Preoperative Evaluation of the Compromised Patient

Robert N. Sladen, MD

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-yr-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 million anesthetics are given each year in the United States. Of these, approximately one-third of patients (8 million) will have coronary artery disease (CAD) or cardiac risk factors. Approximately one million will suffer some cardiac complication, and approximately 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 approximately one-third of cases it may provide a false negative test, and it 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

Table 1. Cardiac Risk Factors (2)

1. Ischemic heart disease
   – angina, previous myocardial infarction, congestive heart failure
2. Diabetes
3. Renal insufficiency (SCr >2 mg/dL)
4. Poor cardiopulmonary functional status
5. High risk surgery*

SCr = serum creatinine. * High risk surgery is defined as thoracic, intraperitoneal or suprainguinal vascular surgery.

Table 2. Preoperative Evaluation of Cardiac Ischemia

1. History and physical
2. Resting ECG
3. Exercise electrocardiography
4. Radionuclide stress testing*
5. Dobutamine stress echocardiography*
6. Cardiac catheterization

ECG = electrocardiogram. *Noninvasive tests indicated when exercise electrocardiography provides inadequate information.

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 after revascularization, mandate more detailed investigation. Both sets of conditions warrant noninvasive 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 dipyridamole, which will reveal areas of impaired perfusion as a defect on a thallium scan. The scan is repeated in 4 h, after dipyridamole "washout". 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), or 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. (4), 56 of 181 patients had a positive dobutamine stress echo. 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.

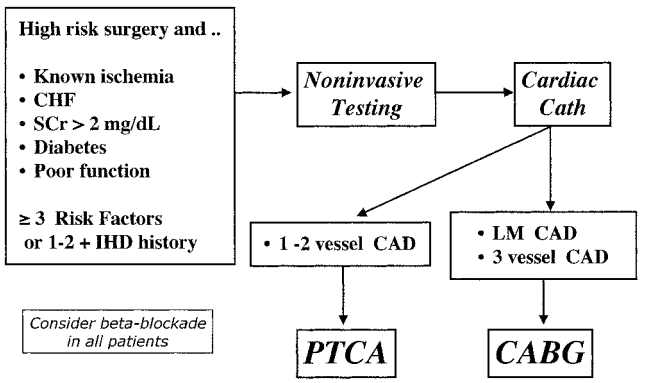

Figure 1. Strategy for evaluation and management of high-risk patients. In a patient undergoing high-risk surgery, the presence of three or more risk factors (or two with a history of ischemic heart disease) warrants noninvasive testing (radionuclide scan or dobutamine stress echo). If noninvasive testing is strongly positive, cardiac catheterization is indicated. If it is positive, coronary revascularization should be considered if justified independently of noncardiac surgery. CHF = congestive heart failure; SCr = serum creatinine; IHD = ischemic heart disease; cath = catheterization; CAD = coronary artery disease; LM = left main; PTCA = percutaneous coronary angioplasty; CABG = coronary artery bypass grafting. (Modified from: Fleisher LA, Eagle KA: Clinical practice. Lowering cardiac risk in noncardiac surgery. N Engl J Med 2001; 345: 1677–82.)

Perioperative Planning and Preparation

Figure 1 summarizes an approach to the high-risk patient as recommended by Fleisher and Eagle (2). A patient scheduled for high-risk surgery who has three or more risk factors (Table 1) should undergo noninvasive 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. However, there is increased thrombosis risk if therapy is withheld.

There is increasing evidence that perioperative β blockade provides myocardial protection. Its 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 is from another study by Poldermans et al. (6) in which high-risk patients undergoing vascular surgery were randomized to the β-blocker bisoprolol or placebo. Treatment was started ≥7 d preoperatively with the goal of

achieving a resting heart rate of ≤60 bpm, and continued for 30 d postoperatively. In patients who received the β-blocker, there was a 91% reduction in myocardial infarction or cardiac death. It is not known how much protection would be provided by starting β-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 α-2 agonists such as mivazerol may also provide perioperative cardiac protection (7).

The Patient with Pulmonary Compromise

A 58-yr-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 3 wk.

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 Figure 2. At one extreme is chronic bronchitis, characterized by excessive sputum production, predisposition to atelectasis, intrapulmonary shunting, and hypoxemia. Positive end-expiratory pressure is helpful in maintaining functional reserve capacity (FRC) and improving oxygenation. At the other is emphysema, characterized by expiratory airway collapse and air trapping, increased dead space, and hypercarbia. Positive end-expiratory pressure 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 chronic obstructive pulmonary disease with superadded acute airway obstruction, which is resistant to cromolyn and responds poorly to bronchodilators; steroids are usually required for perioperative protection or relief.

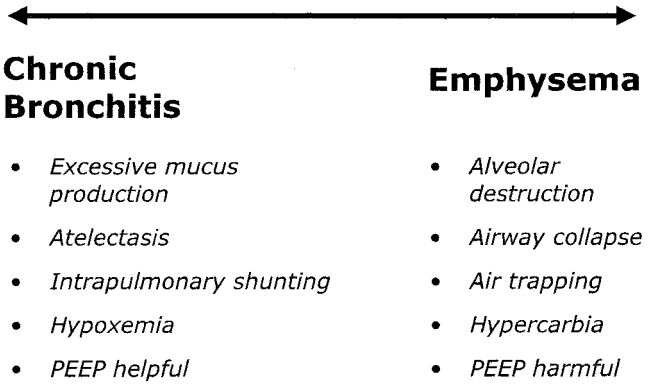

Figure 2. The spectrum of chronic obstructive pulmonary disease. The figure represents the two major forms of chronic obstructive pulmonary disease (COPD). Many patients lie somewhere between the two and may have overlapping features.

Table 3. The Clinical Spectrum of Airway Obstruction

1. *Extrinsic, allergic asthma*	
	Childhood onset
	Well-defined allergies
	Responds to prophylaxis with cromolyn
	Responds to bronchodilators, steroids
2. *Intrinsic asthma*	
	Adult onset
	Poorly defined allergies
	Cromolyn ineffective
	Responds to bronchodilators, steroids
3. *COPD with superadded acute airway obstruction*	
	Elderly onset
	Resistant to cromolyn
	Responds poorly to bronchodilators
	May respond to steroid prophylaxis or therapy

COPD = chronic obstructive pulmonary disease.

A clinical caveat is that audible wheezing may be absent with the most severe bronchospasm, as a result of slow expiratory flow. A simple method for a semiquantitative estimate of expiratory airflow is the forced expiratory time (FET). The patient is asked to provide a maximal exhalation while the observer listens over the trachea. An FET >6 s implies an FEV_1 <1 L and the potential for CO_2 retention.

The simplest laboratory pulmonary function test (PFT) is the spirogram (Fig. 3). 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

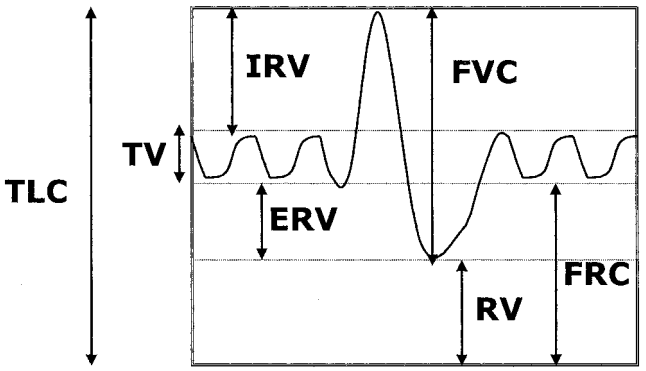

Figure 3. The spirogram. Note that a capacity equals the sum of two or more volumes. TLC = total lung capacity; TV = tidal volume; IRV = inspiratory reserve volume; ERV = expiratory reserve volume; FVC = forced vital capacity; RV = residual volume; FRC = functional residual capacity.

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. (8) devised an algorithmic approach utilizing cardiac evaluation, forced expiratory volume in 1 s (FEV_1), diffusing capacity of the lungs for carbon monoxide (DLCO), and maximal oxygen uptake ($\dot{V}o_2max$), together with their respective ppo values (FEV_1-ppo, DLCO-ppo, and $\dot{V}o_2max$-ppo) based on radionuclide perfusion scans. 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 (e.g., digoxin, diuretics). Cessation of active smoking is the ideal but is seldom accomplished. To restore normal ciliary function, smoking must be stopped at least 6–8 wk before anesthesia. However, even stopping 24–48 h before 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 1 wk before elective surgery. Elective surgery should not be performed in patients who are actively wheezing without a trial of β-2 agonist, anticholinergic, and/or steroid inhalers. Fixed airway obstruction or emphysema warrants the use of oral and IV 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-yr-old woman with chronic relapsing gallstone pancreatitis is scheduled for elective open cholecystectomy. The patient has a history of hypertension and a creatinine level of 3.4 mg/dL.

Assessment of Perioperative Renal Risk

Patients with advanced renal dysfunction or failure have markedly depleted fluid reserve (9). In anuric patients, excess sodium intake exacerbates edema and hypertension; excess water quickly induces hyponatremia. In nonoliguric 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 (Fig. 4). 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. 5). 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 approximately 125 mL/min in a young healthy adult to approximately 60 mL/min in an 80-yr old (Fig. 6). 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.

Table 4. Pulmonary Function Tests

Test	Normal	Explanation	Severe Emphysema
Total lung capacity (TLC)	5–6 L	Distinguishes between restrictive (decreased TLC) and obstructive (increased TLC) lung disease	7–8 L (>120% of predicted)
Residual volume (RV)	33% of TLC	Increased in emphysema	= 50% of TLC
Forced vital capacity (FVC)	4–5 L	Indicates pulmonary reserve	3.5 L (<75% of predicted)
Forced expiratory volume in 1 sec (FEV_1)	75% of FVC	Large airway flow (effort dependent)	1.75 L (<50% of FVC)
Forced expiratory flow (25%–75%) ($FEF_{25–75\%}$)	3–5 L/sec	Small airway flow (effort independent)	0.9 L/sec (<20% of predicted)
Maximal voluntary ventilation (MVV)	70–100 L/min	Measure of endurance or fatigue	<35 L/min (<50% of predicted)
Diffusion capacity for CO (DL_{CO})	80–10%	Represents functional lung area	<30%

Useful pulmonary function tests and their normal values are shown. In the last column, values representative of a patient with severe emphysema are listed for comparison.

Table 5. Perioperative Acidosis in Renal Failure

Time	$PaCO_2$	pH	AB	K
Preoperative	32	7.32	17	5.0
Intraoperative	40	7.25	18	5.3
Postoperative (early)	44	7.21	19	5.6
Postoperative (later)	48	7.18	19	5.9

AB = actual bicarbonate; K = serum potassium. The patient is a 35-yr-old diabetic undergoing cadaveric renal transplant, who had delayed graft function in the early postoperative period. The preoperative arterial blood gas reveals chronic, partially compensated metabolic acidosis with serum potassium at the upper limit of normal. In the operating room the patient is provided with standard minute ventilation during general anesthesia and the $PaCO_2$ increases to normal with a decrease in pH and increase in potassium. In the recovery room, the patient's trachea is extubated and he develops a mild degree of hypercarbia, with a further decrease in pH and increase in potassium. A little later, hypercarbia has increased to moderate levels but because of the absence of buffer base, the pH decreases to less than 7.2 and the potassium increases to dangerous levels. From: Sladen, RN: Anesthetic considerations for the patient with renal failure. Anesthesiol Clin North America 2000; 18: 863–82. Table 1, p 865. Used with permission.

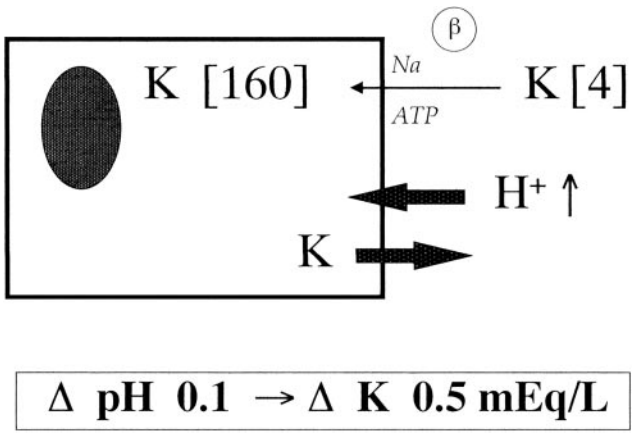

Figure 4. Potassium flux. Schematic illustration of intracellular potassium (K) flux. The 40:1 ratio between intracellular K concentration (160 mEq/L) and extracellular K concentration (4 mEq/L) is maintained by an active sodium (Na) adenosine triphosphatase (ATPase) pump at the cell membrane. Beta-adrenergic (β) agonists stimulate the Na ATPase pump and enhance K uptake into the cell; β-adrenergic antagonists have the opposite effect. The lower section of the figure illustrates the effect of extracellular acidosis, i.e., an increase in hydrogen ion (H^+) concentration causes H^+ to move into the cell along its concentration gradient. To maintain electrical neutrality K moves out of the cell and causes extracellular hyperkalemia. A change (Δ) of 0.1 in. pH results in a change of approximately 0.5 mEq/L in serum potassium concentration. For example, a decrease in pH from 7.4 to 7.2 could result in an increase in serum potassium of 1.0 mEq/L, e.g., from 5.0 to 6.0 mEq/L. (From: Sladen RN: Anesthetic considerations for the patient with renal failure. Anesthesiol Clin North America 2000; 18: 863–82, x. Used with permission.)

Perioperative Planning and Preparation

In anuric or oliguric patients, maintenance fluid should be restricted to urine output plus 500 mL/d (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

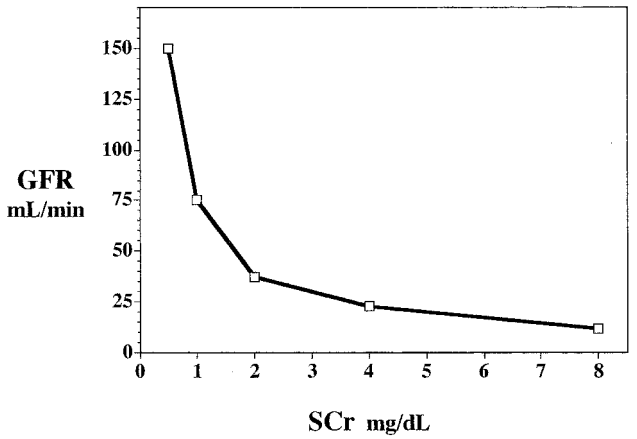

Figure 5. Relationship between GFR and serum creatinine. The relationship between glomerular filtration rate (GFR) and serum creatinine (SCr) is inverse and exponential. A doubling of the serum creatinine represents a 50% decrease in GFR. In this example, an increase in SCr from 0.5 to 1.0 mg/dL (within 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.

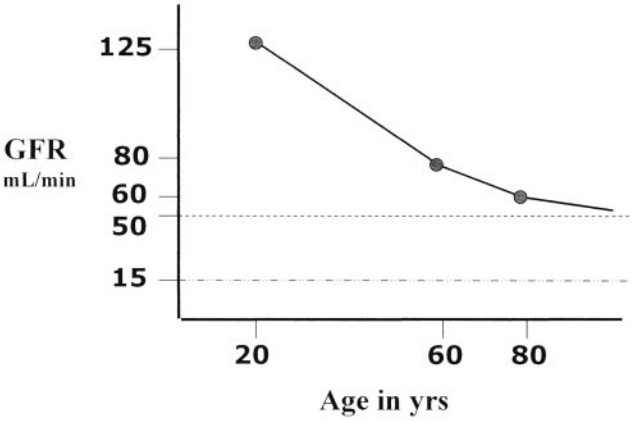

Figure 6. Normal decrease in GFR with age. This schematic illustrates the normal loss of renal reserve with age. At age 20 yr, glomerular filtration rate (GFR) is approximately 125 mL/min. By the age of 60 yr it has decreased to 80 mL/min and by the age of 80 yr to 60 mL/min. The stippled line represents 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.

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 before 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 h 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, 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-yr-old woman with chronic relapsing gallstone pancreatitis is scheduled for elective open cholecystectomy. The patient has a history of ascites, small upper gastrointestinal bleeds, and occasional encephalopathy.

Assessment of Perioperative Liver Risk

It is helpful to distinguish the manifestations of hepatocellular failure from those of portal hypertension (11).

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 gastrointestinal bleeding or renal insufficiency. Portal hypertension presents with ascites and esophageal and gastric variceal bleeding.

The hepatorenal syndrome (Fig. 7) 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 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

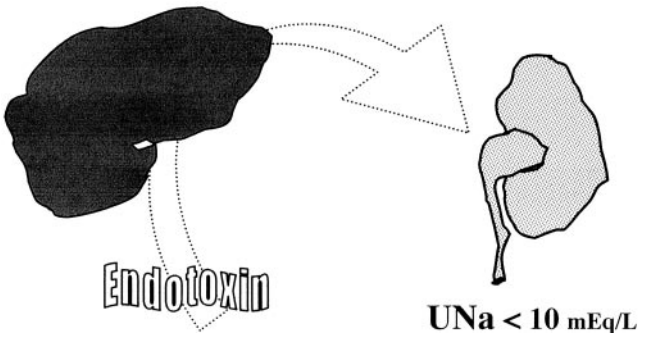

Figure 7. Hepatorenal syndrome. This cartoon illustrates the postulated mechanism of hepatorenal syndrome. Endotoxin, normally detoxified by binding to bile pigments, is absorbed from the gut and bypasses the liver because of portasystemic shunting and Kupffer cell dysfunction. In the renal circulation, 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.

Table 6. Grading of Hepatic Encephalopathy

Level 1: Confabulation, apraxia
Level 2: Confusion, asterexis
Level 3: Stupor
Level 4: Coma

Table 7. Liver Disease: Predictors of Poor Outcome

- Acute viral or alcoholic hepatitis
- Chronic active hepatitis with jaundice, symptoms
- Cirrhosis (Child's C)
- Ileostomy, colostomy
- Cholecystectomy
- PT >3 sec prolonged despite Vitamin K
- Emergent surgery

PT = prothrombin time.

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 nonionic diffusion trapping. When extracellular hydrogen ion concentration decreases, lipophobic ionized ammonium ion converts to lipophilic nonionized ammonia, which can diffuse across the blood-brain barrier.

Table 8. Child-Turcotte-Pugh Classification (11,12,14)

Child's Grade	A	B	C
Bilirubin (mg/dL)	<2	2–3	>3
Albumin (g/dL)	>3.5	3–3.5	<3
PT (sec > control)	1–4	4–6	>6
Coma score	0	1–2	3–4
Ascites (management)	none	easy	difficult
Nutritional status	excellent	good	poor
Operative mortality	0–10%	4–31%	19–76%

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 because of 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 d 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 to improve severe nutritional depletion by enteral or parenteral nutrition. The use of branched-chain amino acid formulations may decrease encephalopathy but does 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 3 s 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 reaccumulates 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.

References

1. Mangano DT, Goldman L. Preoperative assessment of patients with known or suspected coronary disease. N Engl J Med 1995; 333:1750–6.
2. Fleisher LA, Eagle KA. Clinical practice: lowering cardiac risk in noncardiac surgery N Engl J Med 2001;345:1677–82.
3. Lee TH, Boucher CA. Clinical practice: noninvasive tests in patients with stable coronary artery disease N Engl J Med 2001;344:1840–5.
4. Poldermans D, Fioretti PM, Forster T, et al. Dobutamine-atropine stress echocardiography for assessment of perioperative and late cardiac risk in patients undergoing major vascular surgery. Eur J Vasc Surg 1994;8:286–93.
5. Eagle KA, Berger PB, Calkins H, et al. ACC/AHA Guideline Update for Perioperative Cardiovascular Evaluation for Noncardiac Surgery–Executive Summary. A report of the American College of Cardiology/Am Heart Association Task Force on Practice Guidelines (Committee to Update the 1996 Guidelines on Perioperative Cardiovascular Evaluation for Noncardiac Surgery) Anesth Analg 2002;94:1052–64.
6. Poldermans D, Boersma E, Bax JJ, et al. The effect of bisoprolol on perioperative mortality and myocardial infarction in high-risk patients undergoing vascular surgery. N Engl J Med 1999; 341:1789–94.
7. Oliver MF, Goldman L, Julian DG, Holme I. Effect of mivazerol on perioperative cardiac complications during non-cardiac surgery in patients with coronary heart disease: the European Mivazerol Trial (EMIT). Anesthesiology 1999;91:951–61.
8. Wyser C, Stulz P, Soler M, et al. Prospective evaluation of an algorithm for the functional assessment of lung resection candidates. Am J Respir Crit Care Med 1999;159:1450–6.
9. Sladen RN. Anesthetic considerations for the patient with renal failure. Anesthesiol Clin N Am 2000;18:863–82.
10. Ter Wee P, Smit A, Rosman J, et al. Effect of intravenous infusion of low-dose dopamine on renal function in normal individuals and in patients with renal disease. Am J Nephrol 1986;6:42–6.
11. Maze M, Bass NM. Anesthesia and the hepatobiliary system. In: Anesthesia, 5th ed. Miller RD, ed. Philadelphia: Churchill Livingstone, 2000;1960–72.
12. Friedman LS. The risk of surgery in patients with liver disease. Hepatology 1999;29:1617–23.
13. Ziser A, Plevak DJ, Wiesner RH, et al. Morbidity and mortality in cirrhotic patients undergoing anesthesia and surgery. Anesthesiology 1999;90:42–53.
14. Pugh RN, Murray-Lyon IM, Dawson JL, et al. Transection of the oesophagus for bleeding oesophageal varices. Br J Surg 1973;60: 646–9.