant, morbidly obese, normotensive persons have increased pre- and afterload, increased mean pulmonary artery pressure, and elevated right and left ventricular stroke work. The total peripheral resistance is slightly decreased in normotensive obese persons. The cardiac diameter is usually increased by 20% to 55%, and the ventricles are hypertrophied with increased cardiac weight. Cardiac output is increased due to larger stroke volume. Increments in cardiac output are well correlated to weight and proportional to the increasing oxygen consumption that is associated with
obesity. Left ventricular function, as measured by the left ventricular systolic work/pulmonary artery wedge pressure (LVSW/PAWP) ratio, is about 57% of normal in asymptomatic obese persons. In response to exercise, cardiac output (CO) rises faster in the morbidly obese than in normal persons and is often associated with a rise in left ventricular end-diastolic pressure and pulmonary capillary wedge pressure. Hypertension is significantly correlated with obesity, and an increased systolic pressure of 3 mm Hg and a rise in diastolic pressure of 2 mm Hg is associated with every 10-kg increase in body weight. A body mass index greater than 30 is associated with a threefold increase in the incidence of hypertension. The total blood volume is increased in obese persons, but on a volume/weight basis it is decreased, compared to lean persons (50 versus 75 mL/kg body weight). Most of the expanded volume is distributed in the adipose tissue. Splanchnic blood flow is about 20% higher, whereas cerebral and renal blood flow is found to be normal. A 30-fold increase in premature ventricular contractions is seen in obese patients with eccentric left ventricular contractions when compared with lean subjects. Left ventricular hypertrophy (LVH) occurs in response to increased workload. However, there is an association between increased left ventricular mass and increased weight, even after controlling for age and blood pressure, in patients with BMI greater than 30. Excess pericardial fat is not a prominent feature in obese patients with cardiac enlargement. Fatty infiltration of the heart can occur, especially in the right ventricle and perhaps in the conduction system. Eccentric LVH is the major cause of increased heart size in obesity. Inadequate hypertrophy and chamber size may predispose patients to myocardial decompensation. The hemodynamic and respiratory changes seen in morbidly obese persons upon change of posture have been examined in 11 patients before upper abdominal surgery. Change of posture from sitting to supine position was accompanied by a significant increase in CO, cardiac index (CI), PAWP, and mean pulmonary artery pressure, and a significant decrease in heart rate and peripheral resistance. There were no differences in pulmonary vascular resistance (PVR) or mean arterial pressure (MAP). Hypoxemia, if present, causes increased pulmonary vascular resistance. Airway obstruction may also increase pulmonary artery pressure. It was observed that a decline in PAWP occurred from 38 to 5 mm Hg on relieving airway obstruction in an obese patient.

Respiratory Changes

Respiratory changes in morbid obese persons can be differentiated into mechanical, pulmonary, and airway changes.

Mechanical Changes

Total respiratory compliance is decreased mainly due to decreased chest wall compliance and, to lesser extent, decreased lung compliance. The reduction in chest wall compliance is caused by fat accumulation around the ribs, under the diaphragm, and intraabdominally. The increase in pulmonary blood volume is responsible for the decrease in lung compliance.

Pulmonary Changes

Reduced respiratory compliance and mass loading of the lungs results in reduction of functional residual capacity (FRC) caused by a decreased expiratory reserve volume (ERV). A progressive reduction in ERV is seen when the person is brought from a standing position via the seated position to the supine and, further, to the Trendelenburg position. This movement causes a FRC reduction, which may fall within the closing volume and lead to airway closure (gas trapping), especially in dependent lung regions. A ventilation/perfusion mismatch thereby develops, which gives rise to the low PaO2 values seen in many morbidly obese persons.

Bodell differentiated simple gross obesity and the Pickwickian (obesity-hypoventilation) syndrome by classifying obese individuals into three relatively distinct groups according to pulmonary function: 1. Patients with normal arterial saturation but with reduced expiratory capacities 2. Patients with no lung disease who have arterial hypoxemia 3. Patients with intrinsic lung disease with hypoxemia and hypocarbia as a result of alveolar hypoventilation

Relatively young morbidly obese patients can adjust to their hypoxemia with an increase in CO and by the development of polycythemia. With the passage of time, carbon dioxide may be retained and somnolence may ensue. Pulmonary hypertension may occur from the increased blood volume and hypoxic vasoconstriction, and thus cor pulmonale develops. This “Pickwickian” syndrome, as described by Burnwell et al., represents the end stage of cardiopulmonary disease for the morbidly obese individual, this occurs in about 7% to 10% of massively obese patients.

Airway Changes

Obese patients are likely to develop airway obstruction and apnea during sleep, resulting hypercapnia and hypoxia. Some individuals require a continuous airway positive pressure device to overcome airway obstruction.

Gastrointestinal Changes

It is generally believed that the morbidly obese patient is at increased risk for pulmonary aspiration of gastric contents. Hiatal hernia is more common in obese patients than in nonobese patients. Obese patients have delayed gastric emptying, and often the combination of a low gastric pH (<2.5) and a large volume (>2.5 mL) of gastric juice. In a study that compared gastric contents in obese and nonobese subjects, BMI was 46 ± 8 in the obese subjects and 22 ± 2 in the lean subjects. The groups were similar for age (35 ± 12 years and 40 ±