puter application is necessary. The same problem also applies to the use of radioactive labelled compounds injected intravenously.

Even with the availability of cardiac output measurements, therapeutic application requires that the determinants of cardiac output be available. Although heart rate monitoring is not difficult, preload and afterload are not easy to measure clinically. Preload can be estimated from PA occluded pressure if a PA catheter is used. However, there is increasing evidence that the real measure of preload, end diastolic fibre length or ventricular volume, may be dissociated from pressure in a variety of circumstances. Volume measurements may be possible in future with a new type of PA catheter. Also, diastolic diameter measurement can be made from radiolabelled precordial scintillation counting and echocardiography, but cost is a problem. Afterload can be grossly estimated from arterial pressure, but for better assessment, a quantitative cardiac output is necessary so that vascular resistance may be approximated from the quotient of arterial pressure and cardiac output.

Myocardial perfusion and oxygenation

If the patient can undergo regional anaesthesia, then an obvious monitor of inadequate myocardial perfusion and oxygenation is the presence of angina pectoris. But if general anaesthesia is undertaken, currently there is severe limitation to detect imbalance between myocardial oxygen supply and demand. The time-honoured monitor is the electrocardiogram. However, an appropriate lead is necessary (usually V4, V5 or the equivalent) and even so, episodes of ischaemia may be missed or ST and T wave changes misinterpreted. Also, the regional nature of myocardial ischaemia is a problem. Some clinicians believe that monitoring pulmonary artery occlusion pressure (PA_{op}) gives advance information about impending myocardial ischaemia, either because of change in compliance of the ventricle with rising PA_{op} or decrease in global ventricular function. Several more recent approaches to non-invasive detection of myocardial ischaemia include: echocardiography, showing changes in ventricular wall motion which may be a very early sign of regional ischaemia, and the cardiokymograph, which detects similar changes in precordial wall motion through changes in the magnetic field of the chest. However, for best results with either method,

either the ultrasound transducer or the magnetic crystal must be directed at the area of ischaemia.

At present, the most practical method for assuming that there is sufficient myocardial oxygen supply for the demand is to consider blood pressure and heart rate separately. Thus, arterial pressure should be controlled within 10 to 15 per cent of the symptom-free awake state and heart rate should be kept as low as feasible without interfering with cardiac function. Considering rate pressure product in isolation makes no sense, for the implication of changes in heart rate and changes in arterial blood pressure are considerably different.

Thus, clinical monitoring of the heart depends on the physical status of the patient and the surgical procedure to be performed. In patients with minimal heart disease, monitoring of heart sounds, ECG with a precordial lead, and indirect blood pressure are sufficient. If there is to be trespass on the heart, lungs, or cross-clamping of major vessels, then direct arterial and central venous pressure monitoring should be used even in relatively healthy patients. For those with moderately severe heart disease undergoing relatively minor surgery, these two modalities should also be considered. Patients with severe heart disease (non-compensated congestive heart failure, unremitting angina, or a myocardial infarction within three months of surgery) should be monitored with everything that is available today (direct arterial and thermodilution pulmonary artery catheter monitoring).

Monitoring the kidney

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Anaesthetists use renal function tests to assess either preoperative kidney function or postoperative renal failure.

FIGURE

Preoperative Assessment

Patients with preoperative renal impairment may require modification of intraoperative care. The clearance of water soluble ionised drugs, e.g., muscle relaxants, is reduced so that their effects will be prolonged. The ability to regulate water and solute excretion is impaired so that close attention must be given to fluid therapy. When renal dysfunction is severe, the array of symptoms may include hypertension, anaemia, hyperkalaemia and acidosis. Such severe disease is associated with reduction in glomerular filtration with resultant elevation of blood urea and serum creatinine concentrations. In the steady state, serum creatinine concentration doubles for every 50 per cent reduction in glomerular filtration rate (GFR). Measurement of creatinine is a more reliable index of glomerular filtration than BUN because its production and release from muscle are constant and modified little by physical activity or protein balance; creatinine is excreted almost exclusively by glomerular filtration and its excretion is not altered by a change in urine flow.¹

Recognition of renal failure

The signs of renal failure do not appear during oliguria unless there is also a failure of solute excretion. However, if the kidney is exposed to prolonged hypovolemia, it eventually fails to produce urine of appropriate concentration. Thus it is rational to use tests of solute excretion as indices of failure. The total solute excretion of the postoperative patient² is approximately $10 \text{ mosm} \cdot \text{kg}^{-1} \cdot \text{day}^{-1}$. Unfortunately, during surgery both urine volume and concentration are reduced so that urine production during surgery is a poor predictor of postoperative renal performance.

After surgery, renal insufficiency may be recognised easily by the relationship between urine volume and concentration (Figure). Other tests such as the fractional excretion of sodium (FE_{Na}) and free water clearance also depend upon urine concentrating ability. Unfortunately, the FE_{Na} is misleading after the use of natriuretic diuretics. Measurements based upon solute excretion, such as osmometry, are easy and quick to perform and are

related to the symptomatology of renal failure. The values are relevant because any prophylactic effect of furosemide or mannitol, at least in experimental ischaemic renal failure, is a result of improved solute excretion.

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Monitoring the liver

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Assessment and monitoring of liver function begins with a case history and physical examination of the patient.¹ Liver function tests are of limited value unless performed on a regular basis and, in general, are best suited for assessment of chronic liver disease rather than acute episodes. In addition, attention should also be paid to blood gas values and the patient's cardiovascular status. Renal function should also be assessed as there are often concomitant renal problems in patients with severe liver disease. The site of biliary obstruction may be determined by various imaging techniques such as ultrasound, liver scanning and percutaneous hepatic cholangiography.

Screening for viral hepatitis must always be considered because of the risk of infecting operating room personnel. This disease may be caused by a variety of viruses but Viral B and Non A/Non B hepatitis are the most relevant. The presence of these may be inferred from the history, which should include country of origin, drug therapy, exposure to injections and blood transfusions, surgery, alcohol intake, and sexual habits. Serological testing¹ for antigenic and antibody markers

of viral hepatitis can now establish potentially infectious patients.

Cirrhosis is the pathological finding in chronic liver disease; in this condition there is obstruction to blood flow through the hepatic parenchyma. The three common causes are: alcohol abuse, immune disease and hepatitis B infection. Cirrhosis may lead to the development of oesophageal varices and gastrointestinal bleeding as well as secondary renal, cardiac and respiratory failure.

In the presence of chronic liver disease the synthetic storage and excretory functions of the liver may be disturbed and these can be assessed, partly, by standard liver function tests. Jaundice alone may merely indicate some abnormality of the biliary tree and may be due to: increased bile pigment production, defective uptake and transport, defective conjugation or defective excretion of bilirubin. One or more of these factors may be present at the same time. The critical test in the jaundiced patient is the return to normal of the prothrombin time by the administration of intramuscular Vitamin K, in a dose of 10–20 mg six hourly for 24 hours. Patients whose bilirubin is grossly raised (in excess of $150 \mu\text{mol}\cdot\text{L}^{-1}$) are at risk of developing postoperative renal failure and measures should be taken to promote a diuresis pre-, per- and postoperatively in such individuals. This can be accomplished by adequate fluid loading and use of diuretics as necessary.

Patients' hepatic reserve may be estimated by assessment of bilirubin, albumin, the presence of ascites, neurological disorder and assessment of the nutritional state² or alternatively the assessment may be made using bilirubin, albumin, prothrombin time and encephalopathy and the patients graded accordingly.³ By these means the potential surgical risk may be estimated and the outcome following major surgery determined.

Preoperative monitoring requires attention to changes in the cardio-respiratory system, assessment of renal function, perhaps by using urine output, and if necessary, tests of haemostasis to determine that the liver's output of clotting factors is adequate. In the postoperative period, unexplained nausea, jaundice or complaints of feeling "unwell" should focus attention on the possibility that the patient is developing hepatitis. This may be confirmed by abnormal changes in liver function tests unrelated to surgical trauma or pre-existent liver disease.