Anaesthetic considerations with the metabolic syndrome

A. Tung*

Department of Anesthesia and Critical Care, University of Chicago, 5841 S. Maryland Avenue MC4028, Chicago, IL 60637, USA
*E-mail: atung@dacc.uchicago.edu

Key points

- Metabolic syndrome is a distinct obesity-related syndrome characterized by truncal obesity, insulin resistance, dyslipidaemia, and hypertension.
- Metabolic syndrome predisposes to coronary artery disease, congestive heart failure, obstructive sleep apnoea, pulmonary dysfunction, and deep venous thrombosis.
- Since there are few data to support an evidence-based approach to global anaesthetic management, the focus is to mitigate the clinical features of the syndrome that generate the greatest perioperative risk.

Summary. The rising incidence of obesity has led to increased prevalence of a distinct, obesity-related metabolic syndrome. This syndrome is characterized by truncal obesity, insulin resistance, altered lipid levels, and hypertension. Definition of the metabolic syndrome rests on a set of clinical criteria instead of a single diagnostic test. It carries a different risk profile than obesity alone, and poses special challenges for the anaesthesiologist. These include preoperative risk stratification for common comorbidities, identifying reasonable thresholds for implementing preoperative risk reduction, overcoming obesity-related issues in intraoperative management, and delivering safe postoperative care. The metabolic syndrome predisposes to coronary artery disease, congestive heart failure, obstructive sleep apnoea, pulmonary dysfunction, and deep venous thrombosis. Because its different presentations can have different risk profiles, anaesthesiologists should assess the cumulative risk of each component of the metabolic syndrome separately, which significantly complicates preoperative management. Since obesity itself is difficult to treat, preoperative risk reduction can be difficult. Few data exist to inform best practice as to the anaesthetic care of patients with metabolic syndrome. This review evaluates and synthesizes current evidence regarding perioperative care for patients with the metabolic syndrome, including indications for preoperative testing; use of aspirin, β-blockers, statins, heparin, and angiotensin-converting enzyme inhibitors; anaesthetic strategies including regional anaesthesia; and postoperative management including continuous positive pressure ventilation by mask, prevention of pulmonary embolism, and indications for advanced respiratory monitoring.

Keywords: anaesthesia; metabolic syndrome; obstructive sleep apnoea; preoperative evaluation

Technical challenges in anaesthetizing the morbidly obese are considerable. The increasing incidence of obesity worldwide has led to recognition of a distinct obesity-related syndrome with associated insulin resistance and altered lipid regulation. Although this syndrome has been recognized for more than 30 yr, its rising prevalence has led to increasing evidence regarding its definition, pathogenesis, and associated comorbidities.

The metabolic syndrome includes not only obesity (specifically obesity distributed around the midsection) but also dyslipidaemia (elevated triglyceride and reduced high-density lipoprotein (HDL) levels), hyperglycaemia, insulin resistance, and hypertension. Unlike patients with isolated obesity, those with the metabolic syndrome are at greater risk for coronary artery disease (CAD), obstructive sleep apnoea (OSA), hypercoagulability, and pulmonary dysfunction. For anaesthesiologists, the metabolic syndrome increases the complexity and risk of anaesthetic management and complicates the list of pre-, intra-, and postoperative considerations relevant to safe perioperative care.

The underlying links between the different components of the metabolic syndrome are not known, and no single diagnostic laboratory test exists. Instead, the metabolic syndrome is defined by clinical criteria in the same way as the acute respiratory distress syndrome. This unavailability of focused diagnostic testing complicates the assessment of anaesthetic risk. Because each metabolic syndrome component can exist independently of the others, anaesthesiologists with existing criteria for management of hyperglycaemia, hypertension, or CAD alone must adjust those criteria if these conditions present concurrently. The increase in the prevalence of obesity (and thus metabolic syndrome) over time may also shift thresholds for what is considered normal, further complicating perioperative risk assessment.

Morbid obesity and the metabolic syndrome share obesity as a common characteristic. General perioperative management of obesity has been extensively covered elsewhere. This review will focus specifically on the metabolic syndrome, including current knowledge regarding the epidemiology,
prevalence, and definition of metabolic syndrome, morbid-
ities associated with metabolic syndrome that affect anaes-
thetic management, and current approaches to minimizing
associated perioperative complications.

**Definition, pathophysiology, epidemiology, and outcomes**

**Definition**

The metabolic syndrome is formally defined as a specific
‘truncal’ distribution of adipose tissue associated with
(depending on the set of diagnostic criteria) insulin resistance
and hyperglycaemia, decreased HDL levels, elevated triglycer-
ide levels, and hypertension. It differs from obesity alone in
explicitly including metabolic parameters, and focusing on
the distribution of adipose tissue more than the amount.
Unlike morbid obesity, which in most definitions requires a
body mass index (BMI) >35, the metabolic syndrome does
not require severe obesity. Instead, only moderate obesity
(BMI>30) or a specific distribution (defined by waist–hip
ratios or waist circumferences) of obesity are required.

Because the pathophysiology of this syndrome is incom-
pletely understood, and since not all patients with the meta-
bolic syndrome have all of the associated conditions, the
diagnosis of metabolic syndrome rests not on a specific
test, but on whether patients meet predefined sets of specific
clinical criteria. The three most commonly used formal de-
definitions are shown in Table 1.

**Pathophysiology**

Whether the metabolic syndrome arises as an interactive
consequence of its underlying components, or whether
additional environmental or genetic conditions are involved,
is unknown. Twin studies suggest strong concordance for
some syndrome components (glucose intolerance, overall
obesity, low HDL) but weaker heritability estimates for
others (adipose distribution, triglyceride levels). Along with
an inverse correlation between birth weight and the
likelihood of metabolic syndrome, these studies support a
significant effect of both genetic and environmental
components.

Individual metabolic syndrome components such as
hypertension, dyslipidaemia, and obesity clearly have both
environmental and genetic components. How environmental
and genetic conditions interact to produce the metabolic
syndrome is unclear. The best current hypothesis is that its
pathogenesis involves an initial accumulation of endocrino-
logically active truncal adipose tissue. Unlike non-truncal fat,
this truncal fat then secretes proinflammatory adipocyto-

Table 1  Diagnostic criteria for metabolic syndrome. Definitions from the WHO (World Health Organization; 1999), NCEP ATP (National Cholesterolesterol Education Program Adult Treatment Panel; 2001), and IDF (International Diabetes Federation; 2006). Alb/Cr, albumin/creatinine ratio; SAP, systolic arterial pressure; DAP, diastolic arterial pressure.

<table>
<thead>
<tr>
<th>Criteria</th>
<th>Essential</th>
<th>Central obesity</th>
<th>Insulin resistance</th>
<th>Lipid profile</th>
<th>Hypertension</th>
<th>Others</th>
</tr>
</thead>
<tbody>
<tr>
<td>WHO (1999)</td>
<td>Diabetes mellitus or insulin resistance and two of the following</td>
<td>Waist-to-hip ratio: M≥0.90, F≥0.85, BMI≥30 kg m⁻²</td>
<td>Triglyceride≥150 mg dl⁻¹ and/or HDL-C: M&lt;35 mg dl⁻¹, F&lt;39 mg dl⁻¹</td>
<td>≥140/90 mm Hg</td>
<td>Urine albumin: &gt;20 µg min⁻¹ or Alb:Cr ≥30 mg g⁻¹</td>
<td></td>
</tr>
<tr>
<td>NCEP III (2001)</td>
<td>Three of the following</td>
<td>Waist circumference: M≥102 cm, F≥88 cm</td>
<td>Fasting glucose ≥110 mg dl⁻¹ or HDL-C: M&lt;40 mg dl⁻¹, F&lt;50 mg dl⁻¹</td>
<td>SAP≥130 or DAP≥85</td>
<td></td>
<td></td>
</tr>
<tr>
<td>IDF (2006)</td>
<td>Waist circumference (ethnicity specific values and two of the following risk factors)</td>
<td>Waist circumference: M≥94 cm, F≥80 cm</td>
<td>Fasting glucose ≥100 mg dl⁻¹ or diagnosis of diabetes type II</td>
<td>HDL-C: M&lt;40 mg dl⁻¹, or treatment of HDL dyslipidaemia</td>
<td>SAP≥130 or DAP≥85 or previous hypertension treatment</td>
<td></td>
</tr>
</tbody>
</table>
As defined by National Cholesterol Education Program (NCEP) or International Diabetes Federation (IDF) criteria, the incidence of metabolic syndrome is increasing. Estimates from the 2004 National Health and Nutrition Examination survey suggest a 34% overall incidence of obesity.13 Estimates of the overall prevalence of metabolic syndrome are nearly as high.14 These estimates are mirrored in surgical populations. In one study of patients undergoing cardiothoracic surgery,15 49% met criteria for metabolic syndrome. Another study16 had similar findings, with 33% having three and 7% having five contributing conditions. Growing evidence linking metabolic syndrome to colorectal,17 18 prostate,19 endometrial,20 bladder,21 and breast22 cancer also raise the possibility that the need for surgery will be more likely in metabolic syndrome patients. That outcomes of colorectal23 and hip and knee surgery24 are also poorer with the metabolic syndrome underscores the potential impact of metabolic syndrome on anaesthetic care.

Outcomes

Obesity alone may not always increase perioperative morbidity and mortality.25 In contrast, the metabolic syndrome clearly increases the risk of adverse perioperative outcomes. A study in non-diabetic patients undergoing coronary artery bypass grafting (CABG)26 found a 2.5-fold increase in mortality in patients meeting criteria for metabolic syndrome. In the other studies, CABG patients with metabolic syndrome were at higher risk for both graft failure27 and 10 yr mortality.28 The metabolic syndrome also affects outcomes after non-cardiac procedures. Perhaps the largest retrospective study to date29 reviewed data from 310 208 patients from the American College of Surgeons national surgical quality improvement program database and found a two-fold increase in mortality and a three- to seven-fold increased risk of acute renal failure in patients with the triad of obesity, hypertension, and diabetes. A recent study30 found a four-fold increased risk of stroke (4% compared with 1%) after carotid endarterectomy. In patients undergoing percutaneous nephrolithotomy,31 those meeting criteria for metabolic syndrome had a two-fold increased failure rate and a 2.5-fold increased rate of complications including infection, bleeding, and adjacent organ injuries. More recent work has identified an increased incidence of atrial fibrillation15 32 and postoperative cognitive dysfunction33 in metabolic syndrome patients as well.

Perioperative risk assessment, risk reduction, and management

Overview

No single screening test exists for diagnosis of the metabolic syndrome, and different combinations of components affect perioperative risk differently. Thus, one cannot assume that the risk or severity of CAD or of perioperative hypercoagulability is the same in all incarnations of the metabolic syndrome. This section will address the current approaches to preoperative evaluation and treatment of five common metabolic syndrome comorbidities: insulin resistance, OSA, cardiovascular disease, obstructive sleep apnoea and hypercoagulability.

Insulin resistance

Insulin resistance and consequent hyperglycaemia are core findings in the metabolic syndrome. Current literature reports increased perioperative complications with higher glucose levels for some patients,34 35 but conclusive data...
supporting a specific preoperative glucose threshold for case
cancellation due to unacceptable risk are lacking. Similarly,
data to support a specific glucose range for intraoperative
management are inconclusive. While some studies find
benefit to controlling intraoperative glucose levels, patient characteristics or the specific surgical procedure
might affect such benefit.

For the anaesthesiologist, two relevant questions with
respect to acute perioperative management of insulin resistance exist. The first is whether elective surgery should be postponed if a sufficiently high preoperative glucose level exists. Unfortunately, no consensus exists on a universally accepted threshold. Although HbA1c measurements correlate to preoperative glucose levels, their relationship to perioperative outcome is mixed. While a study in vascular surgery patients found higher mortality with HbA1C levels >6%, a larger trial in a wider surgical population found no correlation. No recommendation can thus be made regarding glucose or HbA1c thresholds for cancellation of surgery.

The second question is whether a target glucose level exists for intra- and postoperative management. Current evidence suggests that lower perioperative glucose levels might correlate with better outcomes, but efforts to achieve tighter glucose control lead to a higher incidence of hypoglycaemia. Because of this difficulty in implementation, locally derived target levels are likely to balance risk and gain.

**Obstructive sleep apnoea**

Not all patients with metabolic syndrome have OSA. The presence of OSA itself is independently associated both with individual elements of the metabolic syndrome and the syndrome itself. The presence of OSA increases the likelihood of metabolic syndrome itself (odds ratios ranging from 5 to 91). It is unclear, however, whether obesity itself represents the critical link between OSA and the metabolic syndrome, or whether other aspects of the metabolic syndrome play a role. Some studies find no correlation between OSA and metabolic syndrome even when controlling for obesity. Others find an association not only between OSA and obesity, but also between hypertensive, hyperglycaemic, and dyslipidaemic elements of the metabolic syndrome. Some authors have argued that the metabolic syndrome itself predisposes to OSA by increasing levels of proinflammatory cytokines such as interleukin-6 and tumour necrosis factor-α which themselves can induce sleepiness.

OSA clearly complicates anaesthetic management. In addition to increased incidences of hypertension and CAD, patients with OSA are more susceptible to both airway obstruction and depressed respiratory drive. Airway management, postoperative pain control, and decisions to discharge patients after day-case surgery are thus considerably more challenging. Perhaps the most significant issue facing anaesthesiologists is an exaggerated respiratory depression with sedatives, hypnotics, and opioids. This effect, possibly mediated by sleep deprivation induced changes in the brain adenosine levels, increases the likelihood of postoperative respiratory arrest, and complicates postoperative pain management.

Most patients with OSA are unlikely to carry the diagnosis when they present for preoperative evaluation. In addition, some symptoms of OSA (morning congestion, headache, daytime hypersomnolence, and snoring) are not usually elicited during the preoperative visit. Patients with metabolic syndrome already have at a minimum obesity and hypertension, which should trigger questioning targeted at other signs and symptoms of OSA. Preoperative screening questionnaires assist in identifying patients with sleep apnoea. The STOP questionnaire, is the most widely used and predicts postoperative complications with an odds ratio of 3.0. Other questionnaires are also effective in predicting postoperative complications but have limited sensitivity and specificity.

The definitive diagnostic test for OSA is the overnight polysomnogram or sleep study. This test allows the most accurate assessment of the number of obstructive episodes and the degree of desaturation with each episode. Unfortunately, a polysomnogram is time-consuming, labor intensive, expensive, and frequently difficult to obtain. Owing to the high pretest probability of OSA in patients with metabolic syndrome, delaying surgery for polysomnography is probably unwarranted. When coupled with observation during the immediate postoperative period, a preoperative sleep apnoea screening tool can effectively predict postoperative complications. Patients with both a high preoperative screening score and recurrent apnoeas or desaturations immediately after surgery were at extremely high risk of postoperative respiratory events (odds ratio = 21).

Few preoperative strategies exist to reduce intra- and postoperative risk of complications. Significant weight loss is unlikely, and surgical interventions have limited utility. Nasal or mask continuous positive airway pressure (CPAP) is effective in the chronic management of OSA, and is suggested for preoperative preparation. Although studies demonstrate increased pharyngeal size and improved arterial pressure control after 4–6 weeks of CPAP, evidence that such therapy reduces postoperative complications is limited. Postoperative outcome data specifying best practices are similarly unavailable. Careful observation, limited use of opioids and sedatives, hospital admission, and postoperative CPAP have all been advocated, although not all patients require all therapies.

Preoperative approaches to reducing perioperative risk centre on implementing preoperative CPAP therapy and addressing associated cardiovascular and metabolic abnormalities. The combination of preoperative screening and postoperative respiratory behaviour predicts postoperative respiratory complications, and can facilitate decisions to admit for close monitoring. Postoperative CPAP and advanced pain control techniques that limit opioid use also play a role in selected patients.

**Coronary artery disease and congestive heart failure**

The metabolic syndrome places patients at risk for the development of both CAD and CHF. In addition to the risks induced...
by component conditions such as hypertension, dyslipidaemia, and diabetes mellitus, increased adiponectin\(^62\) and coronary calcium deposits\(^63\) also play a role. Patients with metabolic syndrome demonstrate increased incidence of CAD. In a recent study of cardiac catheterization for chest pain\(^64\) patients with metabolic syndrome were twice as likely to have clinically significant CAD than those without. After adjustment for the Framingham risk score, however, no effect of metabolic syndrome was seen, suggesting that most of the risk derived from the individual components of metabolic syndrome rather than the syndrome itself. Other studies have also found that CAD risk is due to components of the metabolic syndrome rather than the syndrome itself\(^65\).

The incidence of CHF is also increased in the metabolic syndrome. In a 1988–94 National Health and Nutrition survey of 5500 patients\(^66\), the odds ratio of self-reported CHF was 1.8 compared with patients without the metabolic syndrome. It remains unclear whether this increased incidence was due to CAD, hypertension (and associate diastolic dysfunction), increased cardiac output due to obesity, OSA, pulmonary hypertension, or the syndrome itself. Neither altered lipid metabolism nor truncal obesity acted as independent risk factors for CHF, suggesting that hypertension and insulin resistance might produce the bulk of the increased risk.

In the light of the increased incidence of both CHF and CAD in metabolic syndrome patients, how should anaesthesiologists approach their preoperative risk stratification, intraoperative risk reduction, and postoperative management? Risk stratification for CAD should proceed as recommended by current ACC/AHA guidelines\(^67\). Current evidence supports the contention that because little CAD risk accrues from metabolic syndrome vs its components, evaluating cumulative CAD risk from each component condition (obesity, hypertension, altered lipids, and glucose intolerance) is reasonable. Because data supporting preoperative coronary revascularization for subsequent non-cardiac surgery are unclear\(^68\), medical optimization might be a reasonable option in patients for whom revascularization is particularly high risk.

No outcome data support the preoperative administration of aspirin in patients with the metabolic syndrome. However, aspirin might modulate both the increased inflammation and hypercoagulability seen in metabolic syndrome patients. Although aspirin increases the risk of perioperative bleeding, a recent study in patients at high risk for cardiac disease found benefit\(^69\). If postoperative neuraxial block is planned, current American Society of Regional Anaesthesia guidelines\(^70\) recommend against the concurrent use of aspirin and other anticoagulants with indwelling epidural catheterization. The high likelihood of postoperative heparin use for DVT prophylaxis might then prevent use of epidural pain strategies.

Although data in other high-risk groups suggest benefit\(^71\), no clinical trials address the perioperative use of statins in patients with metabolic syndrome. Similarly, no clinical trials address the preoperative use of angiotensin-converting enzyme (ACE) inhibitors. The SMILE study\(^72\) found less early and 1 yr mortality after MI with zofenopril use. Because ACE inhibitors predispose to intraoperative hypotension\(^73\), clinicians should carefully weigh risk and gain with respect to their preoperative use.

Intraoperatively, perioperative β-block might improve outcomes in patients with a revised cardiac risk index \(\geq 2\)\(^74\) and anaesthesiologists should recognize the increased likelihood of CAD. After operation, β-blockers and statins should be restarted promptly in patients chronically receiving those agents to avoid withdrawal\(^75\)\(^76\).

**Pulmonary disease**

Patients with metabolic syndrome are at risk for pulmonary disease. Restrictive lung disease due to severe obesity, pulmonary hypertension due to associated OSA, recurrent DVT, or both, and pulmonary oedema due to CHF, repeated negative pressure events, or both can all complicate anaesthetic care of patients with the metabolic syndrome. When anesthetized, placed supine, and paralysed, patients with metabolic syndrome are more prone to atelectasis, further reducing lung compliance and oxygenation. These changes increase the risk of postoperative respiratory complications.

As with OSA, not all components of pulmonary disease associated with the metabolic syndrome are modifiable in a practical time frame. The likelihood of significant intraoperative atelectasis due to restrictive lung disease is unlikely to change unless the degree of obesity is reduced. Preoperative evaluation should thus be directed at determining the severity of baseline disease (to aid postoperative management) and diagnosis of treatable conditions (pulmonary oedema, asthma, bronchitis, or pneumonia) that may affect surgical recovery.

Pulmonary function tests are unlikely to significantly add to perioperative management of patients with the metabolic syndrome. Restrictive lung disease will almost certainly be present, and quantitating the severity of restriction will likely not change management. The relationship of obstructive lung disease to metabolic syndrome is more complex. Patients with metabolic syndrome are more likely to have asthma-like symptoms\(^77\). However, these symptoms, including resting and post-exercise dyspnoea and wheezing, are non-specific, and can result from CHF, gastro-oesophageal reflux disease, and pulmonary hypertension and also from asthma.

The value of a preoperative chest radiography or arterial blood gas analysis is also limited. Patients with severe OSA, positional dyspnoea, low baseline oxygen saturation, dyspnoea at rest, or a baseline requirement for oxygen have a high likelihood of postoperative respiratory failure. In these patients, a preoperative chest radiogram provides a baseline for postoperative assessments of lung volume, pulmonary oedema, heart size, etc. Diagnostically, a preoperative chest radiogram can also facilitate the diagnosis of pneumonia or CHF, both potentially treatable conditions. Similarly, arterial blood gas analysis can facilitate postoperative management in patients at greater risk for respiratory failure. In cases of severe OSA or CHF, a chronic resting hypercarbia
Anaesthesia and the metabolic syndrome

is noted. In such patients, intra- and postoperative ventilation should target a normal pH instead of a normal $P_a_{CO_2}$.

Preoperative diagnosis and management can also modify the severity of other lung conditions associated with metabolic syndrome. Diagnosis of OSA in advance of surgery can allow initiation of preoperative CPAP therapy. Preoperative smoking cessation can reduce perioperative risk. If suspicion of recurrent pulmonary embolism (PE) is high, venous studies can be performed to identify DVT and patients can be anticoagulated (see below) or a filter placed to reduce the risk of perioperative PE. Inspiratory muscle training and preoperative treatment of pulmonary oedema and pulmonary infection might also reduce the risk of perioperative pulmonary complications.

Intraoperative strategies for airway management and ventilation in patients with metabolic syndrome have been described elsewhere. Strategies to reduce perioperative atelectasis (recruitment manoeuvres, positive end-expiratory pressure, reverse Trendelenberg positioning, extubation in the semirecumbent position, and postoperative CPAP) are generally helpful, although specific levels of positive end-expiratory pressure and duration of postoperative CPAP have not been determined. Similarly, the value of regional pain management strategies is unclear. Intraoperative management should focus on the increased risk of perioperative atelectasis, the potential for resting hypercapnia and other disorders of respiratory drive, and the increased likelihood of a difficult airway. After operation, CPAP use and regional pain management techniques (in high-risk patients) might be helpful.

Deep venous thrombosis

Clinical studies confirm a predisposition to DVT in patients with metabolic syndrome. In a case–control study, metabolic syndrome was independently associated with DVT (odds ratio = 1.94). A retrospective study found a four-fold increase in DVT in patients with metabolic syndrome (15% vs 4%). Current evidence, however, does not identify which component(s) of metabolic syndrome are most predictive. In another large series, a relationship between metabolic syndrome and DVT was found but related primarily to abdominal obesity and not to other syndrome components.

No outcome data exist to guide the perioperative management of hypercoagulability in metabolic syndrome patients. Because of the high likelihood of DVT and the greater impact of perioperative PE in metabolic syndrome patients with associated cardiac and pulmonary dysfunction, a careful preoperative evaluation for DVT should be performed. Insufficient data exist to recommend routine preoperative venous imaging, with one study found no difference in outcomes. Evidence of DVT on history or physical examination or a history of prior DVT should spur further imaging. A history of DVT makes patients high risk for perioperative DVT, particularly if high-risk surgery is involved. If DVT is diagnosed, then elective surgery should be delayed for at least 1 month and the patient should be systemically anticoagulated during that time. If surgery cannot be delayed, or if postoperative anticoagulation is contraindicated, prophylactic inferior vena cava (IVC) filter placement should be considered.

Current evidence is insufficient to determine whether preoperative IVC filter placement should occur for patients with no documented DVT. In gastric bypass patients, prophylactic IVC filter placement has been recommended for patients with venous stasis, BMI $>60$, truncal obesity, and obesity hypoventilation syndrome, or for patients with previous DVT or PE. However, other studies support only routine thromboprophylaxis with subcutaneous heparin, and use of lower extremity compression devices until ambulation. Until outcome data are available, it is reasonable to develop local criteria for IVC filter placement that incorporate prior history of DVT or PE, patient characteristics such as BMI, and the nature of planned surgery. No data yet exist to recommend preoperative aspirin for patients with the metabolic syndrome.

Intraoperatively, a similar lack of data prevents evidence-based deployment of risk-reducing measures. Existing data demonstrate a significant risk-reducing effect of regional anaesthesia but not all patients and procedures are amenable to a regional technique. Data are mixed on the value of intraoperative heparin infusions in addition to postoperative prophylaxis. While one study found a significant reduction in DVT after total hip replacement with intraoperative heparin infusion, another study found no benefit in patients undergoing total knee replacement. The incidence of metabolic syndrome in either study was not documented.

No data exist to support specific therapeutic approaches on the postoperative period. Postoperative strategies with demonstrated benefit in other patient groups might include compression boots, early mobilization, high-dose subcutaneous heparin, daily aspirin, and subcutaneous low-molecular-weight heparin. Because of the increased risk in metabolic syndrome patients, the acute onset of hypotension, new or increasing dyspnoea, or chest pain should raise suspicion of PE. Testing for D-dimer is not useful in high-risk hospitalized patients, and is likely not to have value in metabolic syndrome patients. Haemodynamically stable patients should undergo multidetector computed tomography evaluation, if possible, or venous Doppler evaluation. Echocardiographic examination can identify right heart strain or the presence of emboli in the main pulmonary arteries. When clinical suspicion is high, and the patient is haemodynamically unstable, a finding of right ventricular dysfunction on transoesophageal echocardiography might trigger thrombolytic therapy.

Strategies for overall risk reduction

Comorbidities associated with the metabolic syndrome clearly increase perioperative risk. Although less invasive surgical approaches do not normally fall under the decision control of anaesthesiologists, such strategies might be considered for patients at extremely high risk for complications.
due to the metabolic syndrome. Examples of alternative approaches to surgical intervention include endoscopic, laparoscopic, and robotic procedures. While potential benefits of these less invasive approaches include reduced inflammation, improved postoperative pain, and technical ease, no data exist to support such a practice and this approach has a morbidity of 1.5% to be an effective risk reduction tool. Currently, the answer is not obvious from initial risk estimates. Consider a procedure with a 3% complication rate (such as knee replacement). An odds ratio of 2 for perioperative morbidity with the metabolic syndrome then adds an additional 3% morbidity. If the cure rate for gastric bypass surgery is less than morbidity from the procedure alone is a question that will require large prospective trials.

The answer is not obvious from initial risk estimates. Consider a procedure with a 3% complication rate (such as knee replacement). An odds ratio of 2 for perioperative morbidity with the metabolic syndrome then adds an additional 3% morbidity. If the cure rate for gastric bypass surgery is less than morbidity from the procedure alone is a question that will require large prospective trials. The cumulative morbidity of a gastric bypass followed by a subsequent procedure is less than morbidity from the procedure alone is a question that will require large prospective trials.

The answer is not obvious from initial risk estimates. Consider a procedure with a 3% complication rate (such as knee replacement). An odds ratio of 2 for perioperative morbidity with the metabolic syndrome then adds an additional 3% morbidity. If the cure rate for gastric bypass surgery is less than morbidity from the procedure alone is a question that will require large prospective trials. The cumulative morbidity of a gastric bypass followed by a subsequent procedure is less than morbidity from the procedure alone is a question that will require large prospective trials.

The answer is not obvious from initial risk estimates. Consider a procedure with a 3% complication rate (such as knee replacement). An odds ratio of 2 for perioperative morbidity with the metabolic syndrome then adds an additional 3% morbidity. If the cure rate for gastric bypass surgery is less than morbidity from the procedure alone is a question that will require large prospective trials. The cumulative morbidity of a gastric bypass followed by a subsequent procedure is less than morbidity from the procedure alone is a question that will require large prospective trials. The cumulative morbidity of a gastric bypass followed by a subsequent procedure is less than morbidity from the procedure alone is a question that will require large prospective trials. The cumulative morbidity of a gastric bypass followed by a subsequent procedure is less than morbidity from the procedure alone is a question that will require large prospective trials.

The answer is not obvious from initial risk estimates. Consider a procedure with a 3% complication rate (such as knee replacement). An odds ratio of 2 for perioperative morbidity with the metabolic syndrome then adds an additional 3% morbidity. If the cure rate for gastric bypass surgery is less than morbidity from the procedure alone is a question that will require large prospective trials. The cumulative morbidity of a gastric bypass followed by a subsequent procedure is less than morbidity from the procedure alone is a question that will require large prospective trials. The cumulative morbidity of a gastric bypass followed by a subsequent procedure is less than morbidity from the procedure alone is a question that will require large prospective trials.

The answer is not obvious from initial risk estimates. Consider a procedure with a 3% complication rate (such as knee replacement). An odds ratio of 2 for perioperative morbidity with the metabolic syndrome then adds an additional 3% morbidity. If the cure rate for gastric bypass surgery is less than morbidity from the procedure alone is a question that will require large prospective trials. The cumulative morbidity of a gastric bypass followed by a subsequent procedure is less than morbidity from the procedure alone is a question that will require large prospective trials. The cumulative morbidity of a gastric bypass followed by a subsequent procedure is less than morbidity from the procedure alone is a question that will require large prospective trials. The cumulative morbidity of a gastric bypass followed by a subsequent procedure is less than morbidity from the procedure alone is a question that will require large prospective trials.

The answer is not obvious from initial risk estimates. Consider a procedure with a 3% complication rate (such as knee replacement). An odds ratio of 2 for perioperative morbidity with the metabolic syndrome then adds an additional 3% morbidity. If the cure rate for gastric bypass surgery is less than morbidity from the procedure alone is a question that will require large prospective trials. The cumulative morbidity of a gastric bypass followed by a subsequent procedure is less than morbidity from the procedure alone is a question that will require large prospective trials.
Anaesthesia and the metabolic syndrome


32 Girerd N, Pibarot P, Fournier D, et al. Middle-aged men with increased waist circumference and elevated C-reactive protein level are at higher risk for postoperative atrial fibrillation following coronary artery bypass grafting surgery. Eur Heart J 2009; 30: 1270–7


41 Chaney MA, Nikolov MP, Blakeman BP, Bakhos M. Attempting to maintain normoglycemia during cardiopulmonary bypass with insulin may initiate postoperative hypoglycemia. Anesth Analg 1999; 89: 1091–5

42 Coughlin SR, Mawdsley L, Mugarza JA, Calverley PM, Wilding JP. Obstructive sleep apnoea is independently associated with an increased prevalence of metabolic syndrome. Eur Heart J 2004; 25: 735–41


46 Vgontzas AN, Bixler EO, Chrousos GP. Sleep apnoea is a manifestation of the metabolic syndrome. Sleep Med Rev 2005; 9: 211–24


