Preoperative Evaluation of the Compromised Patient

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In this lecture I shall create four clinical situations with the same planned surgical procedure that explore various aspects of the preoperative assessment and preparation of a patient with organ system compromise – cardiac, pulmonary, renal or hepatic.

THE PATIENT WITH CARDIAC COMPROMISE

A 58-year old woman with chronic relapsing gallstone pancreatitis is scheduled for elective open cholecystectomy …the patient has a history of atypical chest pain on mild to moderate exertion ...

Assessment of Perioperative Cardiac Risk

According to Mangano [1], approximately 27 m anesthetics are given per year in the United States. Of these, about a third of patients, or 8 m, will have coronary artery disease (CAD) or cardiac risk factors. About one million will suffer some cardiac complication, and 50,000 will rule in for perioperative myocardial infarction. The care of these patients adds an estimated annual cost of $20 billion to the health care system.

It is obviously important to be able to predict which patients are at risk for perioperative cardiac complications. Multifactorial analysis of a large at risk population revealed the following significant cardiac risk factors (Table 1) [2]: (1) ischemic heart disease, i.e. angina, previous myocardial infarction, congestive heart failure (CHF); (2) diabetes; (3) renal insufficiency with serum creatinine (SCr) > 2 mg/dL and (4) poor cardiopulmonary functional status. The patient who has had a recent non-Q wave (subendocardial) myocardial infarction with new onset of dyspnea appears to be at particularly high risk, presumably because there is an area that is ischemic and in jeopardy of full-thickness (Q wave) infarction. Renal insufficiency is a cardiac risk factor through its association with diabetes, hypertension and peripheral vascular disease. These risk factors are significant in the context of “high risk surgery” – that is, major surgery of the thorax and abdomen.

The steps in preoperative evaluation of cardiac ischemia are listed in Table 2.

A careful history and physical are essential but may be quite misleading. It is helpful to use the Canadian Cardiovascular Society classification of the severity of angina. This defines grade I angina as that occurring with vigorous exercise only (e.g. sport such as tennis or jogging); grade II with moderate exercise (e.g. household activities such as gardening or cleaning); grade III with minimal exercise (e.g. personal activities such as dressing or bathing) and grade IV at rest (or during sleep). Unstable angina is defined as new onset angina, angina increasing in intensity or rest angina. It is important to recognize that myocardial ischemia can present as acute dyspnea rather than chest pain. The mechanism is the acute onset of left ventricular diastolic stiffness, which elevates left atrial pressure and evokes acute pulmonary congestion or edema. Silent ischemia (i.e. coronary artery disease in the absence of angina) may simply represent subclinical disease or that the patient's activity is restricted enough (usually by peripheral vascular disease) such that angina is not evoked. True silent ischemia notoriously occurs in the presence of conditions that induce autonomic neuropathy (severe diabetes, uremia) or where the cardiac nerves have been transected (transplanted heart).

The resting electrocardiogram (ECG) seldom provides an assessment of risk because in most patients the induction of myocardial ischemia requires the presence of stress (i.e. increased myocardial oxygen demand in the face of fixed supply). Stress testing implies the creation of increased oxygen demand through exercise or inotropic stimulation with dobutamine or decreased oxygen supply through the creation of a coronary steal syndrome with vasodilators such as dipyridamole or adenosine.

The exercise ECG has become a mainstay of assessment of cardiac risk. However in about a third of cases it may provide a false negative test, and does not give information regarding the potential for revascularization. The sensitivity of the exercise ECG as a predictor of diffuse or left main coronary artery disease is markedly enhanced if angina occurs at a low intensity of exercise, if there are diffuse ST changes, if angina persists after the cessation of exercise, if hypotension develops or if angina occurs in the presence of ventricular dysfunction at rest [3].

Certain conditions limit or prevent interpretation of the exercise ECG, including left bundle branch block (LBBB), ventricular pacing, pre-excitation syndromes (e.g. Wolff-Parkinson-White), pre-existing ST depression > 1 mm at rest, or exercise restriction through peripheral vascular disease (PVD). Others, such as recurrent angina following revascularization, mandate more detailed investigation. Both sets of conditions warrant non-invasive studies such as radionuclide imaging or dobutamine stress echocardiography [3].

Radionuclide imaging usually involves creation of a coronary steal syndrome with a coronary vasodilator such as dipyrimadole, which will reveal areas of impaired perfusion as a defect on a thallium scan. The scan is repeated in four hours, after dipyrimadole "wash out". A fixed defect suggests an area of scarring; resolution implies reversible ischemia that might benefit from revascularization.

The dobutamine stress echocardiogram is currently the most popular non-invasive stress test because it is simple, reproducible and requires no radioactive tracer. It enables assessment of a baseline ejection fraction (EF) and its response to inotropic stress (a decrease in EF implies diffuse coronary artery disease), the presence of new or worsened wall motion abnormality (WMA) in response to inotropy. In a study on patients undergoing vascular surgery by Poldermans et al, 56 out of 181 patients had a positive dobutamine stress echo [4]. Perioperative cardiac events (ischemia, infarction etc) occurred in 18 patients, all of whom had had a positive stress test; no events occurred in patients who had a negative stress test. The odds ratio of an event with a new WMA was 45:1.

Perioperative Planning and Preparation

An approach to the high-risk patient has been recommended by Fleisher and Eagle [2]. A patient scheduled for high-risk surgery who has three or more risk factors (Table 1) should undergo non-invasive testing. If this is positive, the patient should undergo cardiac catheterization. If this reveals left main or diffuse coronary artery disease, the patient should go on to coronary artery bypass grafting. Patients with one or two vessel coronary artery disease should proceed to percutaneous coronary angioplasty (PTCA).

Correct timing of elective surgery after PTCA is essential. All patients are placed on antithrombotic therapy after PTCA with clopidogrel (for four weeks) and aspirin (indefinitely). If surgery is performed within four weeks, there is an increased bleeding risk if antiplatelet therapy is continued. On the other hand, there is increased thrombosis risk if therapy is withheld.

There is increasing evidence that perioperative beta blockade provides myocardial protection. Their use in high-risk patients is recommended in the 2002 update of the American College of Cardiology and American Heart Association (ACC/AHA) guidelines for perioperative cardiovascular evaluation for noncardiac surgery [5].

The most dramatic support for this position thus far is from another study by Poldermans et al in which high-risk patients undergoing vascular surgery were randomized to the beta-blocker bisoprolol or placebo [6]. Treatment was started ≥ 7 days preoperatively with the goal of achieving a resting heart rate of ≤ 60 beats / min, and continued for 30 days postoperatively. In patients who received the beta-blocker, there was a 91% reduction in myocardial infarction or cardiac death. It is not known how much protection would be provided by starting beta-blockade shortly before or at the time of surgery, but it would appear to be prudent to do so in high-risk patients, and continue it for as long as possible after surgery. There is also some evidence that the administration of alpha-2 agonists such as mivazerol may also provide perioperative cardiac protection [7].

THE PATIENT WITH PULMONARY COMPROMISE

A 58-year old woman with chronic relapsing gallstone pancreatitis is scheduled for elective open cholecystectomy …the patient has a history of 100 pack years smoking and has dyspnea when walking one block ...

Assessment of Perioperative Pulmonary Risk

Patients with pulmonary compromise are at risk to perioperative complications that include hyperreactive airways and postoperative ventilator dependence as a consequence of retained secretions, atelectasis, pneumonia, respiratory failure, pneumothorax or bronchopleural fistula. The site of the surgical incision plays an important role in determining the risk of these complications: likelihood increases from median sternotomy to upper abdominal, to thoracotomy and ultimately thoracoabdominal surgery, which imposes the greatest risk. After thoracotomy, functional residual capacity (FRC) is decreased by 30-40% for up to three weeks.

Key factors in the history that indicate pulmonary compromise include heavy smoking (expressed in pack years), current smoking, effort-induced dyspnea, productive cough with infected sputum, wheezing, bronchitis and pneumonia. A history of hospitalizations is especially significant.

The spectrum of chronic obstructive pulmonary disease (COPD) is illustrated in Fig 1. At one extreme is chronic bronchitis, characterized by excessive sputum production, predisposition to atelectasis, intrapulmonary shunting and hypoxemia. PEEP is helpful in maintaining FRC and improving oxygenation. At the other is emphysema, characterized by expiratory airway collapse and air trapping, increased dead space and hypercarbia. PEEP is not helpful and may exacerbate dead space. Many patients fall somewhere between the two extremes.

The spectrum of airway obstruction is listed in Table 3. Extrinsic, allergic asthma has its onset in childhood, usually with well-defined allergies, and responds to prophylaxis with cromolyn, a mast cell stabilizer. Intrinsic asthma is of adult onset, with poorly defined allergies; cromolyn is ineffective but airway obstruction responds to bronchodilators. A third category is COPD with superadded acute airway obstruction, which is resistant to cromolyn and responds poorly to bronchodilators; steroids are usually required for perioperative protection or relief.

A clinical caveat is that audible wheezing may be absent with the most severe bronchospasm, due to slow expiratory flow. A simple method for a semi-quantitative estimate of expiratory air flow is the forced expiratory time (FET). The patient is asked to provide a maximal exhalation while the observer listens over trachea. An FET > 6 sec implies an $FEV_1 < 1$ liter and the potential for CO_2 retention.

The simplest laboratory pulmonary function test (PFT) is the spirogram (Fig. 2). Note that a capacity is the sum of two or more volumes. The forced vital capacity (FVC) is achieved by a maximal exhalation after a maximal inhalation. The FRC is defined as the volume of gas in the lung at the end of a normal tidal exhalation. Interpretation of standard PFTs and their deviation in severe emphysema are provided in Table 4.

There is increasing interest in the prognostic value of exercise testing to assess predicted postoperative (ppo) function. Wyser et al. devised an algorithmic approach utilizing cardiac evaluation, forced expiratory volume in 1 sec (FEV_1), diffusing capacity of the lungs for carbon monoxide (DLCO), and maximal oxygen uptake (VO_2max), together with their respective ppo values (FEV_1-ppo, DLCO-ppo, and VO_2max-ppo) based on radionuclide perfusion scans[8]. They were able to stratify patients as functionally inoperable or operable, and this more selective approach resulted in a substantial decrease in their postoperative complication rate.

Perioperative Planning and Preparation

There are several preoperative interventions that may improve the patient's functional pulmonary reserve and decrease the risk of perioperative complications. Patients with severe lung disease may have pulmonary hypertension and right-sided heart failure. Transthoracic echocardiography may provide an assessment and allow optimization of cardiac status (digoxin, diuretics etc.). Cessation of active smoking is the ideal but seldom accomplished. To restore normal ciliary function, smoking must be stopped at least 6 - 8 weeks before anesthesia. However, even stopping 24-48 hrs prior to surgery may allow a decrease in carboxyhemoglobin, which impedes hemoglobin oxygen off-loading and which may reach levels of 10% in heavy smokers. If there are signs of infection (cough, altered sputum) a course of appropriate antibiotics should be administered for at least one week prior to elective surgery. Elective surgery should not be performed in patients who are actively wheezing without a trial of beta-2 agonist, anticholinergic and/or steroid inhalers. Fixed airway obstruction or emphysema warrants the use of oral and intravenous steroid perioperative prophylaxis. If at all possible, patients should be taught how to use incentive spirometry before they come to surgery.

THE PATIENT WITH RENAL COMPROMISE

A 58-year old woman with chronic relapsing gallstone pancreatitis is scheduled for elective open cholecystectomy …the patient has a history of hypertension and a creatinine of 3.4 mg / dL ...

Assessment of Perioperative Renal Risk [9]

Patients with advanced renal dysfunction or failure have markedly depleted fluid reserve. In anuric patients, excess sodium intake exacerbates edema and hypertension; excess water quickly induces hyponatremia. In non-oliguric or polyuric syndromes, urine output may be reassuring, but it is fixed and patients are unable to concentrate urine in the face of hypovolemia.

Chronic metabolic acidosis is prevalent, with an increased anion gap filled by unexcreted sulfates and phosphates. It is usually mild and well compensated by chronic hyperventilation, but patients lack buffer base. Even moderate degrees of hypercarbia, diarrhea, catabolism or shock quickly lead to an acute and profound decrease in pH. A characteristic perioperative scenario is illustrated in Table 5. Acidosis exacerbates hyperkalemia, because transcellular potassium flux is closely associated with extracellular pH. A 0.1 change in pH may result in a 0.5 mEq/L change in potassium. For example, a fall in pH from 7.4 to 7.2 may cause serum potassium to increase from 5.0 to 6.0 mEq/L.

The relationship between serum creatinine (SCr) and glomerular filtration rate (GFR) is not linear, but is inversely exponential. Thus, a doubling of SCr implies a halving of GFR (Fig. 3). This concept is most important in the early stages of renal insufficiency. For example, an increase in SCr from 0.8 to 1.6 mg/dL may be dismissed as trivial but represents a 50% decrease in GFR. The GFR declines with age, from about 125 mL/min in a young healthy adult to about 60 mL/min in an 80-yr old (Fig. 4). Because SCr does not increase above normal levels until the GFR declines below 50 mL/min (and even lower in cachectic patients), the SCr does not reveal that an octogenarian has less than half the renal reserve of a 20 yr-old.

Perioperative Planning and Preparation

In anuric or oliguric patients, maintenance fluid should be restricted to urine output plus 500 mL/day (insensible loss), but all direct fluid losses (e.g. vomiting, diarrhea) should be replaced promptly. It should be anticipated that most patients will have a chronic metabolic acidosis with compensatory respiratory alkalosis. During anesthesia, minute ventilation should be increased accordingly (Table 5).

Should preoperative hemodialysis be performed in the patient presented above? Hemodialysis reliably corrects the most lethal complications of acute renal failure, that is, pulmonary edema, hyperkalemia and metabolic acidosis. It is also effective in relieving the manifestations of acute uremia, that is encephalopathy, enteropathy, serositis and to some degree, thrombocytopathy. However, it is less effective or ineffective in reversing most of the consequences of chronic uremia, including anemia (now usually avoided by the use of human recombinant erythropoietin, EPO), renal osteodystrophy, peripheral neuropathy, impaired resistance to sepsis and poor wound healing.

Patients with acute or chronic renal insufficiency are resistant to diuretic therapy, and require higher doses or dual segment therapy (that is, the combination of a loop diuretic such as furosemide with a thiazide diuretic such as metolazone). Low-dose dopamine may increase urine flow but its impact on enhancing GFR is proportional to renal function [10].

Should we perform dialysis before surgery in the patient presented above? The answer is no. First time dialysis should not be performed immediately prior to surgery. Hemodialysis has a number of adverse effects, including hypovolemia, hypotension, myocardial ischemia and electrolyte imbalance (potassium, magnesium, phosphate). Routine hemodialysis should be performed at least 24 hrs before elective surgery. A dysequilibrium syndrome, caused by movement of water into the still hypertonic brain, may present as seizures or other neurologic abnormalities, and is most likely to occur with the first dialysis treatment in severely uremic patients. Peritoneal dialysis confers stable hemodynamics but the abdominal distension may compromise FRC and there is increased risk of peritoneal infection. Continuous veno-venous hemodialysis (CVVHD) allows the removal of large quantities of fluid with little hemodynamic perturbation, but requires anticoagulation.

Thus, the in the patient presented, the recommendation would be to manage her conservatively during surgery and then start dialysis postoperatively, should it become necessary. Of note, dialysis can be performed during cardiopulmonary bypass in patients undergoing cardiac surgery.

THE PATIENT WITH HEPATIC COMPROMISE

A 58-year old woman with chronic relapsing gallstone pancreatitis is scheduled for elective open cholecystectomy ... the patient has a history of ascites, small upper GI bleeds and occasional encephalopathy ...

Assessment of Perioperative Liver Risk [11]

It is helpful to distinguish the manifestations of hepatocellular failure from those of portal hypertension.

Characteristics of primary liver failure are encephalopathy, spider nevi, hormonal changes, anasarca and renal insufficiency. Ammonia, normally converted to urea via the hepatic arginine cycle, accumulates and BUN may remain < 10 mg/dL even in the face of GI bleeding or renal insufficiency. Portal hypertension presents with ascites, esophageal and gastric variceal bleeding.

The hepatorenal syndrome (Fig. 5) is an intense prerenal syndrome characterized by azotemia and oliguria with low (< 10 mEq/L) urine sodium. It is induced by the renoconstrictive effects of absorbed endotoxin and is resistant to fluid replacement. That the milieu is responsible is evidenced by the observation that if the kidney transplanted into a patient with normal hepatic function will begin to function normally again.

Hepatic encephalopathy is classified into four levels of severity (Table 6). Constructional apraxia, a subtle manifestation of grade 1 encephalopathy, implies the inability to construct simple geometric figures and can easily be assessed in the preoperative clinic.

Predictors of poor outcome in liver disease are listed in Table 7 [12].

Chronic persistent hepatitis represents low risk, and elective surgery should proceed. In chronic active hepatitis, the asymptomatic, anicteric patient is at low risk. However, the presence of jaundice, elevated liver enzymes or coagulopathy mandates deferment of elective surgery until these can be resolved or improved.

Cirrhosis represents a high risk co-morbid condition. In a review of 733 patients with cirrhosis undergoing surgery at the Mayo Clinic between 1981 and 1990, the perioperative mortality was 11.6%. Morbidity was 30.1%, mostly from postoperative pneumonia[13]. In advanced cirrhosis, the combination of portal hypotension and hypoalbuminemia (i.e. low oncotic pressure) results in ascites and intravascular hypovolemia. This in turn stimulates secondary hyperaldosteronism with urinary sodium retention and potassium loss, resulting in hypokalemic alkalosis. Alkalosis worsens encephalopathy by the process of non-ionic diffusion trapping. When extracellular hydrogen ion concentration decreases, lipophobic ionized ammonium ion converts to lipophilic non-ionized ammonia, which can diffuse across the blood-brain barrier.

The Child-Turcotte-Pugh classification (Table 8) is a useful guide to perioperative risk in the cirrhotic patient [11,12,14]. Risk is assessed as A (low), B (moderate) or C (high) based on severity of jaundice, hypoalbuminemia, coagulopathy (prolonged prothrombin time), encephalopathy, ascites and nutritional depletion. Elective surgery may proceed with caution for Child's A, and with preoperative preparation for Child's B. Child's C patients are not candidates for elective surgery.

The surgical procedure itself is a major determinant of outcome in these patients. Laparotomy decreases hepatic perfusion through visceral traction and vasodilatation. Blood loss is the most important determinant of liver injury, and is excessive in redo abdominal surgery due to the association of vascular adhesions, portal hypertension and coagulopathy. Other high-risk surgical procedures include emergency surgery (especially laparotomy), cardiac surgery with cardiopulmonary bypass (coagulopathy), ileostomy and colostomy (prone to ascitic leaks), and hepatic tumor resection (residual liver injury). Cirrhotic patients have an increased propensity for pigment gallstones, but open cholecystectomy is a particularly high-risk procedure because portal hypertension and coagulopathy combine to induce bleeding from the gall bladder bed. Consideration should be given to ERCP or cholecystostomy.

Perioperative Planning and Preparation

Certain measures can be taken preoperatively that may decrease perioperative risk. Fluid balance can be optimized by appropriate salt and water restriction, but attempts at removing ascites by loop diuresis can worsen hepatic perfusion. Spironolactone, an aldosterone antagonist, is quite effective but may exacerbate potassium retention in patients with hepatorenal syndrome. It has slow onset and offset and should be discontinued 2-3 days before surgery if there is a potential for postoperative renal dysfunction. Encephalopathy can be improved by standard measures such as protein restriction, lactulose or neomycin and the correction of metabolic alkalosis. Attempts should be made if possible to improve severe nutritional depletion by enteral or parenteral nutrition. The use of branched-chain amino acid formulations may decrease encephalopathy but do not induce positive nitrogen balance. In high-risk patients the response of the prothrombin time to subcutaneous Vitamin K should be assessed – if it remains more than three seconds above control the risk is similar to that of Child’s C regardless of other parameters.

Aspiration of ascites may allow better positioning of patients for anesthesia and decrease impairment of FRC, but it re-accumulates quickly. Rapid removal of large amounts can cause acute decompensation through intravascular hypovolemia. In patients with intractable ascites consideration should be given to preoperative placement of a transjugular intrahepatic portasystemic shunt (TIPS). There is a risk of pulmonary congestion, endotoxemia and encephalopathy from diversion of portal blood into the systemic circulation, but TIPS often provides dramatic relief of ascites as well as improvement in renal perfusion.

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LEGENDS

Figure 1: The Spectrum of Chronic Obstructive Pulmonary Disease

The figure represents the two major forms of chronic obstructive pulmonary disease (COPD). Many patients lie somewhere between the two and may have overlapping features.

Figure 2: The Spirogram.

Note that a capacity equals the sum of two or more volumes. TLC = total lung capacity, TV = tidal volume, IRV = inspiratory reserve volume, ERV = expiratory reserve volume, FVC – forced vital capacity, RV = residual volume, FRC = functional residual capacity.

Figure 3. Relationship between GFR and Serum Creatinine

The relationship between glomerular filtration rate (GFR) and serum creatinine (SCr) is inverse and exponential. A doubling of the serum creatinine represents a 50% decrease in GFR. In this example, an increase in SCr from 0.5 to 1.0 mg/dL (within the normal range) represents a decrease in GFR from 150 to 75 mL/min – a loss of half the renal reserve. By the time SCr exceeds normal, more than half of renal function has been lost.

Figure 4. Normal Decrease in GFR with Age

This schematic illustrates the normal loss of renal reserve with age. At age 20 yrs, glomerular filtration rate (GFR) is about 125 mL/min. By the age of 60 yrs it has decreased to 80 mL/min and by the age of 80 yrs to 60 mL/min. The stippled line represents the GFR (usually 50 mL/min) below which the SCr increases above the normal range, and the dashed line represents frank acute renal failure. Note that an octogenarian may have the same serum creatinine as a 20 yr-old (or lower, if muscle mass is depleted) with less than half the renal reserve.

Figure 5. Hepatorenal Syndrome.

This cartoon illustrates the postulated mechanism of hepatorenal syndrome. Endotoxin, normally detoxified by binding to bile pigments, is absorbed from the gut and bypasses the liver because of portasystemic shunting and Kupffer cell dysfunction. In the renal circulation, endotoxin induces vasoconstriction as well as renal tubular injury. An intense prerenal state is created with oliguria and low urinary sodium (< 10 mEq/L) that is refractory to fluid replacement. A similar mechanism occurs in sepsis.