Obesity: A Complicating Factor for Sedation in Children

Suher Baker, DMD, BDS, MS[1] John A. Yagiela, DDS, PhD[2]

Abstract

The purpose of this review was to describe the potential influence of childhood obesity on pharmacosedation in pediatric dentistry and provide specific recommendations for managing obese patients. Increasingly common in the United States, childhood obesity poses specific challenges to the dentist. The greatest of these involve the increased potential for respiratory complications because of fat-induced restrictive lung disease and obstructive sleep apnea. Cardiovascular complications associated with obesity alone are rare in the pediatric patient, although hypertension is more likely. Gastrointestinal problems include increased likelihood for aspiration, necessitating strict fasting requirements. Sedative drugs dosed on total body weight may oversedate obese patients; dosages based on lean body mass may undersedate and usually produce a decreased duration of effect. Extra precautions regarding drug selection (such as avoiding opioids) and proper patient positioning can help minimize the incidence of complications. (Pediatr Dent 2006;28:487-493)

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Pharmacosedation is a major adjunct for behavioral management in 15% to 20% of pediatric dental patients.[1] Several hundred thousand children receive oral sedation for dentistry each year in the United States; similar numbers are administered parenteral sedation or general anesthesia. Benefits of pharmacosedation include the provision of comprehensive dental care to patients who would otherwise not receive it and a reduction in dental fear that would otherwise complicate future care. Risks of pharmacosedation include various adverse reactions (eg, vomiting, aspiration, respiratory depression, and cardiac arrhythmias) and even death.

Although the incidence of mortality with pediatric sedation is unknown, estimates of fatal outcomes made 2 decades ago extended from 1:100,000 with opioid analgesics to lower numbers with some nonopioids.[2] Current mortality rates associated with general anesthesia may be as low as 1:250,000,[3,4] which is a dramatic improvement over the 1:1,560 rate of a half century ago.[5]

The dental literature documents a significant association of adverse incidents with sedation in pediatric dentistry.[6-8] Reported nonfatal events range from temporary inconveniences (nausea, delayed recovery) to permanent brain injury. The major cause of serious negative outcomes is hypoxia. Inadequate ventilation in the pediatric population has commonly involved patients who are generally healthy.[9] Nevertheless, loss of airway and acute hypoxia are more likely to occur in patients with pre-existing conditions that affect respiration. Medical disorders that potentially compromise ventilation during sedation include restrictive and obstructive lung diseases.[10] Obesity is a condition in which patients are considered to have chronic extrinsic restrictive lung disease as well as other compromised systems (eg, cardiovascular, gastrointestinal), all of which can contribute to major injuries during sedation.

The purpose of this review was to describe the potential influence of childhood obesity on pharmacosedation in pediatric dentistry and provide specific recommendations for managing obese patients.

Prevalence of obesity

Obesity is pandemic in the United States. In 1990, the prevalence of obese adults was less than 15%. By 2000, 27% of the adult population was obese.[11] The most recent estimates show that two thirds of the adult population are either obese or overweight.[12] Each year, obesity leads to more than 300,000 premature deaths and over $100 billion in associated costs.[13,14]

The prevalence of overweight and obesity is generally higher for women than for men and for African Americans and Hispanics than the general population. Of particular concern is the increasing number of overweight children. Currently, 17% of children between the ages of 2 and 19 years are overweight.[12] This prevalence has tripled in the

[1]Dr. Baker is a pediatric dentist and resident in dental anesthesia, and [2]Dr. Yagiela is professor and chair, Division of Diagnostic and Surgical Sciences, School of Dentistry, University of California, Los Angeles, Calif. Correspond with Dr. Baker at subakertt@yahoo.com

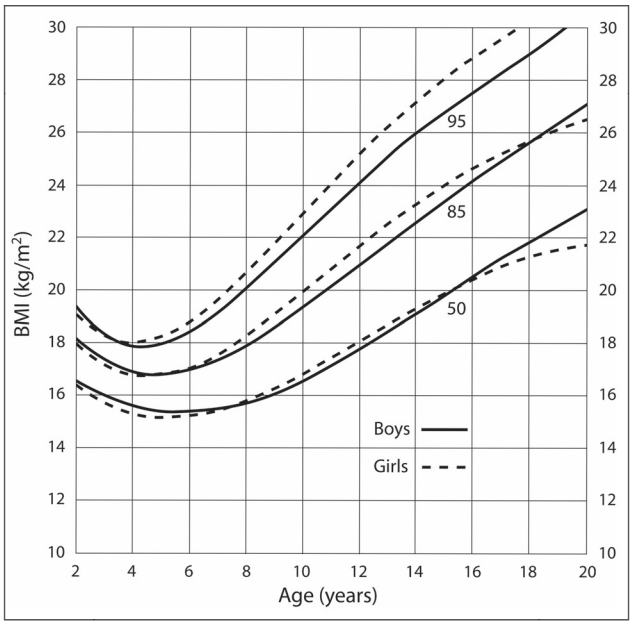

Figure 1. BMI as a function of gender and age. The 50th (median), 85th percentile and 95th percentile curves are shown. In children, overweight is defined as a BMI ¡Ý the 95th percentile; a BMI ¡Ý the 85th percentile, but below the 95th percentile is defined as at risk for overweight. Figure redrawn from data in Ogden CL, et al.[17]

past 2 decades and is especially troubling because overweight children commonly become overweight adults. Nearly one third of overweight preschool children and one half of overweight school-age children remain overweight as adults.[15]

The body mass index (BMI) is used to define overweight and obesity. It is calculated by using either of the following formulas:

$$BMI = \frac{[\text{Weight (kg)}]}{[\text{Height (m)}]^2}$$

or

$$BMI = \frac{[\text{Weight (lb)}]}{[\text{Height (in)}]^2} \times 703$$

In adults, a BMI >25 (which roughly corresponds to a body weight 10% or more over the ideal) identifies a person as being overweight and a BMI of 30 or more (indicating a body weight at least 30% over the ideal) defines obesity.[16] The term morbid obesity refers to a BMI ≥40. In children, BMI criteria for overweight vary by age and gender,[17] as is illustrated in Figure 1.

Fat in different anatomic distributions is associated with differing pathophysiologic consequences. Android obesity, the most common form in children, indicates fat is distributed primarily intra-abdominally and is highly linked to increased oxygen consumption, cardiovascular risk, and left ventricular dysfunction.[18,19] In gynecoid obesity (most prevalent in women), fat is distributed in the buttocks and thighs and is metabolically less active and minimally associated with cardiovascular disease.[20]

Table 1. Definitions of Lung Volumes and Capacities

Lung volume or capacity	Definition
Tidal volume (TV, VT)	The volume of air that moves in and out of the lungs during quiet breathing (6-7 mL/kg in both children and adults)
Inspiratory reserve volume (IRV)	The maximal inspiration of air beyond the volume of a quiet inspiration
Expiratory reserve volume (ERV)	The maximal expiration of air beyond the volume of a passive end expiration
Residual volume (RV)	The amount of air that remains in the lung after forced maximal expiration
Inspiratory capacity (IC)	The largest volume of air that can be inspired after a passive expiration
Vital capacity (VC)	The maximum volume of air expired after maximal inspiration
Functional residual capacity (FRC)	The volume of gas remaining in the lungs at passive end expiration (25-35 mL/kg in children and 30-40 mL/kg in adults)
Total lung capacity (TLC)	The maximum amount of air the lungs can hold and the sum of the VC and RV (60-65 mL/kg in children and 80-85 mL/kg in adults)

Obesity's biological consequences

Obesity is associated with a number of anatomic, physiologic, and biochemical deviations from normality that may affect all body systems.

Respiratory system

Respiratory derangements associated with obesity include restrictive lung disease and obstructive sleep apnea, either of which can compromise the quality of sedation and put the patient's life at risk. A basic understanding of the various lung volumes and capacities (Table 1, Figure 2) is essential in understanding the physiologic changes that happen during obesity.[22,23]

Obesity imposes a restrictive ventilation defect because the excess weight added to the thoracic cage and the abdomen impedes the motion of the diaphragm, especially in the supine position. The manifestations of this added weight and associated pressure on the diaphragm include decreases in the functional residual capacity (FRC), expiratory reserve volume (ERV), and, in the morbidly obese, vital capacity (VC) and total lung capacity (TLC; Figure 3). The FRC declines exponentially with an increasing BMI.[24] In addition, mass loading of the chest wall and abdomen by fat alters the static and dynamic performance of the respiratory system, reducing chest wall compliance and increasing respiratory airway resistance.[25] The FRC may be decreased to the closing capacity (CC), which is the point where small airway closure occurs and atelectasis ensues, with

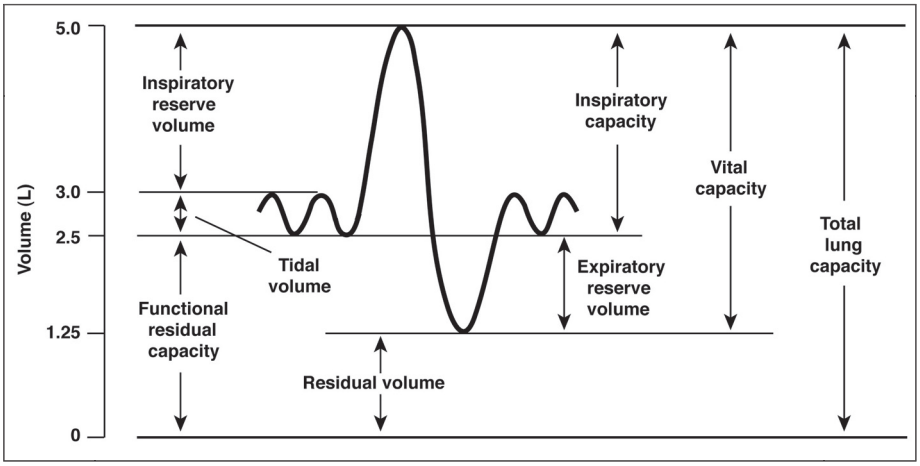

Figure 2. Dynamic lung volumes and capacities. Figure used by permission.[21]

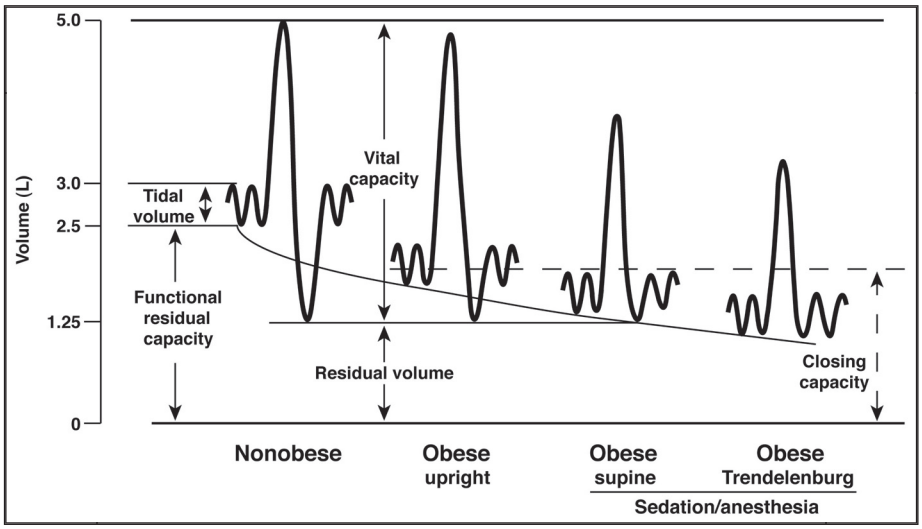

Figure 3. The effect of change in position and sedation/anesthesia on various lung volumes in nonobese and morbidly obese patients. Figure adapted from Ogunnaike BO, et al.[43]

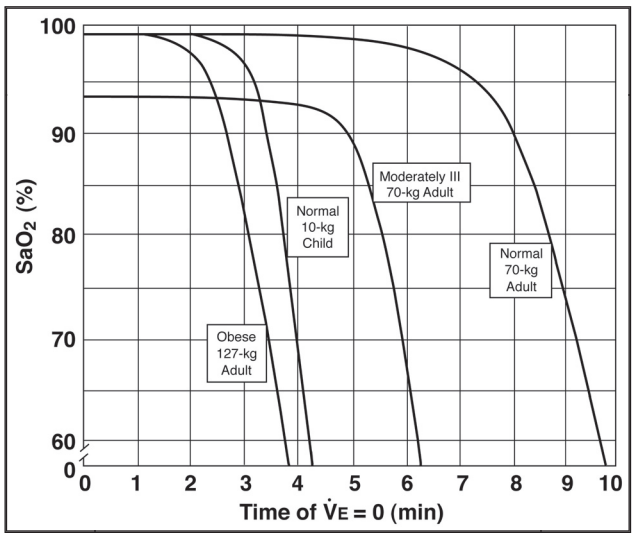

Figure 4. Oxygen saturation vs time of apnea for various types of patients. Figure adapted from Benumof JL, et al.[28]

resulting ventilation-to-perfusion (V/Q) mismatching, right-to-left shunting, and arterial hypoxemia.[26] Respiratory depressant medications used during anesthesia and sedation accentuate these changes, causing on average a 50% decrease in FRC in obese anesthetized patients in the supine position, compared with a 20% decrease in nonobese patients.[27] Because of the drop in FRC, a smaller oxygen reservoir is available during apneic periods. Even with preoxygenation, cessation of breathing can quickly lead to arterial hypoxemia (Figure 4).[28]

In the upright position, ERV and FRC are reduced such that the tidal ventilation falls within the range of the CC, with ensuing V/Q abnormalities and a trend toward hypoxemia. In the supine and Trendelenburg positions, the FRC falls even further and well below the CC, worsening the hypoxemic side effect. Obese patients also have an increased oxygen consumption (VO_2) and carbon dioxide (CO_2) production (VCO_2) as a result of the increased workload on supportive tissues.[29,30] Most obese patients maintain a sufficient minute volume of ventilation (VE) to remain normocarbic and preserve a normal response to increased CO_2, but there is an increased oxygen cost (work) of breathing. Therefore, these patients tend to breathe rapidly and shallowly. These conditions are further exacerbated in pediatric patients because of the normally higher respiratory rate (20-24 breaths/minute vs 12-20 breaths/minute) and alveolar minute ventilation (100-150 mL/kg/minute vs 60 mL/kg/minute). The heart rate is almost doubled (80-120 beats/minute vs 60-80 beats/minute) to compensate for the increased oxygen consumption (5 mL/kg/minute vs 3 mL/kg/minute). The significant decrease in FRC increases the incidence of atelectasis.[31] The administration of respiratory depressant drugs for sedation is especially likely to promote CO_2 accumulation and oxygen deficits in obese pediatric patients placed in the supine position.

The other complication associated with obesity in regard to respiration is obstructive sleep apnea. An estimated 5% of obese subjects develop obstructive sleep apnea. In these individuals, increased adipose tissue in the neck and pharynx promote airway narrowing.[32] Pharyngeal patency depends on the action of muscles (which include the genioglossus,

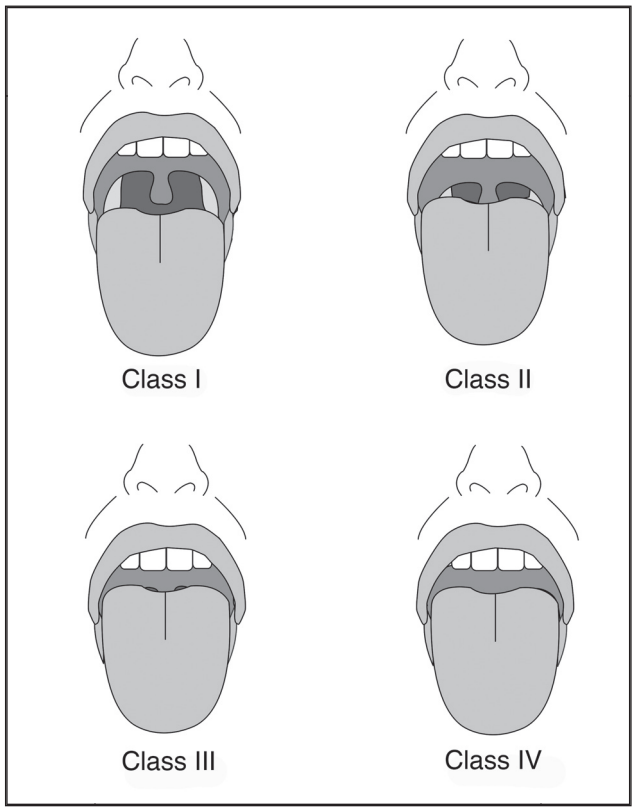

Figure 5. Mallampati classification, as modified by Samsoon GLT and Young JRB.[49] Class I—uvula, faucial pillars, and soft palate are visible; Class II—faucial pillars and soft palate are visible; Class III—soft palate is visible; Class IV—hard palate only is visible.

tensor palatini, and various hyoid muscles) that prevent upper airway collapse. Pharyngeal muscle tone decreases during physiologic sleep and pharmacosedation, leading to significant narrowing of the upper airway, turbulent airflow, and snoring.

Obesity hypoventilation syndrome is the long-term consequence of sleep apnea.[27] As the obesity hypoventilation syndrome develops, there is evidence of nocturnal alterations in the control of breathing manifesting as central apneic events (apnea without respiratory efforts). These episodes of central apnea cause desensitization of the respiratory centers to hypercarbia. At its extreme, obesity hypoventilation syndrome culminates in the "Pickwickian syndrome," which is characterized by gross obesity, somnolence, periodic breathing, hypercapnea, hypoxemia, polycythemia, and pulmonary hypertension.

Cardiovascular system

Although primary cardiovascular complications are minimal in healthy young patients given oral sedation, they have to be considered during the treatment of obese pediatric subjects. When excess fat is distributed intra-abdominally, it is associated with increased cardiovascular risk and left ventricular dysfunction.[18,19,30] Systemic hypertension is present in 50% to 60% of obese patients, and, when combined with hypervolemia, increases the potential for congestive heart failure.[27,33-36] Cardiac dysrhythmias in obese individuals may be precipitated by arterial hypoxemia, hypercarbia, ischemic heart disease, obese hypoventilation syndrome, or fatty infiltration of the cardiac conduction system. Obese patients tolerate exercise poorly, with any increase in the cardiac output achieved by increasing the heart rate and/or stroke volume. Obese patients positioned supinely already tend to have an increased cardiac output. Given the decreased perioperative tissue oxygenation associated with obesity and the increased cardiac workload, the likelihood for myocardial hypoxia is enhanced. [37] Also, because the cardiovascular system itself is adversely affected by obesity, acute respiratory difficulties encountered during sedation are more likely to precipitate cardiovascular complications in significantly overweight children.

Gastrointestinal system

Hiatal hernia and increased intra-abdominal pressure are more likely in the obese. A fasting adult obese patient has a gastric fluid volume in excess of 25 mL and a gastric fluid pH less than 2.5, which are greater than for lean patients.[38] In obese pediatric patients, the gastric volume is approximately 0.4 mL/kg. Such volume and pH figures are generally accepted as indicating a high risk of aspiration pneumonitis if the gastric fluid reaches the lungs. Because the risk of gastric regurgitation is relatively high in obese patients, specific measures should be taken to guard against it.[39] There is also marked steatosis and increased serum enzymes, indicating fatty degeneration of the liver even in moderately obese individuals.[40] Other complications associated with obesity include:

1. fat face and cheeks;
2. short neck; and
3. limited flexion of the cervical spine and atlantoaxial joint.

Mouth opening may be restricted because of: (1) submental fat; (2) fleshy cheeks; and (3) a large tongue.

Pharmacokinetics in the obese

Multiple pharmacokinetic deviations are associated with obesity.[41,42] Drug absorption, distribution, metabolism, and excretion are all affected by an increase in BMI. Hydrophilic drugs (eg, aminoglycosides, vecuronium) exhibit no significant differences in peak blood concentrations between obese and normal-weight patients when dosed on the lean body mass (LBM).[43]

LBM includes all tissues other than fat. It is usually determined by measuring skinfold thickness, bioelectrical impedance, hydrostatic weighing, or dual-energy X ray absorptiometry, but can be estimated by the method of James[44] as follows:

$$\text{LBM (males)} = [1.10 \times \text{weight}] - [128 \times (\text{weight}^2/\text{height}^2)]$$

or

$$\text{LBM (females)} = [1.07 \times \text{weight}] - [148 \times (\text{weight}^2/\text{height}^2)]$$

where weight is in kg and height is in cm.

On average, an increase in LBM contributes 20% to 40% of the total increase in body weight with obesity.

Lipophilic drugs, however, including most agents used for sedation (eg, midazolam), act differently. Intravenous bolus

doses of drugs such as propofol calculated on lean body mass tend to produce similar initial effects irrespective of obesity, but the duration of clinical effect is shortened by increased redistribution to body fat.[45] Repeated doses also show less early accumulation of effect in the obese because the excess fat increases the ability to sequester drugs away from the brain. Upon cessation of drug administration, the fat-sequestered drug will slowly return to the systemic circulation, resulting in a longer elimination half-life. While initial recovery from sedation may tend to be faster in the obese, complete recovery may be delayed.[41]

Oral administration of sedative drugs (barbiturates, benzodiazepines, and opioids) is usually dosed by total body weight because distribution occurs in concert with absorption.[43] Nevertheless, depending on the relative rates of absorption and distribution of the agent, increased initial effects may occur in the obese, and the clinical duration is likely extended. Local anesthetics are also dosed according to total body weight. Nitrous oxide is a relatively insoluble inhalation anesthetic and has a rapid onset and fast elimination.[46] Even with nitrous oxide, however, prolonged administration to an obese individual may result in delayed recovery because of the modest tendency for nitrous oxide to accumulate in body fat.

Morbidly obese patients have significantly higher pseudocholinesterase activities than nonobese patients,[47] which might be of some benefit when using topical ester local anesthetics. (Since all injectable local anesthetics are amides and not metabolized by this enzyme, they are not affected.) Pathophysiologic disturbances in liver function in obesity may affect hepatically mediated drug clearance, but sedation after single doses of a drug is usually not influenced. Obesity is not associated with changes in Phase I metabolism (oxidation, reduction, hydrolysis); drugs eliminated through some Phase II conjugation pathways (eg, glucuronidation) appear to be cleared faster in the obese.[41] Both glomerular filtration and tubular secretion are also increased in the obese, and renally excreted drugs (eg, amoxicillin, gentamicin) may need more frequent dosing.

General recommendations

A thorough medical evaluation is recommended to identify signs and symptoms indicative of significant respiratory disease as well as any history of a difficult airway during previous sedation/anesthesia.[48] Conditions compromising the respiratory system, such as sleep apnea, hypoventilation syndrome, or restrictive lung disease, have to be considered before sedating the pediatric dental patient because of the increased risk of intraoperative complications.[10,49] Although obesity-associated cardiovascular disorders are unlikely in young subjects, obese adolescents may have systemic conditions such as diabetes or hypertension that might increase treatment risk. More important is the increased potential for respiratory disorders to promote hypoxia-associated changes in cardiovascular rhythm or function.

Physical examination of the patient should include range-of-motion testing of the: (1) atlantoaxial joint; (2) cervical spine; and (3) jaw. The mouth, tonsils, and pharynx should be examined for excessive tissue. The Mallampati classification (Figure 5), based on an ability to visualize the uvula and nearby structures, may help identify patients at risk for airway obstruction.[50]

Although the dose of oral sedative agents is usually calculated according to the total body weight, reductions in maximum dosage are advisable to avoid respiratory depression and possible apnea. Because oral sedative drugs cause unpredictable effects, they should not be administered until the patient is in a monitored environment.[6]

The use of nitrous oxide (≤50%) is a good option for pediatric sedation because of its: (1) rapid onset; (2) minimal respiratory depression; and (3) easy reversibility. Hydroxyzine (up to 2 mg/kg) alone or with nitrous oxide/oxygen sedation is also indicated in obese children because of a relative lack of respiratory depression and airway relaxation. Benzodiazepines, such as midazolam in doses up to 0.6 mg/kg, are also suitable. Chloral hydrate and opioids, especially in large doses, should be avoided because of their greater likelihood of producing excessive sedation and respiratory compromise.[6,7,51] The accumulative side effects of sedative agents with local anesthetics, nitrous oxide, and particularly opioids must also be considered in selecting an oral sedative regime.[7]

Because the gastrointestinal system may be compromised by obesity, strict fasting requirements as promulgated by the American Academy of Pediatric Dentistry (AAPD), should be followed.[52] The prophylactic administration of agents against aspiration pneumonitis (eg, H_2-receptor antagonists, gastric prokinetics, and/or soluble antacids) should be considered for patients with a history of aspiration or vomiting during sedation or anesthesia.[53,54] The supine position, especially during sedation, is commonly used in pediatric dentistry. Unfortunately, this position compromises the obese child's ability to ventilate and maintain adequate oxygenation, and a semisitting position may be preferred.[55] Monitoring should include a pulse oximeter to identify oxygen desaturation and a precordial/pretracheal stethoscope to detect ventilation problems such as airway obstruction. In addition to their use as mandated by state law and advised by AAPD guidelines, a blood pressure cuff, electrocardiogram, and capnograph may be needed in obese individuals with associated medical problems. Referral to the hospital or surgical center operating room should be considered if the patient's obesity is such that serious systemic complications are likely.

Postoperative hypoxemia is a common complication associated with sedation and anesthesia in obese patients.[10] Therefore, obese patients should be monitored with a pulse oximeter during the recovery period and not be discharged until their respiratory status has been cleared and they remain awake without stimulation. Postoperative hypoxemia can be minimized by keeping the patient in

a semisitting position following dental treatment.[56] The use of postoperative opioid analgesics should be avoided. Any patients experiencing emesis during dental treatment with sedation should have a medical consult and a chest X ray if there is suspicion that the child may have aspirated stomach contents.

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