The anesthetic management of the patient with cardiac disease requires special consideration. The author provides a concentrated discussion of the following factors involved in these cases: risk, cardiac output and what influences it, premedication, monitoring devices, actual induction of anesthesia, choice and maintenance of anesthesia, arrhythmia management, and the postoperative period. Examples are cited from the author's own experience at St. Elizabeth Hospital in Youngstown, Ohio.

"Anesthesia is a controlled depression of the various organs of the body. All anesthetics depress the myocardium." How many times have we heard these statements throughout our training period and our postgraduate conferences? Is it then any wonder that we look a little more closely at the cardiac patient who presents himself for surgery and are a little more vigilant during the course of the anesthetic.

In using the term "cardiac disease," I refer to a broad spectrum of disease entities that affect the heart. Coronary artery disease, rheumatic heart disease with valvular dysfunction, congenital heart disease, and hypertensive heart disease, are all examples of the types of conditions that are included in the category of "cardiac disease."

The successful management of anesthesia for these patients depends upon our knowledge of the disease state, the patho-physiology of the disease process, an estimation of the myocardial reserve, our ability to alter correctible situations in the patient preoperatively, and our ability to select an anesthetic technique and agent that will not adversely affect an already stressed myocardium.

The level of medical expertise that the anesthetist has today gives the cardiac patient a good chance to undergo a successful anesthetic and surgical procedure. Gone are the days that we could refuse to anesthetize a patient because he had a cardiac problem. We have seen that patients who are in severe risk categories, because of their cardiac status, can successfully undergo anesthesia and major surgery.

I refer here to the patients with serious cardiac problems who undergo cardiac surgery, but the cardiac patient scheduled for noncardiac surgery can have the same good chance if we approach him intelligently, monitor him correctly, and are vigilant and concerned with his welfare.

Risk

Retrospective studies have shown that patients with heart disease have increased operative morbidity and mortality. Yet, there are no specific data relating heart disease and anesthetic mortality per se. All of the studies in the literature do not separate the effects of anesthesia from those of operation. Still, in certain situations, we can pre-
dict with a high index of accuracy which patient with heart disease may die or have a complicated postoperative course. In general, we can make the statement, "The sicker the patient, the greater the risk."

There has not been a great deal written about the patient with both coronary artery disease and a history of a recent myocardial infarction and his risk of a recurring infarction and the mortality associated with anesthesia and surgery. In literature reviews, it is shown that about 2% of general surgical patients have a history of myocardial infarction prior to the surgical procedure.1

In several articles, we find some sort of answer to the question, "If a patient has had a myocardial infarction and is scheduled for non-cardiac surgery, what are his chances of developing another infarct and what is the mortality associated with this recurring myocardial infarction?"

In Dr. Tarhan's study,1 all males and females over the age of 30 who received only general anesthesia were included. The results were, that if the surgery is performed within 3 months of myocardial infarction (MI), the risk of reinfarction is 37%; from 3-6 months after the MI, the risk is 16%; and if the MI is older than 6 months, the risk is 5%.

For all patients who develop recurrent postoperative myocardial infarction, the mortality is 50%.1 So, the figures add up to this situation: If there are four patients presenting themselves for surgery who have had myocardial infarctions within three months of the operation, two of these patients will have a recurrent postoperative myocardial infarction and one of these two patients will die, giving an average mortality of 25%.

Dr. Tarhan's study also implicated the type of operation. He found that vascular procedures, thoracic procedures, and upper abdominal surgery increased the risk factor by three times over all other types of surgery. Specific cardiac factors which tend to seriously increase the surgical mortality are the functional severity of the existing cardiac disease, intractable congestive failure, severe progressive angina, and the recent myocardial infarction spoken of previously. Other factors leading to the risk are hypertension, arrhythmias, coexistent pulmonary disease, extremes of age, and impaired physical status from other systemic diseases.

Another factor leading to increased risk and mortality is the situation where the cardiac patient requires emergency surgery. In the elective case, some of the more common problems can be brought under control. The patient with impending or frank congestive failure and the patients who exhibit arrhythmias can have the surgery postponed until the specific preoperative problems are corrected. The emergency situation is less favorable in that it is complicated by both the illness, which requires the surgical intervention, and the likelihood of poorly compensated or inadequately treated heart disease.

The length of the surgical procedure and the type of anesthesia administered, do not seem to make any appreciable difference, as far as the risk is concerned. As in so many other situations, the theme seems to be: "Use what you are most familiar with and what works best in your hands."

Does the nature and location of the infarction influence the risk factor? To a certain degree it does. For example, the transmural myocardial infarctions carry a much higher morbidity and mortality rate than does the subendocardial infarction. This is easily explained because the morbidity relates to the amount of muscle that is destroyed by the infarct.

The anterior infarction is due to occlusion of the anterior descending branch of the left coronary artery. This artery does not supply the S-A node or the A-V node, so arrhythmia problems are unlikely. However, this artery has
a wide distribution and its occlusion may lead to a large infarcted area and congestive heart failure may be a problem.

The posterior infarction is caused by occlusion of the right coronary artery, which has a limited distribution but does supply the S-A and the A-V nodes. In this type of infarction, the incidence of congestive heart failure is small, but we look for Stokes-Adams attacks and heart block.

The lateral infarction is caused by occlusion of the left circumflex artery, whose distribution is variable and the effects of which are variable.

The cardiac output

In dealing with the cardiac patient, we are concerned with the ability of the heart to maintain an adequate cardiac output. Cardiac output is dependent upon the condition of the heart itself, its contractile force, its rate, blood supply and anatomic integrity.

It is also dependent upon peripheral factors; such as, the resistance against which the heart works, the tone of the autonomic nervous system, and the rate of venous return. The volume and viscosity of the circulating blood, as well as its pH, oxygen tension, carbon dioxide tension, and electrolyte levels influence the cardiac output.

At St. Elizabeth Hospital in Youngstown, Ohio, where I practice, the cardiac output determinations are made by the thermodilution technique. This is done both in the cardiac laboratory and in the operating room. We have a certified heart-lung perfusionist, who is available on a 24-hour basis; and as a result, the calculation of the cardiac output can be done within a matter of 15 minutes. Other methods of determining the cardiac output are based upon the Fick method and the indicator dilution (dye) methods, which I will not discuss in this article.

The normal cardiac output at rest is usually considered to be 6 lit/min.8 This resting cardiac output gradually decreases with age. In order to compare the cardiac output of individuals of different size, cardiac output is frequently expressed as cardiac index. This is merely the cardiac output in lit/min divided by the surface area of the individual in square meters. The cardiac index is normally about 3 lit/min/square meter.

Factors influencing cardiac output

The regulation of cardiac output is quite complex. No one unifying concept is presently available to explain the interaction of all the known factors and feedback mechanisms which participate in its regulation. Here is a listing of some of the more important determinants of cardiac output.

1. Resistance, or Starling's law of the heart.

The heart itself has an autonomous ability to adapt to an increased volume or to increased resistance. Either of these events results in a larger volume of blood remaining in the ventricle at the end of systole. This stretches the ventricular muscle, which in turn increases the force of the next contraction. Thus, within physiologic limitations, the heart can automatically adjust to the increased load and maintain a fairly steady or slightly adjusted cardiac output. Both ventricles are capable of adjusting independently.

2. Autonomic nervous system.

Sympathetic stimulation increases the cardiac output. It is capable of producing outputs of up to 20-25 lit/min when maximally stimulated. Under normal circumstances there is a normal minimal sympathetic stimulation. If this normal sympathetic tone is eliminated, as in total spinal anesthesia, or diminished with drugs, the cardiac output diminishes. Sympathetic stimulation produces its effect by increasing the strength of the myocardial contractility and the heart rate.

The parasympathetic nervous system has the opposite effect. Stimulation of the parasympathetic system produces
a fall in cardiac output. It produces its effect not by altering the contractile strength, but by diminishing the heart rate.

3. Effective heart rate.
Within the limits of each individual heart, the heart rate will affect cardiac output. Increased rate up to a certain optimal rate, approximately 120 beats/min, will increase the cardiac output. Beyond this point and up to a rate of about 160 beats/min, cardiac output will be above normal, but not as great as the optimal rate. Beyond this limit, the cardiac output will fall below normal because there is insufficient time for the heart to fill with blood between beats.

Slowing of the heart rate to very low rates, such as 40 beats/min, will also diminish the cardiac output.

4. Disease.
Any disease of the heart, such as coronary occlusion with myocardial infarction, cardiac trauma, myocarditis or valvular disease, diminishes the effectiveness of the heart as a pump. Continued activity of the compensatory mechanisms is required to maintain near normal cardiac output even at rest. The ability of the heart to react to increased demand is seriously diminished.

5. Peripheral factors.
The peripheral resistance, which greatly affects the cardiac output, is itself affected by the volume and the velocity of the blood, the tone of the autonomic nervous system, the pressure and composition of the interstitial fluid, the effects of hormonal substances such as epinephrine and norepinephrine, the pH, oxygen, carbon dioxide and lactate content of the blood.

Also important is the ability of the venous side of the circulation to adapt by the pooling of blood in the various venous reservoirs, such as in the portal circulation and the pulmonary circulation. External forces exert considerable effect on the low-pressure, highly distensible venous bed, which can be favorably or adversely affected by such factors as positive-pressure breathing, abdominal compression, gravity, and body position.

As you can see, we are able in many ways to alter the cardiac output in the anesthetized patient, and this will be discussed later.

The preoperative period
A thorough preoperative workup is important in every patient. In the cardiac patient, it is extremely important. Included in this workup is the relationship between the cardiologist or internist and the anesthesiologist. If the working relationship is a good one, as in my institution, the evaluation of the cardiac patient and his ability to tolerate the proposed surgical procedure is greatly simplified.

Prior to surgery, all of our patients have a history and physical exam, CBC, urinalysis, and a SMA/18 study. All males over the age of 35 and all females over the age of 40 have routine ECG's. If lung disease is suspected, the patient has arterial blood gas studies and pulmonary function tests.

The routine ECG has proved interesting in our hospital. The age limits were stated previously for the routine ordering of the tracing, but we also order them in any patient younger than our routine limits if they give us any history that may lead us to suspect the presence of heart disease. These tracings are read by members of our internal medicine staff; and if any significant abnormality is discovered, the anesthesiologist is notified.

Our experience with the computerized reading of the ECG's was not a good one, as many errors were found in the interpretations. The fact that the interpreting cardiologist is usually the patient's own physician has helped us immensely, since he is usually familiar with the patient and is quick to pick up any ECG abnormality.

Another interesting aspect of the routine ordering of the ECG tracing is that a significant percentage, somewhere around 5%, show evidence of a myocardial infarction or myocardial isch-
emia in the absence of any medical history from the patient that normally would lead one to suspect heart disease. Finally, if the cardiologist recommends serial tracings or an exercise tolerance test, the surgery is postponed until these studies are carried out.

We are also concerned at this time about the presence and the severity of any coexisting disease the patient may have, such as, hypertension, diabetes, pulmonary disease and electrolyte disturbances. If we feel we cannot bring these disease states under reasonable control by the time of the proposed surgery, the surgery will be postponed.

Many patients with heart disease are on a multiple regimen prior to hospitalization. Some of the more common drugs are digitalis, diuretics, antihypertensives, antiarrhythmics, anticoagulants, beta-blockers, and miscellaneous drugs such as tranquilizers.

Electrolyte disturbances are common in patients who are taking digitalis and diuretics; and we find ourselves frequently dealing with the patient who is digitalized, has hypokalemia, a reduced blood volume, and may be in impending digitalis toxicity. If the patient has a serum potassium below 3.5 mEq/L, we administer potassium, usually 40 mEq over 4-6 hours, and then repeat the electrolyte study. A low potassium increases the rate of diastolic depolarization and this leads to arrhythmias and possibly to arrest.

The laboratory can give us the digitalis level of the blood and thus assist us in diagnosing the possibility of digitalis toxicity. Combined with an overnight fast, the patient who is on diuretics will have a reduced blood volume; and we can correct this by giving 1-2 liters of 5% Dextrose in 1/2-strength saline overnight to hydrate them.

We do not discontinue antihypertensive medication prior to the surgery. It is important to know both the drug and the dosage the patient is receiving to control his hypertension. Knowing this, you then know the mechanism of action of the drug and you will be able to intelligently treat hypotensive problems caused by the drug.

During the operation, the untreated, uncontrolled hypertensive patient is much more liable to suffer wide blood pressure swings and be a victim of a cerebrovascular accident or acute heart failure with elevated blood pressures. Conversely, the patient may have a severe reduction in cardiac output and arterial blood pressures when we use an anesthetic capable of reducing systemic vascular resistance. Untreated hypertensives also have a reduced blood volume, and this simply adds to our problems.

The patient exhibiting cardiac arrhythmias is not uncommon, and we run rhythm strips to determine the type of arrhythmia. Most of the arrhythmias are benign in nature and do not require therapy. Arterial tachycardia is treated with digitalis, even in the absence of heart failure, until the rate is controlled. Troublesome premature contractions can be controlled with quinidine or procainamide. Ventricular tachycardia will demand cardioversion.

First-degree heart block is innocuous and does not require therapy. Second-degree block is a different story. If there is any disorder of the Purkinje system (a long, wide QRS complex), then you must have means of pacing this patient at hand. In general, a long P-Q interval is not serious, but if it is accompanied by dropped beats, then transvenous pacing is mandatory. It takes less than 15 minutes to insert a transvenous pacemaker, and it may prove to be life saving. Third-degree heart block, of course, is a very serious situation with the slow ventricular rate, and some form of pacing is mandatory.

Adequate control of arrhythmias, will result in an improved cardiac output and a much smoother anesthetic course.

Anticoagulant therapy will cause hazardous operating conditions for the
surgeon and will preclude any form of spinal or epidural anesthesia. I prefer to discontinue the anticoagulant and allow the coagulation mechanism to return to near normal. Then, if indicated, the patient can receive small doses of heparin for the surgical procedure. This does not result in excessive bleeding during the surgery and offers the patient some degree of protection against thrombosis.

An increasing number of patients are receiving the beta-adrenergic blocking drug propanolol (Inderal®) for the treatment of angina pectoris, arrhythmias, hypertension, and occasionally, for the treatment of certain types of tremors. Propanolol reduces myocardial oxygen requirements by decreasing myocardial contractility, cardiac output, and heart rate.

The known cardiac depressant action of propanolol has made many anesthetists uneasy when dealing with the patient on this medication. Some authorities have advocated the discontinuance of this drug from two weeks to two days prior to the proposed surgery, on the assumption that there is a long lasting pool of the drug in the myocardium which will block the beta-receptor sites.

Recent evidence, and our own experience with the drug, has shown that it does not pose a significant problem when it is on board. The half-life of propanolol is from 3-6 hours after discontinuance after chronic administration, and it disappears from the plasma and the myocardium in 24-48 hours.4

In our hands, hypotension believed to be caused by the propanolol has responded readily to lightening of the volatile anesthetic, fluid administration, and the administration of mild vasopressors, such as ephedrine. Bradycardia has responded to intravenous atropine.

Other drugs used to combat propanolol myocardial depression are: isoproterenol (2-20 micrograms via IV drip to compete for the beta-receptors), glucagon (5-10 mg bolus followed by 1 mg/min IV drip) which has a separate receptor site on the cell wall and works independently of the beta-receptors, and calcium (250-1000 mg slowly IV) which works intracellularly at the level of the contractile proteins to improve myocardial contractility.4 For non-cardiac surgery, we allow the patient to take the drug until the day of surgery.

The question of prophylactic digitalization occasionally arises in the belief that it will prevent intraoperative or postoperative heart failure or arrhythmias in the cardiac patient. The incidence of intra and postoperative heart failure is small and usually responds to simple treatment measures, with the exception of those cases where the heart failure is secondary to a myocardial infarction.

The diagnosis and treatment of arrhythmias in digitalized patients who may have hypokalemia, alkalosis of acidosis, bleeding, and respiratory insufficiency may be exceedingly difficult. Most of the arrhythmias we commonly encounter are readily controlled by lidocaine, potassium, diphenylhydantoin, or discontinuation of the digitalis. It is known that digitalis toxicity is a primary or contributory cause of postoperative arrhythmias which can, in certain situations, prove hazardous. It is unwise to administer a potent drug that can cause complications; such as, digitalis, prophylactically without specific preoperative indications for the drug.

The only class of drugs that I feel should definitely be discontinued two weeks preoperatively are the monoamine-oxidase inhibitors. Patients on these antidepressants have a very variable response to premedication, anesthetic agents, and anesthetic adjuncts. In the cardiac patients, these responses could prove lethal. I do request that this class of drugs be withdrawn and have the patient placed on other forms of sedation or tranquilization.

May I also stress, that the preoperative period is the time to tell the...
patient of the risks of morbidity and mortality associated with the anesthesia and surgery. In these days, with our current malpractice problems, it would seem foolhardy to do otherwise. The doctrine of informed consent hangs over our heads, and we must respect the patient's bill of rights.

**Premedication**

The premedication given to the patient will depend both upon the anticipated type of anesthesia and the assessment of the patient's anxiety concerning the surgical procedure. In general, the sicker the patient, the less the premedication. If the patient is very anxious or if the patient is to have regional anesthesia, heavier premedication is preferred. The drug dosage must be individualized and this comes only with experience. Moderate doses of a tranquilizer-narcotic combination plus atropine or scopolamine, usually suffices. Others prefer a moderate barbiturate-belladonna combination.

In the cardiac patient, I prefer the use of scopolamine, because it enhances the sedation of the premedication and there is less chance of developing a tachycardia. If the patient arrives at the surgical suite with a slow pulse, it can be easily corrected with small intravenous doses of atropine. If the patient is scheduled for some form of regional anesthesia and the premedication is unsatisfactory, the desired tranquility can be achieved by sequential doses of droperidol, fentanyl, narcotics, or other drugs.

In our institution, the patients are given their premedication on the floors and may be unattended for a long period of time. The effects of heavy premedication may go unnoticed and respiratory depression, hypotension, or tachycardia may develop. The situation is serious in any patient, but may prove to be disastrous in the cardiac patient. Therefore, we lean to the theory of light premedication. The ideal situation would be to bring the patients to a holding area and administer the premedication intravenously, having adequate personnel available to treat any untoward effects.

The use of narcotics in the premedication has proved to be very satisfactory in our institution. Some authorities have a purist view concerning narcotic premedication and state that if the patient is not having pain, narcotics should not be used. In contrast, we have found a certain sense of relaxation and detachment in the patient under narcotics which we cannot seem to duplicate with hypnotics, tranquilizers, or other sedatives.

It is not deemed wise to use morphine to premedicate the patient with coronary artery disease, because it can cause contraction of the sphincter of Oddi and produce increased pressure in the biliary system; thus, leading to a pain state that is frequently misinterpreted as being the result of myocardial ischemia.

The patient with valvular heart disease also poses a problem. These patients are regarded as having a fixed cardiac output; that is, they can eject only a certain amount of blood from the heart during each systole, and they usually have a high degree of vasoconstriction to maintain their cardiovascular requirements to support life. These patients are particularly sensitive to the effects of myocardial depression, vasodilation, and alterations in cardiac rhythm and rate. Heavy premedication with large doses of parasympatholytics can easily upset the delicate cardiovascular balance these patients exhibit.

Because the prospect of surgery always produces some degree of anxiety, it is not inconceivable that patients with coronary artery disease and angina may have an anginal attack while waiting in the surgical suite for operations. It is just good practice to allow these patients to bring their own nitroglycerine to the operating theater, then they can feel secure in knowing that the nitroglycerine is immediately available to them should they need it.
Monitoring devices

Prior to the induction of the anesthesia, an appropriate monitoring system must be decided upon. The elaborateness of the monitoring system will depend upon the nature of the surgical procedure; so it follows that for minor surgical cases, the system will be simpler and will utilize non-invasive techniques. In all cases, the cardiac patient should have standard 4-lead electrocardiographic monitoring, with read-out devices, if possible, to watch for, diagnose, and if necessary, to assess the results of treatment of arrhythmias.

The read-out mechanism is particularly useful when the arrhythmia is associated with a rapid heart rate, and the complexes slide by on the oscilloscope screen at a fast rate. A rhythm strip taken on the read-out assists in deciphering the arrhythmia diagnosis, but occasionally a 12-lead strip is required.

The simpler surgical cases can have the traditional auscultatory method of blood pressure monitoring. This method is adequate for most cases, but does not function well in the patient who is hypotensive or in whom vasospasm is present.

Another non-invasive technique for blood pressure monitoring utilizes the Doppler effect. Here, a transducer is placed over an artery and ultrasonic sound waves are directed toward the artery. The ultrasonic waves are reflected from a moving surface, such as the pulsating arterial wall or the moving red blood cells, and are converted to an audible signal and the blood pressure is taken. This method is reliable for patients with low blood pressure and very useful for monitoring the blood pressure in infants.

For major surgical cases, intra-arterial monitoring of the blood pressure is extremely useful and the intra-arterial line can also be used to obtain blood samples for blood-gas determinations.

The measurement of central venous pressure is another useful monitor, especially in the patient who is in borderline congestive heart failure, or in the case where the anesthetist anticipates using large volumes of balanced salt solutions and/or blood. Before one can rely heavily on this monitoring device, the accurate placement of the catheter into the right atrium must be assured, and this can be determined usually by x-ray study. Central venous pressures in excess of 20 cm of water or abrupt rises in the CVP to this level or higher in response to limited fluid infusion can signal impending failure.

In our institution, we have found that the CVP does not always accurately reflect the hemodynamic and fluid volume patterns in the critical surgical patient. Usually, the CVP is used as an aid in determining the adequacy of fluid replacement in critical patients. If in these patients the CVP is low or in the low normal range, colloid solutions or salt-containing solutions are administered, in the belief that the volume load is insufficient. If the CVP is high, fluid administration is withheld.

However, in critically ill patients it is incorrect to assume that the right atrial pressure is always an accurate reflection of the pressures found in the left heart. Disparate hemodynamic forces between the right and left ventricles can occur and must be taken into consideration when fluids are given to critical patients.

Elevation of the CVP can be caused by a variety of pathologic entities which exert their effects between the aortic valve and the tip of the measuring CVP catheter, but these may not be related to body fluid volumes. Examples of such entities are: congestive heart failure, valvular heart disease, myocardial infarction, pulmonary hypertension, cor pulmonale and congenital heart disease. Here again, the CVP may be in the normal or low range and yet the patient may have pulmonary edema and left ventricular failure.

The pressure which best defines left ventricular function and the ability of
the heart to handle a fluid load is the left ventricular end diastolic pressure, but this cannot be measured routinely in most critical patients. The next best measurements that directly reflect left ventricular pressure changes and reserve, are the left atrial and pulmonary capillary wedge pressures. When these pressures are elevated, the indication is that the left ventricle is failing, cannot empty completely, has a poor output, and cannot accept any further fluid load. These pressures seem to be much more sensitive than the CVP in determining fluid management in critically ill surgical patients.

The Swan-Ganz Flow-Directed Thermodilution Catheter is becoming more commonly used today to determine left heart function. This catheter is introduced via the subclavian vein into the right atrium, its balloon tip is inflated, and then is floated into the pulmonary artery. The catheter is then further advanced to obtain a pulmonary wedge pressure. This single catheter placement provides the means for measuring pulmonary artery and pulmonary capillary wedge pressures, right atrial pressure, and sampling of blood from either the right atrium or the pulmonary artery, as well as allowing for an injection of a cold solution and detection of temperature change for determination of cardiac output. Pulmonary congestion rarely occurs with wedge pressures lower than 18 mmHg.

Other monitoring devices that are useful are: the measuring of the urinary output to determine renal function, and the monitoring of the body temperature. Shivering in the postoperative period can be costly to the cardiac patient, and every effort should be made to keep body temperature near normal throughout the surgical procedure. Tympanic membrane, esophageal or rectal probes can be utilized.

Induction of anesthesia

The induction period is one where the anesthetist should insist on complete silence on the part of all operating room personnel. The patient is anxious, sometimes confused by the premedication, and frequently misinterprets remarks made in the operating room. Every effort should be made to make induction as smooth as possible.

In the adult cardiac patient, the use of thiopental for induction is usually satisfactory. Following a period of pre-oxygenation, a sleep dose of thiopental is administered. Occasionally, this is followed by some degree of hypotension and it may be severe enough to require treatment. In any patient with a disease state that is accompanied by a slowed circulation time, one must remember that the onset of action of any intravenous drug is delayed. These patients will take a longer time period to go to sleep; and if you are not aware of this, overdosage with thiopental will occur.

Patients who have valvular disease are victims of the fixed cardiac output state written about earlier. In these patients, compensatory mechanisms such as vasoconstriction are in play to provide a cardiovascular status sufficient to support life. Administering a drug such as thiopental, which can cause myocardial depression and vasodilation to this type of patient, can lead to serious hypotensive states.

These patients should be induced with ketamine at a dose rate of 1-2 mg/kg IV. Ketamine does not stimulate catecholamine secretion in the patient, but rather acts like cocaine, preventing the uptake of the catecholamines at the post-ganglionic sympathetic nerve terminals. The result is one of a mild sympathetic stimulation.

At dose ranges of 100-150 mg for induction, we have not observed any great increases in pulse rate or blood pressure, and we have not observed any episodes of postoperative hallucinations or bad dreams. Ketamine has also proved to be a good induction agent for the critical patient in general; but it is wise not to use the drug in the patient who has severe hypertension, elevated intracranial pressure, or in whom eye surgery is contemplated.
Infants and children with cyanotic heart disease will have prolonged induction with an inhalation agent because of their large intracardiac shunts and diminished pulmonary blood flow. Examples of this state are Fallot's tetralogy, pulmonary stenosis with an atrial septal defect, and transposition of the great vessels. In congenital heart disease associated with septal defects and pulmonary stenosis, the onset of arterial hypotension can increase the right-to-left cardiac shunt and the degree of cyanosis. This is because the pulmonary resistance is fixed and when there is a decrease in the systemic vascular resistance, blood flows from right to left through the septal defect to the lower resistance of the systemic arterial tree.

If, in these patients, an inhalation induction is deemed necessary, it is best carried out with an agent that has a low blood gas solubility coefficient, such as cyclopropane. The use of cyclopropane in the infant-circle systems allows a high concentration of oxygen and does not cause hypotension in anesthetic dose ranges. These patients also do well with intramuscular or intravenous ketamine inductions.

For the cardiac patient in general, it is best to avoid rapid induction and intubation techniques. Intubation in light planes of anesthesia produces arrhythmias by elevation of blood pressure and sympathetic stimulation.

When the patient's disease state is so severe that a depth of anesthesia is unattainable because the reflex activity of the trachea cannot be obtunded without marked depression of the cardiovascular system, then it is advisable to spray the cords and upper part of the trachea with a topical anesthetic to eliminate these reflexes prior to the intubation. A 4% topical lidocaine spray is satisfactory for this purpose. In any event, clinically deep levels of anesthesia should be avoided.

Choice and maintenance of anesthesia

The failure to pinpoint indicators of myocardial infarction or other complications in the cardiac patient indicate multiple or unknown factors at play. This point of view is bolstered by a similar inability to blame any anesthetic agent or any anesthetic technique as a primary cause of myocardial infarction following surgery.

Infarcts occur during or following general, regional or local anesthesia often in spite of apparently flawless technique. Neither circulatory lability, degree and duration of hypotension during the operation, duration of anesthesia and surgery, volume of blood replaced, nor deviation from normal or preinduction blood pressure can be correlated with myocardial stress as assessed by ECG changes, blood enzyme values, and clinical course. Still, as for many years, the best advice is to avoid hypoxia and unstable blood pressure during and after anesthesia and surgery.

For operations on the extremities, nerve block anesthesia is very satisfactory in the hands of an expert, especially when the cardiac patient has a pulmonary problem. The axillary approach for brachial plexus block, elbow block, wrist block, and digital block can be useful for surgical procedures on the upper extremity. The intravenous lidocaine block is also excellent for this type of surgery; but the anesthetist must be very careful to check the function and adequacy of the dual cuffs and must make sure that the cuffs are deflated slowly at the end of the surgical procedure.

For prostatic, perineal, and lower limb surgery, spinal and epidural anesthesia can prove excellent if care is taken to avoid hypotension. Epidural anesthesia is often recommended because it takes effect more slowly than a spinal block and allows more time to prevent or correct hypotension by infusion of balanced salt solutions or the judicious use of a vasopressor. Placement of a catheter permits continuous spinal anesthesia even into the postoperative period, if this is desired for analgesia.
Ankle block anesthesia has proved very useful for surgery on gangrenous toes. Block techniques permit adequate oxygenation, avoid postoperative depression, and in properly selected and prepared patients, often provide the simplest and safest anesthesia.

General anesthesia should include at least 50% oxygen in the inspired mixture to provide a moderate safety factor in case airway obstruction or transient hypotension should develop. The selection of the primary anesthetic agent will depend upon the experience of the anesthetist. In general, it is best to use the agent with which you are most familiar.

However, in many situations, this is impossible. The surgeon’s preference to use electrocautery or the need for intraoperative x-rays will preclude the use of explosive agents. The surgeon’s technique for a particular case may include the injection of epinephrine, and this will preclude the use of agents incompatible with epinephrine.

The patient’s history and physical status may preclude the use of other agents. For example, the patient with liver disease should not receive halothane; the patient with severe hypertension should not receive cyclopropane or ketamine; the patient with renal disease should not receive methoxyflurane, and so on.

If they can be used, the older agents—diethyl ether and cyclopropane—are excellent agents for the poor risk patient. There have been many times in my experience that I have switched from an inhalational agent to light diethyl ether anesthesia to recover and maintain cardiovascular stability in the poor risk patient.

Light halothane anesthesia, in a dose range of 0.3-0.5%, has proved to be an effective agent with minimal myocardial depression and vasodilation. Enflurane anesthesia is well tolerated by cardiac patients and has the advantage of good muscle relaxation, especially with pancuronium as the muscle relaxant. The patient receiving enflurane maintains good spontaneous respirations, which are slow and deep in contrast to the tachypnea, frequently seen with halothane.

Both halothane and enflurane are incompatible with epinephrine and have hypotensive effects as the level of anesthesia is deepened. Fluroxene, in a dose range of 1.2%, has proved to maintain good cardiac output, sympathetic tone, central blood volume, cardiovascular reflexes, and peripheral resistance.

The use of balanced anesthesia has also worked well in experienced hands. Innovar®, droperidol, fentanyl, morphine, and Demerol® combined with nitrous oxide and a muscle relaxant can be a very effective anesthetic technique. The anesthetist must remember that the one disadvantage associated with the use of balanced anesthesia is in the inability to lighten the level of anesthesia quickly, as can be done when primary inhalation anesthesia is employed.

For many years, cardiologists have used morphine via the intravenous route to relieve the pain caused by myocardial infarction. It provided good analgesia with minimal cardiovascular depression. Anesthesia employing the narcotic technique is an extension of narcotic analgesia which is widely used for poor risk patients.

This technique involves induction with sequential doses of a narcotic and 50-60% nitrous oxide, maintenance with nitrous oxide, a muscle relaxant, and sequential doses of a narcotic as required. Induction is relatively slow, hypotension is rare, and the respiratory depression caused by the narcotic can be obviated with controlled respiration. At the end of the operation, residual narcotic depression can be reversed with the narcotic antagonist naloxone.

Morphine in doses of 1-2 mg per kg of body weight, can be used if the patient is to have assisted ventilation overnight following operation. Occasionally, this morphine technique is complicated by the development of hy-
pertension, which may have to be treated in the cardiac patient, and by the liberation of antidiuretic hormone in the patient, which may pose a problem in the postoperative period.

Meperidine has a shorter duration of action and is so rapidly metabolized that some patients will not require narcotic reversal after doses of 5-10 mg per kg used in 3-5 hour procedures. However, the problem of hypotension does occur with meperidine. Recently, fentanyl, a potent, rapid onset, ultrashort acting narcotic has become available. An induction dose of 300-500 micrograms can be supplemented with 50-100 micrograms every 30-45 minutes.

Thus, the availability of narcotics with varying durations of action and a specific narcotic antagonist, make the narcotic anesthetic technique extremely versatile in experienced hands.

A word of caution is indicated on the subject of narcotic reversal. The use of naloxone to reverse residual narcotic respiratory depression is commonplace. When reversing a long-acting narcotic, such as morphine, an intravenous dose of 0.4 mg naloxone may effectively reverse the respiratory depression for a limited period of time.

As the naloxone is metabolized, the depressant effect of the longer acting narcotic is re-established, and if this is not recognized, the patient may be in jeopardy. If prolonged ventilator therapy is not indicated, the intravenous dose of naloxone should be accompanied by an intramuscular dose also to provide a longer duration of action of the antagonist.

The choice of muscle relaxants for the surgical procedure is again tempered by experience. For relatively short cases, the use of a 0.1% succinylcholine drip has proved satisfactory when precautions are taken to avoid overdosage. For the longer surgical procedures, the use of the non-depolarizers, d-tubocurarine or pancuronium, are indicated. The non-depolarizers have the advantage of effective reversal at the termination of the surgical procedure with anticholines-

terase drugs, but this is not always innocuous in the cardiac patient.

The use of the nerve-muscle stimulator during the surgical procedure does aid in preventing overdosage of the patient with muscle relaxant. There are reports in the literature documenting the development of tachyarrhythmias with the use of pancuronium; and this has been our experience, especially when the relaxant is combined with an Innovar®-fentanyl-nitrous oxide technique. The cardiac patient may not be able to tolerate this situation.

Blood pressure control during anesthesia for cardiac patients, especially those with coronary artery disease, is always of great concern to the anesthetist. As the coronary artery disease progresses, the coronary vessel becomes more rigid, and the blood flow through it becomes more pressure dependant. Moderate reductions of blood pressure are tolerated quite well by cardiacs, but the arterial pressure should not be allowed to fall more than 15-20% below the preoperative pressure level.

A greater danger to the cardiac patient is the development of hypertension. This should be avoided or treated, because an excessive increase in systolic pressure increases cardiac work in an already overburdened heart.

Management of arrhythmias

Cardiac arrhythmias do occur during anesthesia and surgery in the patient with a normal heart. Such arrhythmias can be of many types, have many etiologies, and are usually innocuous. However, when they develop in the cardiac patient, they have a more serious connotation and should be carefully evaluated.

Not all arrhythmias require treatment, but when the ventricular rate approaches 150 beats/minute, the result is a fall in cardiac output because of decreased ventricular filling time. Runs of premature ventricular contractions can lead to ventricular tachycardia or ventricular fibrillation. The onset of bigeminal rhythm in the cardiac patient
denotes an ectopic focus in the ventricle and ventricular irritability. When arrhythmias occur in spite of, or are not corrected by, both adequate ventilation and oxygenation, as well as when possible, by lightening the depth of anesthesia, then they should be treated.

When atrial flutter or fibrillation occurs with rapid ventricular response and cardiac output is decreased, as evidenced by hypotension, a fast-acting digitalis preparation should be given. Digoxin, administered intravenously, is suitable; and its onset of action is within 15 minutes. An initial dose of 0.25-0.50 mg is given, and the ventricular rate is watched. If no improvement is noted within 20 minutes, the same dose is repeated. Usually 1 mg of digoxin in divided doses will reduce the ventricular rate. The patient who has been on digitalis prior to the surgery will, of course, require smaller doses of digoxin to control the ventricular rate.

Lidocaine is of little value in the treatment of atrial arrhythmias; but it is of considerable value in the treatment of ventricular arrhythmias, such as, premature ventricular contractions and bigeminy. It can be given in the dose range of 50-100 mg every five minutes or in an intravenous drip should the situation warrant it.

Procainamide is used to treat both atrial and ventricular tachyarrhythmias. The dose is 100 mg every five minutes. Procainamide has a slower onset of action than lidocaine, but a longer duration of action.

Diphenylhydantoin, in doses of 50-200 mg given slowly IV, is also effective therapy for many of the atrial arrhythmias.

Propanolol is an effective agent for the treatment of ectopic atrial and ventricular tachycardias in the dose range of 0.5-3.0 mg. It is a very potent beta-adrenergic blocker; and it must be used carefully in the patient with poorly compensated heart disease, because it can lead to congestive heart failure. It has been used effectively in the treatment of ventricular arrhythmias associated with digitalis toxicity.

The postoperative period

The normal recovery room care given to cardiac patients does not vary a great deal from the proper care given to non-cardiac patients. However, one must keep in mind that the margin of the cardiac patient's tolerance to even slight deviations from his preoperative physiologic status is less than in other patients. If the patient is not watched closely and if the postoperative stresses are not kept to a minimum, the already overburdened myocardium may not be able to cope with the stressful state.

Any patient undergoing major abdominal surgery or thoracic surgery can exhibit hypoxia and electrolyte imbalance for as long as three days postoperatively. If this type of surgical patient also has a positive history of a recent myocardial infarction, arrangements should be made to send him to an intensive care facility upon discharge from the recovery room. Eighty percent of the patients who die from recurrent myocardial infarction expire within the first 48 hours after the infarction.

This suggests that arrhythmias, rather than a low cardiac output, may be the primary cause of death. It can be assumed that if all patients with coronary heart disease and previous infarction were monitored and arrhythmias were treated in the intensive care units soon after operation, the mortality rate would decline.

The cardiac patient is prone to develop arrhythmias in the recovery room. Continuous ECG monitoring with rhythm strip availability is mandatory. Arrhythmias occur readily in the patient who is digitalized and in the patient who is alkalotic and hypokalemic. Arterial blood gas studies and electrolyte determinations will determine proper treatment in these situations. If ventilator adjustment or electrolyte therapy does not correct the arrhythmia, then the administration of the anti-arrhythmic drugs considered earlier is indicated.
Adequate ventilation must be assured and states of hypoxia, acidosis, and alkalosis must be prevented. Except for those patients having minor surgery, every patient should be placed on a ventilator. The endotracheal tube should be left in place until respiratory function is attained and adequate oxygenation with room air is assured. This may involve the careful use of the technique of muscle relaxant reversal and the use of narcotic antagonists. The adequacy of ventilation can be assured only by arterial blood gas studies.

Blood volume determinations, if interpreted correctly, will give an indication of the adequacy of the blood and fluid replacement during surgery and will help in deciding upon further fluid therapy. Effort should be made to reduce the viscosity of the blood in cardiac patients to provide for improved peripheral blood flow with less myocardial work. This can be done by keeping the hematocrit in the range of 35%.

Control of shivering is important. The body temperature must be monitored in the recovery room as it was during the surgical procedure. The shivering process creates a large oxygen demand and may render the patient relatively hypoxic. The cold patient should be warmed by K-pads or heated blankets.

The patient should have an assist in awakening, especially if inhalation agents with low blood-gas solubility coefficients have been used for anesthesia and are blown off rapidly, leading to a state of mental confusion with the accompanying disorientation and thrashing. Small doses of a narcotic or sedative can make the awakening process a smoother one. These patients should have an increased inspired oxygen tension administered to correct the hypoxia from shivering. If necessary, the use of chlorpromazine can be used to control the shivering process.

Summary

The successful management of the patient with cardiac disease for anesthesia and surgery can be enhanced by remembering the following:

1. The cardiac patient, depending on his degree of compensation for the disease, may have a greatly reduced tolerance to cardiovascular stress.

2. The anesthetist may be able to reduce the degree of cardiovascular stress by: (a.) Understanding the pathophysiology of the disease state. (b.) Deferring elective surgical procedure for at least six months following a myocardial infarction. (c.) Using the preoperative period to thoroughly evaluate the patient and to correct abnormal heart function and associated disease states as much as possible. (d.) Effective monitoring of the patient to detect significant changes in cardiorespiratory function and by employing effective therapeutic techniques should these changes occur. (e.) Selecting the appropriate regional or general anesthetic techniques most suitable to the patient's physical status and to the site of operation. and (f.) Utilizing the postoperative period to effectively monitor and treat the patient to maintain optimum cardiorespiratory function.

3. The patient with a cardiac problem is also a human being. He is usually medically sophisticated and very much aware of his surroundings. Expression of genuine concern on the part of the anesthetist concerning the patient's welfare and a thorough explanation of the role the anesthetist will assume as a part of the surgical team will do a great deal for the patient's emotional well being.

REFERENCES


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This paper was presented at the American Association of Nurse Anesthetists 42nd Annual Meeting, Clinical Session, and Graduate Course held in Chicago during August, 1975.