Preoperative Assessment of the Patient with Liver Dysfunction

"The preoperative assessment of liver function is important, but often neglected. Liver dysfunction may be accompanied by renal dysfunction and impairment of the blood clotting mechanism. Certain liver problems may be amenable to treatment before operation and others may require steps to be taken during anaesthesia...

"It can be seen, therefore, that a number of factors are relevant in the assessment of liver function before operation. The first steps are a careful history and examination of the patient. Although many liver function tests are available, it should be remembered that these are rarely specific in their own right, and the tests are often somewhat crude and inaccurate. Abnormalities are only seen when considerable liver damage has already occurred. ...

"Five categories of liver dysfunction are: previous history of jaundice; jaundice as the presenting symptom; chronic liver disease; viral hepatitis; acute liver failure. These should not be considered as water-tight compartments, indeed there is obvious overlap, but they form a useful guide for the anaesthetist when determining what steps to take for patients with liver dysfunction in relation to their presenting symptoms. ...

"The patient gives a history of jaundice, usually many years previously, but is now well and has apparently normal liver function. The most likely diagnosis is viral hepatitis. If liver function tests such as serum bilirubin, proteins, aspartate aminotransferase (AST), alkaline phosphatase (ALP) and prothrombin activity are within normal limits and the surface antigen for hepatitis B virus (HBsAg) and e-antigen are absent, then it is unlikely that any problem will be encountered during anaesthesia and surgery in such a patient. If antibodies to HBsAg are detected, this would confirm that the patient has had a previous attack of viral B hepatitis and is now immune. ...

"If abnormalities in liver function tests are detected, such as increased AST, bilirubin and globulin concentrations, with reduced serum albumin concentration, and particularly if HBsAg or e-antigen is detected, this might imply that the patient has developed chronic liver disease as a result of a previous infection with hepatitis B virus. In the patient with a history of previous jaundice, specific questions should be asked concerning anaesthesia and surgery. ...

"Jaundice is a clinical observation characterized by yellow pigmentation, seen first in the sclera and then in the skin and other tissues, and occurs when serum bilirubin exceeds 20 μmol litre⁻¹. Such an increase may be associated with disorders of bilirubin metabolism or may be evidence of cellular liver disease. Many causes of jaundice are not
amenable to surgical treatment. On the other hand, unnecessary delay in the patient with obstructive jaundice, amenable to surgical treatment, may lead to cholangitis and renal failure.

The causes of jaundice may be classified according to mechanism: increased bile pigment production, defective uptake and transport within the hepatocyte, defective conjugation, or defective excretion. Defective excretion of bilirubin, that is obstructive jaundice, may occur either within the bile canaliculi and small ducts within the liver, that is intrahepatic cholestasis. Extra-hapatic obstruction may result from gall-stones, strictures, carcinoma of the biliary tree or carcinoma of the head of the pancreas. The differentiation of intrahepatic cholestasis from extrahepatic obstruction is obviously crucial in determining whether surgery is indicated.

The differential diagnosis of medical or surgical jaundice is not always straightforward. However, a careful history and examination of the patient, with the results of various liver function tests and diagnostic aids, may be helpful. Needle biopsy of the liver is one of the main diagnostic methods of determining the causes of jaundice. A needle biopsy is of necessity a blind procedure and, as an alternative, needle biopsy via a laparoscope enables samples to be taken from specific areas of the liver; general anesthesia may be required.

The usual method of visualizing the biliary tree is by an oral or i.v. cholangiogram. Percutaneous cholangiography is usually carried out without a general anaesthetic, often under diazepam sedation, but as a precaution a large-bore i.v. infusion should be set up before the procedure so that, in the event of cardiovascular collapse, the circulating blood volume may be rapidly restored by transfusion.

A prolonged prothrombin time is commonly found in the jaundiced patient. Prothrombin is synthesized by the liver but requires the presence of vitamin K. In obstructive jaundice vitamin K is not absorbed in the intestine as bile salts are absent. All jaundiced patients should receive a course of parenteral vitamin K therapy before anaesthesia and surgery. If prothrombin activity does not return to normal, it is indicative of hepatocellular damage, and fresh frozen plasma infusion may be helpful in reducing the risk of severe haemorrhage during surgery or diagnostic procedures.

It has been known for some time that there is an increased incidence of renal failure following anaesthesia and surgery in patients with obstructive jaundice. Such renal failure has been attributed to anoxia associated with the normal decrease in renal blood flow during anaesthesia and surgery, and the possible toxic effects of bilirubin.

Renal failure following surgery could be prevented in the jaundiced patient by inducing a diuresis before operation, and maintaining it during operation and in the period immediately after operation with mannitol i.v. Mannitol 5% or 10% is all that is required.

Chronic liver disease, hepatocellular disease or cirrhosis are often used interchangeably to describe the same thing. Compensated chronic liver disease is compatible with a complete feeling of well being, and when symptoms do occur they are usually vague, such as malaise, dyspepsia, weight loss, loss of libido and menstrual disturbances. One of the most useful signs of cirrhosis is the presence of spider naevi on the skin of the face, arms and upper torso. The size of the liver is very variable, ranging from shrunken to very large, and may change during the course of the disease.

A constant feature of cirrhosis is increased resistance to flow of blood in the portal venous system, resulting eventually in congestive splenomegaly and collateral venous channels — portasystemic shunts. Three major compli-
cations may occur in the cirrhotic patient — encephalopathy, ascites and gastrointestinal haemorrhage. Other problems include infection, jaundice and vitamin deficiency.

"Patients with chronic liver disease may present for anaesthesia and surgery either for procedures unrelated to their liver dysfunction or as a result of complications such as gastrointestinal bleeding. The neurological response of the patient to analgesic or sedative drugs used for premedication may be a useful guide to the use of such drugs in the period after operation. All general, regional and spinal anaesthetic techniques will decrease splanchnic blood flow.

"Good oxygenation, avoidance of hypotension and the maintenance of arterial carbon dioxide tension within the normal range are sensible precautions. Volatile anaesthetic agents which directly affect the splanchnic circulation, such as cyclopropane, diethyl ether and methoxyflurane, are best avoided. Halothane is not contraindicated, but the dose should be adjusted in accordance with the patient's cardiovascular system.

"Viral hepatitis may cause liver dysfunction ranging from a mild systemic illness, with or without jaundice, to acute liver failure. Although it is accepted that a number of different viruses can cause hepatitis, most cases are related to either hepatitis A or hepatitis B virus infection. Hepatitis B is of most concern, since infection with this virus may be followed by chronic liver disease.

"It should be recognized that patients with liver disease associated with hepatitis B virus are not usually as potentially infective as the patient with renal disease who acquires hepatitis B infection. When anaesthetizing the HbsAg-positive patient it is essential that a routine is followed. The number of personnel in the operating theatre should be kept to a minimum and all should wear disposable caps, masks, gowns, gloves and overshoes. For the anaesthetist the wearing of gloves (some prefer two pairs) is mandatory. unnecessary injections and blood sampling should be avoided. Clearly marked rubbish bags should be available so that every disposable item that comes into contact with the patient may be incinerated. Non-disposable anaesthetic items may be sterilized.

"The clinical manifestations of acute liver failure are many and, as well as those directly related to hepatic damage, include neurological, acid-base, cardiac, renal, metabolic and haematological disturbances. The most outstanding feature is the development of coma. Although acute liver failure is a rare event, it is a diagnosis which the anaesthetist should consider when encountering a comatose patient.

"The characteristic features of hepatic coma include hyperventilation, fluctuating level of consciousness and haemorrhage, usually from the gastrointestinal tract. The more obvious signs of liver disease may not necessarily be immediately apparent. Since one of the commonest causes of fulminant hepatic failure is virus infection with either hepatitis A or B, precautions may be necessary.

Cardiovascular Effects of Droperidol During Enflurane and Enflurane-Nitrous Oxide Anaesthesia in Man


"Droperidol is not infrequently used as an amnesic supplement and an 'afterload' reducer during anaesthesia in critically ill patients because of its reported benignity on myocardial dynamics. In this study we measured the cardiovascular effects of droperidol 5 mg during light steady state enflurane-oxygen and enflurane-nitrous oxide-oxygen anaesthesia in 20 patients undergoing cardiac or major vascular surgery.

"None of the patients were receiv-
ing beta-adrenergic receptor blocking drugs, but four were taking digitalis preparations. Premedication included morphine . . . diazepam . . . and atropine . . . intramuscularly, 90 minutes before the scheduled operation. . . . A central venous pressure catheter was placed percutaneously into the right atrium from the ante-cubital fossa or neck and a radial or brachial artery catheter was inserted percutaneously and threaded 30 to 72 cm into the central aorta. The aortic pressure catheter was attached through an arterial pressure transducer to a central digital computer substation in the operating room.

"All patients had anaesthesia induced with sodium thiopentone, were paralyzed with succinylcholine, had their tracheas intubated and were mechanically ventilated with a volume limited respirator. . . . Anaesthesia was maintained with enflurane one to two per cent and oxygen or enflurane 0.5 to 1.5 per cent, nitrous oxide 60 per cent and oxygen. . . . A semiclosed circle system provided carbon dioxide absorption and a total fresh gas inflow of 5-6 1/min. Muscle relaxation was maintained with pancuronium . . . intravenously initially and subsequent doses . . . as necessary.

"In ten patients receiving enflurane-nitrous oxide-oxygen anaesthesia, droperidol produced significant (P<0.025) decreases in SVR and BP and increases (P<0.05) in HR and QT which were maximal after five minutes, somewhat less 5 minutes later and essentially back to control values 15 minutes following administration. . . . During enflurane-oxygen anaesthesia droperidol reduced SVR BP after five and ten minutes (P<0.05) but did not significantly alter any other variable measured. . . . All variables were back to control levels 15 minutes after droperidol during enflurane-oxygen anaesthesia. . . .

"Emphasis on preserving myocardial dynamics in modern anaesthesia, especially in patients with cardiac disease, has supported the concept of 'light' anaesthesia. One complication of this approach has been hypertension during operation. . . . As a result, the use of vasodilators, ganglionic blockers or other compounds that reduce or preserve BP, SVR and left ventricular 'after-load' has become popular, especially in patients with coronary artery disease.

"The results of this study demonstrate that small amounts of intravenous droperidol produce a significant, though relatively transient, reduction in BP and SVR during enflurane-nitrous oxide-oxygen and enflurane-oxygen anaesthesia which is associated with an increase or no change in QT. Similar changes have also been found when droperidol is employed during halothane ... or morphine anaesthesia.

"Although large doses of droperidol (1 mg/kg) have been shown to impair left ventricular contractility in right-heart bypass preparations in the dog, smaller doses (0.5 mg/kg) do not influence myocardial dynamics.

"The cardiovascular effects of intravenous droperidol 5 mg were measured in 20 patients during steady state enflurane-nitrous oxide-oxygen or enflurane-oxygen anaesthesia. . . . Our findings suggest that droperidol causes minimal or no myocardial depression when used during potent inhalation anaesthesia and may have a place as an amnesic supplement and/or 'afterload' reducer during light enflurane anaesthesia."

Perioperative Management of the Pacemaker Patient


"Temporary and permanent cardiac pacing have evolved from sophisticated experimental procedures performed in only a few medical centers to the point where they are now performed on a routine basis in many hospitals."
The ease of insertion of temporary electrodes has made it feasible to manage successfully otherwise fatal arrhythmias until a permanent pacing system can be implanted. The availability of transvenous permanent pacemaker catheter electrodes has made it possible for even the infirm or clinically ill patient to benefit from permanent pacing.

"It is the purpose of this report to familiarize the reader with some of the newer concepts in pacing as they relate to preoperative assessment of the patient with conduction disturbances, and the anticipation of potential perioperative problems of the patient who has a permanent pacemaker and their management. . . .

"The . . . major concerns whenever a patient with a conduction disturbance or bradycardia syndrome comes to the operating room can be divided into three areas: (1) Does the patient who has an atioventricular or intraventricular conduction disturbance require temporary pacing during the operative procedure or during induction of anesthesia? (2) What special preoperative assessment is necessary for the patient who has a permanent pacemaker? (3) What intraoperative evaluation needs to be made and what special precautions are necessary to avoid the risks of electromagnetic interference with pacemaker function in the operating and recovery rooms? . . .

"In recent years the natural history of intraventricular conduction defects has been clarified. Long-term studies have indicated an increased risk of development of complete atioventricular block in patients who have bifascicular block. However, the risk appears to be quite low: complete atioventricular block occurs in only 5 to 8 per cent of such patients per year. . . . There are situations, however, in management of the patient who has bifascicular block where prophylactic pacing prior to induction of anesthesia may be indicated.

"Except under unusual circumstances a temporary lead is indicated prior to induction of anesthesia in any patient receiving an epicardial pacemaker electrode system. Pharmacologic therapy probably has no place whenever temporary pacing is feasible, but may be necessary as an emergency measure until a temporary electrode can be inserted. Care must be taken during transporting the patient who has a temporary pacemaker to the operating room so that the electrode does not become dislodged or perforate the right ventricle. . . .

"Initially, permanent pacing was utilized only for the patient with complete atioventricular block complicated by Stokes-Adams episodes or congestive failure. Subsequently, the indications for permanent pacing were extended to patients who had other arrhythmias as well. For example, patients who have symptomatic sinus bradycardia or periods of sinus node arrest, accompanied by dizziness, syncope or congestive heart failure. . . . Patients who have sinus node dysfunction accompanied by paroxysmal supraventricular tachycardia, atrial flutter, or atrial fibrillation (the 'bradycardia-tachycardia syndrome') are treated with a combination of antiarrhythmic agents for the supraventricular arrhythmia and a permanent pacemaker for the bradycardia. . . . Permanent pacing has also been utilized to suppress ventricular tachycardia and to convert supraventricular arrhythmias resistant to antiarrhythmic agents. . . .

"It needs to be emphasized that the patient who has a permanent pacemaker has significant underlying cardiovascular disease. . . . During stress or sympathetic stimulation, such as during an operative procedure, return to normal sinus rhythm may occur in patients previously in complete heart block: ventricular arrhythmias are also common in patients with pacemakers. It is important that the patient who has a pacemaker be evaluated for any progression of symptoms of the underlying heart disease . . . as well as for electrolyte disorders and adequacy of digital-
ization, prior to any planned operative procedure. Likewise, it is important to evaluate the pacemaker itself for its ability to pace the heart properly. . . .

“It is also important to establish the etiology of the heart disease responsible for the arrhythmia being treated with the pacemaker, . . . . Finally, it is important to establish whether electrocautery will be used during the operative procedure. Since 90 per cent or more of currently implanted pacemakers are of the ‘demand’ (synchronous) variety, they are sensitive to direct or indirect electromagnetic interference (EMI) . . . . Although there is some variation in opinion as to the risk to the patient who has a pacemaker from electromagnetic interference, deaths have been reported and caution should be exercised.

“‘To decrease the possibility of adverse effects of electrocautery on the pacemaker patient, the following intraoperative measures have been employed.

1. The indifferent plate of the electrocautery unit should be placed as far away from the pulse generator and lead as possible.

2. The patient should be electrocardiographically monitored during the operation. . . .

3. If the patient is being paced at the time of the procedure, palpation of the pulse or esophageal stethoscopic cardiac monitoring during the electrocauterization is necessary. . . .

4. When the electromagnetic field intensity is high . . . . the frequency and duration of electrocautery should be limited to one-second bursts every 10 seconds to prevent repetitive asystolic periods.

5. When the pulse generator is located close to the operative field, it may be necessary to bring a sterilized magnet into the field. . . .

“Some pacemakers are sensitive to field intensities as low as 1 volt per meter, and since the field intensity from electrocautery may be as high as 60 volts per meter, caution with all sources of electromagnetic interference is indicated. . . . Since pacemakers may vary in sensitivity to external electromagnetic interference, the maneuvers described . . . . should be utilized with each pacemaker patient for whom electrocautery is used. . . .

“Inasmuch as the pacemaker electrode is in direct contact with the endocardium, a direct electrical pathway to the heart is present in the paced patient. This is most likely to be a hazard . . . . when the electrical contact points between pacemaker lead and external generator are not properly shielded. . . . Since patients who have permanent pacemakers have underlying cardiac disease, recognition and treatment of significant arrhythmias in the perioperative period are essential. Hypoxemia should be corrected, and antiarrhythmic agents utilized when necessary. Continuous monitoring is indicated even after the procedure, and until vital signs have stabilized. . . .

“Although permanent pacemakers have protective circuits to guard against externally applied high-voltage discharge. . . . pulse generator malfunction has been reported to occur following external defibrillation or cardioversion. In elective cardioversion the lowest voltage necessary should be utilized. Ventricular fibrillation in the patient who has a permanent pacemaker should be managed with techniques similar to those used for any other patient, with the exception that the defibrillator paddles should not be placed directly over the permanent pulse generator.”

Errata (December, 1978 issue)

p. 649
2nd paragraph, line 3 should read: reference to race or eye color

p. 681, under H
include the following:

Hannigan, John: Anesthetic Management of Conray® Toxicity, 627
Hubbert, Charles H.: Anesthetic Management of Conray® Toxicity, 627