Obesity is a multi-organ, multi-system disease that enhances the morbidity and mortality of other diseases. The anesthetic management of the obese patient presents the anesthetist with a formidable challenge. In Part I of this review, the author discusses the pathophysiology of obesity. Part II, to be published in the next issue, will present the anesthetic management of the obese patient and suggested management techniques.

Obesity is a disease that is found only in man and in animals kept by man. Despite a widespread knowledge of the medical and surgical problems associated with obesity, obesity remains a common disorder among the civilized nations of the world. Statistics indicate that 10-50% of Americans weigh at least 20-30% more than their ideal or desirable weight based on age, height, and sex. Because obesity enhances the morbidity and mortality of other diseases, it is not surprising that the morbidly obese person rarely lives past 60 years of age.

The first task in discussing obesity is to clinically define obesity and differentiate between overweight, obese, and morbidly obese. By definition an individual is overweight if he or she weighs up to 20% in excess of ideal weight. If an individual weighs in excess of 20% of the ideal weight he or she is considered to be obese. A body weight of two times or more the ideal weight constitutes morbid obesity. Vaughan further defines obesity as a condition that exists when there is more body fat present than there should be normally. Since 15-18% of a male's body weight and 20-25% of a female's body weight is fat, obesity exists in females when 30% or more of body weight is made up of body fat. In males a body weight composed of 25% or more body fat constitutes obesity.

Because of the loose interpretation of these terms among various clinicians, numerous assessment tools have been devised to gauge ideal weight and obesity. Some of these are desirable weight tables derived from insurance statistics; measurements of skinfold thickness; comparisons of weight to height, which tend to under-estimate the degree of obesity; the ponderal index (inches divided by the cube root of pounds), which tends to overestimate obesity; and the Body Mass Index (BMI), which is the best indicator of obesity. The Body Mass Index is easy to compute:

\[
\text{BMI} = \frac{\text{Weight (kilograms)}}{\text{Height}^2 \text{ (meters)}}
\]

- \( \leq 25 \) Non-obese
- \( 26-29 \) Overweight
- \( \geq 30 \) Obese

Figure 1

The Body Mass Index (BMI)
pute and more accurately identifies obesity. Additionally, the Body Mass Index allows for an assessment of risks associated with anesthesia.

A quick and easy guide for use in the clinical setting is the Broca Index (Figure 2). This measurement relates weight in kilograms to height in centimeters, but makes no allowance for sex. By doubling the calculated ideal weight one can determine which patients are morbidly obese.$^{11,12}$ Brodsky$^6$ has modified the Broca Index to allow for sex (Figure 3) based on the premise that females have a higher ratio of fat tissue compared to total body weight.

**Physical problems of the obese**

Morbid obesity can be subdivided into two types based on ventilatory function. Ninety to 95% of morbidly obese patients have normal arterial carbon dioxide (PaCO$_2$) levels and are considered to have simple morbid obesity. The other 5-10% hypoventilate, are hypercarbic independent of intrinsic lung disease, and are considered to have obesity-hypoventilation syndrome (OHS) or Pickwickian Syndrome.$^{1,5,8,10-18}$ Table I defines the clinical features of the Pickwickian Syndrome. It is evident that the primary defect in Pickwickian Syndrome is hypoventilation unrelated to restrictive or obstructive pulmonary disease.

The primary cause of obesity is increased caloric intake as compared to caloric expenditure.$^{1,5,7,14}$ In fact, 95-98% of obesity can be related to the problem of overeating.$^{18}$ Given that obesity is found in civilized nations, there appears to be a cultural-socioeconomic influence. This population generally eats three meals a day plus snacks, and experiences no periodic or insufficient food supplies. This eating pattern, coupled with the unique property of efficient energy storage in human adipose tissue, leads to a prevalence of obesity.$^{1,7,10,16}$

In infancy or early childhood a great multiplication of fat cells occurs. The number of cells that develop is largely influenced by eating habits. A meal pattern guided by social or cultural customs, rather than by appetite, leads to an excess of fat cells.$^{1,10}$ Later in life the number of adipocytes, or fat cells, does not change. Only the size of the fat cell does. This accounts for the rapid return of lost weight often seen in obese patients.$^{19}$

In addition, a tendency toward obesity is a genetic trait that is passed from parent to offspring.$^{1,10,14,16}$ In fact, the offspring of an obese parent will have a 40% chance of also becoming obese, while the offspring of two obese parents stand an 80% chance of becoming obese.$^{10}$

The cerebral cortex ultimately controls human eating behavior. However, psychological influences such as stress, anxiety, loneliness, and a sense of rejection may override the cortical controls and lead to obesity through excessive caloric intake. Thus, in the obese patient, eating may serve as a method of self-reward or a form of release from tension.$^{1,7,10,14}$

Appetite control is one of the functions of the ventromedial and lateral hypothalamus.$^{1,14}$ Therefore, the effects of tumors, trauma, or infection on this control area could lead to increased appetite, caloric intake, and insulin production, leading to an increase in fat deposition and subsequent obesity.

Even though overeating is the major cause of obesity, many patients believe that their condition is the result of a metabolic abnormality.$^7$ To the contrary, no positive metabolic link has been found to be a cause of obesity.$^{1,6,7}$ However, there are some metabolic-endocrinologic conditions,

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**Figure 2**
The Broca Index

Height (cm) − 100 = Ideal weight (kg)

For example: A patient whose height is 64" is 162.5 cm tall. Ideal weight = 162.5 − 100 = 62.5 kg

**Figure 3**
The Modified Broca Index

Male: Height (cm) − 100 = Ideal weight (kg)
Female: Height (cm) − 105

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**Table I**
Clinical features of the Pickwickian Syndrome

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<th>Symptom</th>
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<td>Extreme obesity</td>
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<td>Right ventricular hypertrophy/failure</td>
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<td>Pulmonary hypertension</td>
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listed in Table II, that contribute to the development of obesity.\textsuperscript{7,10,13,14} There is an increased incidence in obese persons of cholelithiasis, hiatus hernia, cirrhosis, renal calculi, osteoarthritis, fractures, varicose veins, and diabetes mellitus in obese persons.\textsuperscript{8,11,12,17} Furthermore, studies have shown that a 20-30\% increase above ideal weight is associated with a 40\% increased risk of death from heart disease and a 50\% increased risk of death from cerebrovascular accident.\textsuperscript{1,6,7,10} Weight in excess of 30\% above ideal weight is associated with a mortality rate two to three times that of a normal patient presenting for surgery, whereas risks are minimal if excess weight is less than 20\% above ideal weight.\textsuperscript{8,17}

These findings correlate well with the calculated Body Mass Index. It has been demonstrated that a body mass index of 26-29 (\leq 30\% overweight) carries minimal risks. A rating greater than 30 (35-40\% overweight) has a significant increase in associated risks.\textsuperscript{8,10,12} The obese patient is at a higher risk for developing coronary artery disease, angina, and hypertension. This causal relationship was demonstrated by the Framingham Study, although the exact mechanism was not clear.\textsuperscript{1,11,12} In fact, 50-60\% of all obese individuals manifest systemic hypertension, while the risk of coronary artery disease is doubled.\textsuperscript{5,8} Obesity increases the metabolic demand to such a degree that an obese patient may need to maintain a cardiac output up to two times that of his ideal weight counterpart.\textsuperscript{1} The metabolic demand is increased because the excess adipose tissue must be perfused. It is estimated that 30 pounds of fat contain 25 miles of blood vessels, or that each kilogram of fat contains 3,000 meters of blood vessels.\textsuperscript{5,10} This leads to an increase in the circulating blood volume, cardiac output, and stroke volume without a change in heart rate.\textsuperscript{5,9-12} These metabolic demands are secondary to the increased oxygen consumption and carbon dioxide production brought about by the increase in body mass.\textsuperscript{5,9,15}

These increased metabolic demands result in an increased workload for the heart, primarily the left ventricle.\textsuperscript{1,5,8} It is estimated that an excess weight of 100 kilograms doubles the cardiac output or increases the cardiac output 0.1 L/minute/kg of adipose tissue.\textsuperscript{4,5} This increase in cardiac output, secondary to an increase in circulating blood volume, leads to an increase in the stroke volume. This in turn leads to an increase in left ventricular work, as evidenced by an increased left ventricular end-diastolic pressure, left ventricular hypertrophy, and eventual failure as cardiac reserve is decreased.\textsuperscript{1,4,5,9} Additionally, to meet the increased oxygen needs of the excess adipose tissue, there is an increase in the number of erythrocytes that improve oxygen transport, furthering the workload of the heart.\textsuperscript{1,11,12} These factors make the obese patient at risk of rapid decompensation. Table III lists the cardiovascular changes observed when the obese patient goes from a sitting to a supine position. Fifty to 60\% of all obese patients manifest systemic hypertension. There is a positive correlation with increased weight and systemic hypertension, which is believed to be due to the increased cardiac output and stroke volume.\textsuperscript{5,10,18} This follows the direct linear relationship of increasing cardiac output as weight increases. Obese patients may also have pulmonary hypertension accompanying the systemic hypertension. This is primarily due to the increase in circulating blood volume, cardiac output, and stroke volume which increase pulmonary blood flow.\textsuperscript{1,4,8,8} Another factor is the hypoxemia and hypercarbia associated with obesity which cause pulmonary vascular constriction and polycythemia.\textsuperscript{8,16}

The increased metabolic rate required to perfuse excess adipose tissue increases oxygen consumption and carbon dioxide production. Alveolar and minute ventilation double to accommodate this increase.\textsuperscript{1,8} In turn, breathing for the obese patient requires greater effort, at the expense of energy and respiratory efficiency.\textsuperscript{1,4,8,12} In fact, about 85\% of all obese patients have some degree

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<td><strong>Cardiovascular changes associated with a position change</strong></td>
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of exertional dyspnea and orthopnea. Some obese patients even exhibit a sleep apnea, or nocturnal hypoventilation.\textsuperscript{5,7,9,12} The increased adipose tissue found on the abdomen and thorax decreases the bellows action of the thoracic cage by causing a certain degree of thoracic kyphosis and lumbar lordosis.\textsuperscript{8,10,19} These anatomic changes, coupled with an elevation of the diaphragm by the abdominal contents and a reduction in efficiency of the respiratory muscles, decrease pulmonary and chest wall compliance, resulting in position-dependent diaphragmatic ventilation.\textsuperscript{1,5,7-9,12}

The decrease in compliance and the position-dependent ventilation leads to a decrease in lung volumes and capacities (Table IV), as evidenced by pulmonary function tests. These decreases in volumes and capacities, particularly the functional residual capacity, lead to hypoxemia without hypercarbia due to early airway closure and shunting with ventilation-perfusion mismatching. These changes are accentuated in the anesthetized patient who is supine and spontaneously breathing. Objectively, the arterial blood gases show a decreased PaO\textsubscript{2} and an increased PaCO\textsubscript{2}. Therefore, obese patients exhibit a restrictive pattern of lung disease.\textsuperscript{5,10,21} Weight loss results in a reversal of these conditions to the point that a decreased PaCO\textsubscript{2}, increased PaO\textsubscript{2}, vital capacity, total lung capacity and overall ventilation occur. The patient even exhibits an enhanced response to inhaled carbon dioxide.\textsuperscript{7}

Diabetes mellitus is the most common disorder associated with obesity, while 2\% of obese patients have other endocrine abnormalities as well. There is a seven-fold increase in the incidence of mature-onset diabetes in the obese patient which increases to ten-fold during pregnancy.\textsuperscript{5,8,10,12-18,10,22,28} Adult diabetics are often obese, with 80-90\% of non-ketotic insulin dependent diabetics having their condition aggravated by obesity.\textsuperscript{7} This not only complicates the anesthetic management of the patient but also contributes to an increase in morbidity. Glucose tolerance tests are usually abnormal in the obese patient but, with weight loss, tend to return to normal.\textsuperscript{5,7,8,10}

In the obese patient there is a resistance to insulin, although the exact mechanism for this resistance is not understood.\textsuperscript{5,8,11} Therefore, a probable cause of diabetes mellitus in the obese patient is an excess insulin production secondary to the insulin resistance that leads to an eventual burnout of the beta cells in the Islet of Langerhans.

Obese patients are more susceptible to liver damage, which is associated with the duration, not the degree, of obesity.\textsuperscript{4,9,11,12,15} Adipose tissue is deposited around the liver, and a liver biopsy is the only way to accurately assess the degree of fatty infiltration, or morphological change, within the liver. In fact, 90\% of obese patients will have fatty infiltration of the liver.\textsuperscript{4,5,7,10-12} This chronic hepatic strain leads to an increased incidence of hepatic cirrhosis resulting in a mortality rate 1.5-2.5 times that of non-obese patients.\textsuperscript{6,11,12,28}

Hepatic lipids, especially triglycerides, accumulate in the obese patient. There is speculation that this is the result of an increased delivery of fatty acids to the liver leading to an increase in synthesis, a decrease in oxidation, and possibly an impaired removal as low-density lipoproteins of fatty acids. The most acceptable reason is an increase in carbohydrate intake when compared to protein intake.\textsuperscript{5,8,11,12} This intracellular accumulation of triglycerides leads to a cellular disruption and the subsequent release of enzymes that are detectable in the serum. Therefore, the liver function tests will be abnormal, showing an increase in serum glutamic oxaloacetic transaminase (SGOT), serum glutamic pyruvic transaminase (SGPT), alkaline phosphatase, and bilirubin.\textsuperscript{5,8,10,11}

In addition, the obese patient has a higher incidence of hypercholesteremia leading to an increased cholesterol turnover rate. This in turn leads to an increased biliary excretion of cholesterol. This predisposes the obese patient to a three-fold increase in the incidence of cholelithiasis.\textsuperscript{5,8,7,12}

Because there is an increased deposition of adipose tissue around all organs, the kidneys are found to be larger in obese patients when compared to non-obese patients. Renal blood flow is normal, or decreased, in obese patients. When coupled with pregnancy there is a five-fold increase in the incidence of pyelonephritis.\textsuperscript{7,10}

As can be seen, the physical status of the obese patient is often quite different from that of a normal weight patient, requiring special considera-

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<td>Lung volume/capacity changes seen in obesity</td>
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tion by the anesthetist. Part II of this article will focus on the assessment and anesthetic management of the obese patient.

REFERENCES

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