Anesthetic Implications In Pediatric Obesity

Essay
Submitted for partial fulfillment of Master Degree in
Anesthesia

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2008

Abstract

The rapidly increasing prevalence of obesity among children and adolescents is one of the most challenging dilemmas facing pediatric care professionals today. Childhood and adolescent obesity are important risk factors for adult obesity, with its consequent morbidity and mortality. Therefore, prevention and/or treatment of childhood and adolescent obesity offer the best hope of preventing adult obesity and its related morbidities. A variety of adverse consequences are associated with being overweight in childhood or adolescence, including but not limited to type 2 diabetes mellitus, dyslipidemia, hypertension, and poor self-esteem. Type 2 diabetes mellitus currently accounts for up to 45% of all newly diagnosed diabetes in pediatric patients and is more common in ethnic and racial groups with higher rates of obesity.

Key Words :

Adjustable gastric banding – Body mass index - Residual Volume .

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Abbrev	
AGB	Adjustable gastric banding
BIPAP	Bilevel-positive airway pressure
BMI	Body mass index
CDC	Centers for Disease Control and Prevention
CPAP	Continuous positive airway pressure
ERV	Expiratory reserve volume
FRC	Functional residual capacity
IOTF	International Obesity Task Force
LVEDP	Left ventricular end-diastolic pressure
OSA	Obstructive sleep apnoea
PCAS	Patient-controlled analgesia system
PEEP	Positive end-expiratory pressure
REM	Rapid eye movement
RV	Residual volume
T1DM	type 1 diabetes mellitus
T2DM	type 2 diabetes mellitus

Acknowledgement

First and foremost, praise and thanks must be to ALLAH who guides me throughout life.

I would like to express my deepest gratitude and thanks to ***Prof. Dr. Ashraf Mohamed Mohsen, Professor of Anesthesia, Faculty of Medicine – Cairo University*** for his kind continuous encouragement and great support throughout the work. It was a great honor to work under his meticulous supervision.

Also I am really deeply grateful to ***Prof. Dr. Manar Mahmoud Radwan El-Kholy***, ***Professor of Anesthesia, Faculty of Medicine – Cairo University*** for her great help, valuable time, careful supervision and continuous advices and her efforts that made this work come to light.

I am also greatly indebted to ***Prof. Dr. Hala Mostafa Gomaa,*** **Assistant** ***Professor of Anesthesia, Faculty of Medicine – Cairo University*** for her careful and great support. She did not spare any effort in guiding me towards the best and her valuable advices.

I am really thankful to every one who took part in exhibiting this work to light.

Atef Mohamed Sayed

Introduction

The rapidly increasing prevalence of obesity among children and adolescents is one of the most challenging dilemmas facing pediatric care professionals today. Childhood and adolescent obesity are important risk factors for adult obesity, with its consequent morbidity and mortality ***(1)***. Therefore, prevention and/or treatment of childhood and adolescent obesity offer the best hope of preventing adult obesity and its related morbidities. A variety of adverse consequences are associated with being overweight in childhood or adolescence, including but not limited to type 2 diabetes mellitus, dyslipidemia, hypertension, and poor self-esteem. Type 2 diabetes mellitus currently accounts for up to 45% of all newly diagnosed diabetes in pediatric patients and is more common in ethnic and racial groups with higher rates of obesity ***(2)***.

Salazar-Martinez et al. (3) compared body mass index and correlates stratified by sex and country through multivariate linear regression in Egypt. The overall prevalence of overweight and obesity was 12.1 and 6.2%, respectively, among the Egyptian adolescents. Based on U.S. Centers for Disease Control and Prevention (CDC) definition growth charts, in the Egyptian sample, 7% of boys and 18% of girls were overweight and 6% of boys and 8% of girls were obese.

Epidemiology:

Body mass index (BMI, [calculated as weight in kilograms divided by the square of height in meters]) is the most widely accepted method used to screen for obesity in children and adolescents because the measurements needed to calculate BMI are noninvasive. Body mass index is a reliable indicator of body fat content for most children and adolescents. Although the BMI does not measure body fat directly, it correlates well to direct measures of body fat, such as underwater weighing and dual-energy x-ray absorptiometry. Additionally, BMI has been found to correlate well with obesity-related complications ***(4)***.

The Centers for Disease Control and Prevention uses the term *overweight* to designate children (aged 2-19 years) with BMI at or above the 95th percentile for age and sex and does not use the term *obese* in describing childhood weight categories. The term *at risk for overweight* is used for children with BMI between the 85th percentile and the 95th percentile for age and sex ***(5)***.

Childhood obesity has reached epidemic proportions in developed nations throughout the world. According to the National Health and Nutrition Examination Surveys, the prevalence of obesity in preschool children (aged 2-5 years) and children (aged 6-11 years) from 1999 to 2002 was double that between 1976 and 1980; for adolescents (aged 12-19 years), triple ***(6; 7)***. Among children aged 6 to

19 years in 1999 to 2002, 31.0% were overweight or at risk of being so and 16.0% were overweight ***(7)***. The risk for being overweight is increased among persons with high birth weight (≥4000 g) or with obese parents ***(8)***.

According to ***WHO (9)*** at least 50% of adults and 20% of children in U.K. and U.S.A. are currently overweight. Prevalence of overweight amongst Australian children has increased from 11% in 1985 to 20% in 1995. Childhood obesity has tripled in Canada in last 20 years. It has been estimated that, in 1995, the direct costs of treatment of obesity in USA accounted for $70 billion with far greater indirect costs ***(10)***.

The calculated global prevalence of overweight (including obesity) in children aged 5-17 years is estimated by the International Obesity Task Force (IOTF) to be approximately 10%, but this is 'unequally distributed' with prevalence ranging from over 30% in Americas to <2% in sub Saharan Africa ***(11)***.

I. Pathophysiology:

Almost all obesity in children is exogenous, caused by a caloric intake that is greater than needed. An excess intake of only 50 to 100 kcal per day can lead to a 2.0 kg to 5.0 kg weight gain over a 1-year period. Energy output is derived from resting energy expenditure, the thermal effect of food and activity. The epidemic of obesity in the past 2 decades is likely a result of a gradually increasing

caloric intake and a decreasing level of physical activity. Endocrine and genetic diseases are rare causes of obesity in childhood, accounting for less than 1% of childhood obesity in tertiary care centers. Certain antidepressants such as monoamine oxidase inhibitors and tricyclic antidepressants tend to be more associated with weight gain than are selective serotonin reuptake inhibitors. However, this association does not imply causation ***(5)***.

Relation between Fetal Weight and Obesity:

Most investigations into the relationship between prenatal exposures and later obesity have studied associations between birth weight and attained BMI. Birth weight can be easily measured, has reference norms, is part of the routine medical record, and may be available historically. Variation in weight at birth serves as a surrogate to reflect underlying mechanisms influencing growth ***(12)***.

BMI (kilograms per meter squared), a gauge of weight for height, is the most common measure of obesity in child and adult epidemiological studies. One attraction is its simplicity of measurement; even self-report of BMI can be quite accurate ***(13)***. BMI predicts morbidity and mortality in a strong, graded relationship ***(14)***. BMI has been used in many populations worldwide, allowing comparison among study results. Nevertheless, the use of

BMI as a proxy for adiposity has recognized limitations ***(13)***.

More than two dozen studies have addressed the association between birth weight and attained BMI. Most have measured the outcome in childhood, but several have examined adult BMI ***(8)***. Almost all of the studies have found direct associations, i.e., that higher birth weight is associated with higher attained BMI ***(15; 16)***. Some of the smaller studies have found no association ***(17)***; none have found an inverse association. The typical magnitude ranges from 0.5 to 0.7 kg/m^2 for each 1-kg increment in birth weight ***(18)***.

Limitations of most of these studies have included incomplete data on gestational age, birth length, parental body size, tobacco use, and socioeconomic factors. Disentangling prematurity from impaired fetal growth is important, because the two may have different determinants and different sequelae. Whereas many published data emanate from an era when few premature babies survived until adulthood, a few more recent studies of children and adolescents have found that the relationship between birth weight and obesity persists after adjustment for gestational age. In a study of Danish military conscripts, BMI at ages 18 to 26 years rose monotonically over the range of birth weight, after

controlling for gestational age, birth length, and maternal factors ***(12)***.

Maternal and paternal body habitus predict offspring fatness, particularly fatness during childhood. A combination of genetic and both pre- and postnatal environmental causes is likely. In addition, parental adiposity is directly associated with offspring birth weight, with stronger associations for the mother than for the father, which implicates prenatal environmental factors. Associations of maternal and paternal birth weight with offspring birth weight also suggest genetic or intergenerational environmental influences ***(19)***. Thus, one needs to interpret results after adjustment for maternal BMI carefully. Empirically, where data have been available, the relationship between birth weight and later BMI tends to be attenuated after control for maternal BMI. In the U.S. Growing Up Today Study, a cohort study of over 14,000 adolescents, a 1-kg increment in birth weight among full-term infants was associated with an ~50% increase in the risk of overweight at ages 9 to 14 years. The increase in risk was ~30% after adjustment for maternal BMI, with no further attenuation after control for additional social and economic factors ***(20)***.

Similarly, social and economic factors may confound the relationship between birth weight and later adiposity. Low socioeconomic status was associated with obesity in

children and adults during the late 20th century ***(21)***. Babies born to women with lower social status have lower birth weights in some recent studies, although not in studies using data from the early part of the century. Thus, the direction of confounding may differ depending on the era of data collection. More appropriate strategies to control for social status, such as information on diet and household crowding, are needed to minimize residual confounding of the birth weight–obesity relationship ***(22)***.

II. Evaluation of Obese Children:

Obesity can be seen in association with a wide variety of genetic and endocrine disorders **(table 1)**, the signs and symptoms of which include hypogonadism, short stature, dysmorphic features, and mental retardation. Growth failure characterizes endogenous obesity secondary to endocrine disorders such as hypothyroidism and hypercortisolism. Children with obesity secondary to genetic syndromes typically have characteristic physical examination findings. Prader Willi syndrome, the most common syndromic form of obesity, is characterized by failure to thrive early in life, short hands and feet, short stature, hypotonia, and genital hypoplasia. Children with endogenous obesity are short and often have a delayed or normal bone age. Conversely, children with idiopathic or exogenous obesity are taller and often have growth acceleration along with advancement in bone age. A

careful history and physical examination are usually adequate to rule out or suspect an endogenous cause for obesity in most children **(fig. 1)** ***(5)***.

Childhood and adolescent obesity are important risk factors for adult obesity, with its consequent morbidity and mortality. Therefore, prevention and/or treatment of childhood and adolescent obesity offer the best hope for preventing adult obesity and its related morbidities.

A variety of adverse consequences are associated with being overweight in childhood or adolescence, including but not limited to type 2 diabetes mellitus, dyslipidemia, hypertension, sleep apnea and gall bladder disease.