The Patient with Heart Disease: Preoperative Evaluation and Preparation

"The surgical patient with heart disease and diminished cardiac compensation has decreased cardiac output and decreased tissue perfusion. We are concerned about this patient for several reasons. Because of his increased intravascular volume, the management of his fluid and electrolyte replacement becomes very complicated. He is a candidate for thromboembolic phenomena. . . . With decreased ventilation, there is increased likelihood for hypoxia and hypercarbia as well as respiratory infections and pneumonia. He has impaired liver and kidney function, which can change the effects of drugs. In addition, the various stresses to which a surgical patient is exposed can push him into acute heart failure. . . . He may be inadvertently given too much fluid for his cardiac status.

"This patient may also get into trouble in the postoperative period. . . . It is not the patient with overt failure whose cardiac status will be missed and who will come to surgery without some form of therapy; it is easy to recognize the patient with severe dyspnea, edema, enlarged heart, or a loud murmur. It is the patient with borderline incipient failure who was not recognized immediately as being in failure who may go on to have stress-precipitated severe failure.

"The typical patient who gets into trouble is one of the following:

(1) The patient with rheumatic heart disease with mitral stenosis.

(2) The patient with aortic stenosis, particularly an elderly patient who may have a very impressive murmur. . . .

(3) The patient with chronic lung disease. . . .

"With these patients, it is important to keep in mind the concept of cardiac reserve. . . . Many new techniques have been developed for assessment of myocardial function. . . . In the clinical situation, . . . we must still employ simple bedside tools, primarily the history and the physical examination. Concerning the history, a major warning symptom in the patient with incipient failure is cough, particularly nocturnal cough. . . .

"Insomnia is another insidious symptom of impending left heart failure. . . . Unexplained fatigue is another manifestation of incipient heart failure. . . . Abdominal discomfort, indigestion, or pain, particularly right upper quadrant pain, suggests distention of the liver capsule. One of the compensatory mechanisms in failure is increased sympathetic activity. This results in increased perspiration. . . . The patient may describe dyspnea on effort, but you have to distinguish this from hyperventilation. . . . The rapidity of onset of dyspnea is a valuable clue. . . .

"Looking further into the history, has the patient been receiving any medication that might impair cardiac function? . . . One of the subtle findings of heart failure is persistent unexplained sinus tachycardia. . . . If this cannot be related to something like fear, apprehension, or fever, think of borderline fail-
Some patients with incipient left heart failure have a potent element of bronchoconstriction that may present as asthma or wheezing, when in fact it reflects pulmonary vascular distention due to the cardiac condition.

"Pulsus alternans, observed when feeling the arterial pulse, also detects impaired left ventricular function. . . . If the patient has a slight murmur across the outflow of the left ventricle, as in an elderly patient, for example, with some atherosclerotic change in the aorta or the aortic valve, pulsus alternans translates itself into murmur alterans. . . . The neck veins should be examined. The external jugular vein, which is often tortuous and partly compressed, may be distended even in the absence of true elevation of venous pressure . . .

"Gallops are diastolic events related to 2 periods of filling of the ventricles. . . . The ventricular diastolic gallop (S₃) is frequently one of the first detectable signs of serious heart disease and/or cardiac decompensation. . . . The ECG should of course be examined. . . . It can . . . alert you to the possibility of infarction, ischemic disease, or rhythm disturbance. . . . Characteristic of a patient in congestive heart failure is the so-called butterfly pattern. . . . This is not a subtle sign of failure, as the patient will be in pulmonary edema.

"A sign seen very early in the course of failure is the Kerley-B line, a linear density caused by thickening of the intralobular septa due to an increase in pulmonary venous pressure. . . .

"If you have the time, it is better to undertake digitalization and diuretic therapy deliberately over a period of 2 to 3 days, rather than to have to move in with acute digitalization. But if the patient requires early and immediate operation, you can digitalize rapidly, using IV digoxin. Given IV, this drug has a rapid onset of action, reaching its peak effect in about 2 to 3 hours and has a strong effect even before that. Since you can continue to use the same drug thereafter, it probably represents the agent of choice even in the circumstances of fairly rapid digitalization.

"Diuretics should also be given. However, these may cause a loss of potassium, which represents a hazard through digitalis intoxication, with consequent arrhythmias. For this reason, it is advisable to monitor the serum K⁺ levels carefully and replace K⁺ as needed. Another problem associated with the use of diuretics is excessive reduction of effective circulating blood volume . . . The same principle applies to digitalis. It is wise to underdigitalize rather than overdigitalize. . . . Many subtle signs and symptoms of diminished cardiac compensation may be present before the onset of decompensation. These should be looked for and the appropriate therapy instituted before overt heart failure occurs."

Physostigmine as an Adjuvant to Neuroleptanaesthesia in Neurosurgical Procedures


"Neurosurgical operations which require the patient to cooperate with the surgeon may be done with local analgesia alone. However, this may be unpleasant for the patient and because of this and other factors such as the duration of the procedure, age, abnormal movement or severe pain, some form of sedation is often necessary. . . . For a good surgical result, the site of the proposed lesion must be verified. For this the patient has to be alert enough to answer questions about sensory phenomena and to allow motor function to be tested. Ideal neuroleptanaesthesia would provide these conditions while suppressing physical and mental discomfort. This ideal is not always achieved. . . .

"The anticholinesterase inhibitor physostigmine can be a useful aid in neuroleptanaesthesia. A patient may be deeply sedated and, when cooperation is required, can be awakened with intravenous physostigmine. Physostigmine is
a tertiary amine and, unlike neostigmine, it is unionised at body pH and so will cross the blood brain barrier. It reverses the central effects of a number of drugs including droperidol, diazepam, the phenothiazine derivatives and the belladonna alkaloids. The use of physostigmine is illustrated in the management of two children with dystonia muscularorum deformans.

“At Toronto General Hospital stereotactic thalamotomies are done in two stages. The first stage is done with a conventional general anaesthetic. It involves making a burr hole, screwing the stereotactic apparatus to the skull and making initial measurements of the thalamus with a ventriculogram. The second stage a few days later requires patient cooperation. The stereotactic frame is reattached to the skull. The burr hole is re-opened and the site of the lesion is found and verified by electrostimulation of the thalamus.

“The use of physostigmine selectively to reverse the effects of droperidol and diazepam has permitted an optimum level of neuroleptanaesthesia in neurosurgical operations where cooperation of the patient is required during part of the procedure. The patient can be put to sleep or readily awakened to be fully cooperative depending on the needs of the surgeon. In this series of seven anaesthetics there were no side effects from the small doses of physostigmine employed. Bradycardia and salivation were not a problem.

“Atropine was not necessary. Since a narcotic antagonist is not needed, a reasonable degree of analgesia can be maintained in these patients while they are awake. The latent time for the effect of physostigmine was two to four minutes and the effect of an intravenous dose lasted from 35 to 45 minutes. With physostigmine, these patients wake up gently as though from normal sleep. If neurological assessment is required post-operatively, drowsiness due to drugs can be reversed by giving more physostigmine and the level of consciousness can then be assessed.”

**Anticholinergic Premedication**


“One of the traditional reasons for the preanaesthetic administration of anticholinergic agents has been to reduce oropharyngeal and tracheobronchial secretions. The practice of administering anticholinergics before general anaesthesia has persisted in spite of the non-irritant nature of modern anaesthetics.

“The present authors determined to ask a group of anaesthetists in a large teaching hospital what differences they observed (as far as mouth and respiratory tract secretions were concerned) between patients who received anticholinergic premedication and those who did not.

“The following questions were asked:

1. If anticholinergic premedication was omitted, did secretions constitute a problem during or after the anaesthetic?
2. If there was a problem, what was its nature?
3. Was an anticholinergic then administered? If so, with what result?

“Eleven anaesthetists participated in the study for one year. When anticholinergic drugs were used, either atropine or scopolamine was administered approximately one hour before anaesthesia. The fundamental question of what constitutes ‘troublesome secretions,’ defies definition. It is apparent that this is a subjective evaluation on the part of the individual anaesthetist. The present investigators decided to consider that secretions constituted a definite problem if the anaesthetist was constrained to administer an anticholinergic drug for their control. Anaesthetists were asked to describe any problems which they encountered whether or not they administered an anticholinergic agent before or during anaesthesia.

“Excessive mouth or respiratory tract secretions might present the anaesthetist with problems in the conduct of
The only truly 'troublesome' secretions, however, are those which present a threat to the patient's well-being or hinder the efficient conduct of the anaesthetic.

"It is a significant finding that in only 2 per cent of 244 anaesthetics where there had been no anticholinergic premedication were secretions considered to be such a problem that the anaesthetist was constrained to administer atropine. The fact that no additional anticholinergic was administered to those atropinized patients who nevertheless demonstrated 'problem secretions' possibly reflects the stoicism with which anaesthetists accept such troublesome secretions in the patient who has received anticholinergic premedication.

"In patients who have not received anticholinergic premedication secretions are not viscid and are more easily removed by suction than is the case after administration of anticholinergic. . . . Two hundred and forty-four surgical patients who received no anticholinergic premedication were compared with 160 patients who had received atropine or scopolamine before the induction of anaesthesia. Infants and patients undergoing heart surgery were excluded. Eleven anaesthetists participated in the study. They were asked to report problems with oropharyngeal and tracheobronchial secretions. Two per cent of unpremedicated patients experienced problems with secretions of a degree sufficient to require treatment. This small percentage appears insufficient to warrant routine preoperative anticholinergic medication."

A Comparison of Some Cardiorespiratory Effects of Althesin and Ketamine When Used for Induction of Anaesthesia in Patients with Cardiac Disease


"Induction of anaesthesia in patients with heart disease is a hazardous procedure because the impaired circulatory system is less tolerant of depression. . . . Recently, ketamine has been recommended for these patients because it is associated with cardiovascular stability. . . . However, marked cardiovascular stimulant effects by ketamine have been reported by others in fit patients. . . . In this study some cardiorespiratory effects of ketamine and Althesin have been measured in premedicated patients with heart disease. . . . Patients with heart disease awaiting major cardiac surgery were studied. Most of them had long-standing disease with damage to one or more heart valves. . . ."

"E.c.g. and arterial pressure were measured continuously, displayed on an oscilloscope and recorded. . . . Heart rate was counted manually from either trace. . . . The transducers were calibrated against a column of mercury. The zero point was taken as the mid-axillary line. Arterial pressure was measured using an indwelling femoral artery catheter. . . . Pulmonary artery pressure was measured. . . . The signals were amplified and recorded as two separate traces (phasic and mean). . . . Only mean values of the pulmonary artery pressure and wedge pressure were used in view of the low frequency response of this system. . . ."

"Cardiac output was measured by thermal dilution. . . . Only limited respiratory measurements were possible because the use of a close-fitting mask tends to distress patients and interfere with resting control measurements. . . . Arterial pH, Po2 and Pco2 were measured in all the patients, control samples being taken shortly before induction. . . . Arterial pressure, central venous pressure and, wherever applicable, wedge pressure were estimated every 12 s from trace. . . . Althesin 0.05 ml/kg was administered over 60 s into a previously cannulated vein. . . . Ketamine 2.0 mg/kg was administered over 60 s. . . ."

"Ketamine caused a marked and sustained increase, in excess of 40%, in arterial pressure. The average systolic
pressure increased to more than 200 mmHg with no evidence of a return to the pre-induction value when the second dose was given. However, after the second dose the mean pressure decreased significantly by a maximum of 11%. Althesin reduced the mean arterial pressure by 19%, this decrease being maintained until the second dose was given, whereupon there was a further small decrease of 9%.

"Ketamine increased the pulmonary artery pressure by a maximum of 59%, the increase coinciding with a change in systemic pressure. This was sustained until the second dose and then there was a small reduction (11%). Althesin caused little change in mean pulmonary artery pressure after the initial dose (−7%). A 12% reduction occurred after the second dose. Ketamine increased heart rate by 39%. This was sustained until the second dose when there was a decrease of 15%. Althesin failed to alter heart rate significantly after either the first or the second dose. No change in cardiac rhythm occurred after induction of anaesthesia with either agent.

"Ketamine increased central venous pressure by 55% after the induction dose and then reduced it by 7% after the second dose. Following Althesin there was a maximum decrease of 11% after the first dose and 6% after the second dose. Following ketamine pulmonary wedge pressure changes were similar to the changes in central venous pressure. After the first dose of Althesin wedge pressure decreased by 24%. Little change occurred after the second dose. Cardiac index increased to a maximum of 14%, whereas the stroke index decreased by 17% after the first dose of ketamine. The second dose of ketamine caused a decrease in cardiac index (13%), probably as a result of the decrease in heart rate; stroke index was little changed. Althesin decreased both cardiac index and stroke index by 9% after the first dose, and there was a further decrease in cardiac index of 5% after the second dose.

"Left and right ventricular work index was increased markedly after induction with ketamine. In contrast, there was a decrease after the second dose reflecting the reduction in arterial pressure and cardiac output at that time. Ketamine increased systemic vascular resistance by 33% after the initial dose, and by 11% after the second dose. Althesin reduced systemic vascular resistance after both doses. Pulmonary vascular resistance was increased after ketamine by a small amount and continued to increase after the second dose.

"Althesin increased pulmonary vascular resistance after the induction dose, but there was little further change after the second dose. Following the induction dose, PaCO₂ increased by comparable amounts; an increase of 0.9 kPa after ketamine and 1.2 kPa after Althesin. However, PaCO₂ continued to increase following the second dose of Althesin by a further 0.6 kPa, whereas there was little change after the second dose of ketamine. Mean Pao₂ did not change significantly after either drug. With three major exceptions, the effects of Althesin and ketamine in this study were very similar to those in a previous study of fit, unpremedicated patients.

"First, Althesin did not cause tachycardia. The second major exception was that ketamine increased cardiac index by only 14% despite a large increase in heart rate and central venous pressure. The third exception was that PaCO₂ increased considerably probably as a result of the combined effect of premedication and the induction agent. This increase could have contributed to some of the cardiovascular changes. Both these agents have marked effects on the cardiovascular system. Clearly, neither agent is ideal alone. However, modern techniques require the use of combinations of drugs and these can be selected to counteract the disadvantageous effects, each of the other, and thus achieve cardiovascular stability."