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POINTS OF VIEW: MEDICAL TRANSFER

Retrieval and transfer of bariatric patients in NSW

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SUMMARY

Rates of obesity are rising throughout the world. Obese patients have a number of co-morbidities and associated diseases which can impact the safe transportation of these patients. This overview of the retrieval of bariatric or obese patients will explain some of the physiological and different co-morbidities found in these patients, as well as some more practical advice when faced with transporting these patients.

1. Introduction

Bariatric comes from the Greek 'Baros' and means 'treatment of weight'. Obesity comes from the Latin 'Obedere' meaning 'to consume', and is becoming much more prevalent especially in western society. In the USA 34.3% of Americans are now obese.[1] In both Australia and the UK rates are rising sharply. In Australia obesity rates have doubled since 1990 (9–18%), and 62% of the adult population are now overweight or obese,[2] with similar numbers for the UK (obesity rates having increased from 15% to 24% since 1993[3]).

New South Wales (NSW) has an area of approximately 810,000 square km and a population of around 7 million and around 6 million of these live in Sydney alone. This means that there are vast areas with very sparse populations and correspondingly limited access to health care. So some patients must often travel substantial distances to access specialised tertiary care. Obesity being associated with an increased incidence of different medical and surgical pathologies,[4] means that moving or retrieving them in NSW is becoming much more common. In 2009 the Ambulance Service of NSW (ASNSW) transferred almost 300 patients weighing over 150 kg (the heaviest was 318 kg), of which 23 were medical retrievals with a Doctor/Paramedic escort.

This overview of the retrieval of bariatric or obese patients will explain some of the physiological and different co-morbidities found in these patients, as well as some more practical advice when faced with transporting these patients.

2. Definitions

Through convention Body Mass Index (BMI) has become the standard measurement of obesity. BMI is calculated by weight (in kg) divided by height (in metres) squared. Normal is defined as a BMI of between 20 and 25 kg/m^2, 25–30 kg/m^2 is considered overweight, >30 kg/m^2 is obese, >35 kg/m^2 is morbidly obese, >55 kg/m^2 is super-morbidly obese.[5] But some authors consider a BMI >35 kg/m^2 with associated co-morbidity or >40 kg/m^2 without a significant co-morbidity to be the definition of morbidly obese.[6] Although it is a robust and very practical way of assessing obesity it is not without its limitations. The rugby player with a high lean muscle mass will tend to be overestimated on the BMI scale, whilst an elderly lady with close to zero lean muscle mass will tend to be underestimated.

Ideal body weight comes from life insurance studies looking at the weight associated with the lowest mortality rates.[5] It can be calculated from the formula IBW (kg) = height (cm) – x (where $x = 100$ for males and 105 for female adults).

3. Co-morbidities

3.1. Respiratory

Obesity is an important risk factor for Obstructive Sleep Apnoea (OSA). The prevalence of OSA has been reported as high as 30% in those patients with a BMI >30 kg/m^2 and 40% in those with a BMI >40 kg/m^2.[7] OSA is defined as apnoeic episodes secondary to pharyngeal collapse that occur during sleep, and is diagnosed by sleep studies. Apnoeic episodes are defined as ten seconds or more of total cessation of airflow despite respiratory effort against a closed airway. More than thirty per night is often quoted as clinically significant. Snoring is another of the characteristic features of OSA, as are day-time somnolence associated with impaired concentration and morning headaches. Recurrent apnoea can lead to physiological changes such as hypoxaemia (leading to secondary polycythaemia), hypercapnia, systemic or pulmonary vasoconstriction which can lead to right ventricular failure.[5]

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Obesity hypoventilation syndrome is at the more extreme end of the sleep-disorder breathing spectrum. It is synonymous with the "Pickwickian" syndrome, named after Joe the fat red faced boy who used to fall asleep uncontrollably during the day in Charles Dickens' The Pickwick Papers. It arises when nocturnal hypoventilation is so severe the patient is unable to clear the hypercapnia and hypoxia and therefore leads to a type II respiratory failure during the day. It leads to a desensitisation to the CO_2 control of respiratory drive, which is partly under the control of leptin; to which the obese patient has a relative insensitivity to. This effect is accentuated by respiratory depressant drugs such as analgesics and anaesthetic agents.[6]

Morbid obesity is associated with a decreased functional residual capacity (FRC). It declines sharply with increasing BMI reaching volumes of around 1000mls in patients with BMI's >40 kg/m^2.[6] Closing capacity is also reduced and can encroach on FRC during normal tidal volume and causes airway closure and ventilation perfusion (V/Q) mismatching thereby increasing intra-pulmonary shunt. This effect is increased after anaesthesia[8] with FRC falling by 50% in the anaesthetised obese patient compared to 20% fall in the anaesthetised non-obese patients. The reduction in FRC results in a smaller oxygen reservoir and with an increased oxygen consumption means obese patients tend to desaturate quicker than their non-obese counterparts. Sitting the patient in a 25° head-up position will achieve a higher pre-induction oxygen tension and take longer to desaturate (down to 92%) compared to a supine group after pre-oxygenation[9]; a head-up position can also improve the view at laryngoscopy.[10] Once asleep PEEP can improve both FRC and arterial oxygen tension, but unfortunately at the expense of cardiac output and therefore oxygen delivery.[11]

Increasing BMI is associated with a reduction in respiratory compliance. This is partly due to an increased weight on the chest wall and restriction of diaphragmatic movement but also due to an increased pulmonary blood volume.[12] This reduction in compliance, increased mechanical pressure from the abdomen results in an increased work of breathing and therefore increases the metabolic demand of breathing.

Traditional teaching tells us that with morbidly obese patients it may be very difficult to manage the airway and with the incidence of difficult intubation as high as 13%.[13] Causes of this increased difficulty include features such as a fat face and cheeks, large breasts, short neck, large tongue, excessive palatal and pharyngeal soft tissue and fat pad on their backs which can make positioning difficult.[5]

But this traditional teaching may be called into question with more recent literature suggesting that intubation is no more difficult than in non-obese subjects.[14] In a prospective trial by Erzi et al.[15] of 764 mixed surgical patients it was found that increased BMI correlated with increased Mallampati scores but not with increased Cormack and Lehane (C+L) grade of laryngoscopy. Another recent study using C+L grade 3–4 as the descriptor of difficult laryngoscopy in 397 patients with a BMI of >50 kg/m^2, found an incidence of 6% which is similar to the incidence found in the general population.[16] Likewise there are mixed results from studies looking at the ease of facemask ventilation.[14,17]

In the authors experience careful positioning (sitting head-up ~30 degrees) and planning (with secondary intubation techniques available, e.g. LMA) a rapid sequence induction using succinylcholine is practical and safe. A polio blade, short handle and McCoy laryngoscopes together with a bougie can also be extremely effective.

3.2. Cardiovascular

There is an increase in blood volume, cardiac output, ventricular workload, oxygen consumption and carbon dioxide production.[6] The increase in cardiac output is proportional to degree of obesity,[5] i.e. fat needs 2–3 ml/100 g/min therefore an extra 50 kg requires 1–1.5 l/min extra cardiac output.

The above may cause systemic and pulmonary hypertension possibly leading to cor pulmonale and right heart failure. Systemic hypertension is seen in up to 60% of obese patients,[18] and can lead to left ventricular hypertrophy and so making it less compliant; and together with the increased blood volume it can increase the risk of heart failure.[5] The increase in cardiac output is largely due to the result of ventricular dilation and an increased stroke volume.[5] When the dilation outpaces the hypertrophy it gives rise to systolic dysfunction and is termed obesity cardiomyopathy.

Obesity is an independent risk factor for ischaemic heart disease; compounding the problem is the associated hypertension, diabetes mellitus and hypercholesterolaemia all commonly seen in obese patients. Together with an increased oxygen demand and a decreased supply there is an increased risk of myocardial ischaemia.

3.3. Other systems

There is a high incidence of gastro-oesophageal reflux disease and hiatus hernia.[6] Gastric volumes are increased (up to 75%[5]), low gastric pH, and with high intra-abdominal pressures it is sensible to take precautions against acid reflux and aspiration. These can include H_2 receptor antagonists, antacids, rapid sequence induction with cricoids pressure and sitting the patient up prior to induction.

Adipose tissue is active and produces many substances including Leptin. Leptin has many functions including at the hypothalamus to decrease appetite although resistance is often seen in obesity; CO_2 sensitivity and respiratory drive are partly under control of Leptin and the relative Leptin insensitivity is associated with a reduced response to CO_2. Leptin also increases sympathetic drive (together with increased Insulin levels and free fatty acids) via the hypothalamus which causes sodium and water retention worsening hypertension.[19] Insulin resistance is often seen giving rise to type II diabetes and so it is important to monitor blood glucose levels. The risk of deep vein thrombosis is approximately twice that of lean patients (undergoing non-malignant surgery – 48% vs 23%[20]). There is also the potential risk of pressure sores due to decreased blood supply/ischaemia as a result of prolonged immobility during the transfer. Correct positioning and adequate pressure reducing padding must be used (e.g. foam or gel pads).

3.4. Pharmacokinetics

Calculating the appropriate dose of different drugs can be difficult. Should calculations be based on total body weight, lean body mass or ideal body weight? The answer is not simple. Physiological changes associated in obesity lead to changes in the distribution, binding and clearance of many drugs. Associated co-morbidities and their effects on the organs involved in drug elimination (e.g. liver and kidney) can make the pharmacokinetics more difficult and complex. The volume of distribution will be affected by an increased lean body mass, increased blood volume, increased cardiac output, reduced total body water, alterations in plasma protein binding and the fat solubility of the drug.[5] For example highly fat soluble drugs (e.g. Thiopentone) there is a significant increase in its volume of distribution. For these drugs the ideal body weight should be used when calculating the dose.[6] However less fat soluble drugs show minimal changes in their volume of distribution (e.g. neuromuscular blocking agents) and so lean body mass should be used (or ideal body weight plus 20%).[6] Often the monitoring of clinical end-points (e.g. heart rate, blood pressure and sedation) is more important that weight based formulae.[5]