CLINICAL PROGRESS

Anesthesia in Patients with Heart Disease

By Leroy D. Vandam, M.D., and Thomas K. Burnap, M.D.

The consensus among internists, surgeons, and anesthetists today is that patients with heart disease fare well when anesthetized. Although this is primarily a clinical impression, there is a growing volume of literature to support this thesis. One must distinguish, however, between patients with heart disease who are undergoing surgery on other organ systems and those who are to undergo cardiac surgery itself. It is not easy to gather data on this subject because of the difficulty in comparing anesthetic morbidity and mortality in like groups of patients. For example, one clinic might examine the results of an anesthetic technic employed for mitral valvuloplasty and find no mortality, whereas another group might find the mortality to be 15 or 20 per cent. The explanation for this difference probably would be that in the first report the individuals anesthetized and operated on had relatively benign mitral stenosis while in the second, the patients might have fallen into a group IV functional classification. Thus the first consideration in evaluating the effects of anesthesia must be the severity of the heart disease.

In discussing anesthetic agents and technics it is well to remember that usually it is the skill with which an anesthetic is given rather than the choice of a particular agent that determines the safety of the procedure. Likewise the other phases of the operation are just as important as the anesthetic in the outcome. It should not be forgotten that anesthesia is but a small part of the total surgical experience. The patient with heart disease often undergoes a relatively long period of medical preparation. Before operation he may be beset with great anxiety. Preoperative medication is given to counteract this. Immediately prior to operation there is starvation and then the administration of the anesthetic. The patient then undergoes a surgical procedure that has many consequences from the standpoint of the postoperative course. Finally, after anesthesia and operation are over, there are many natural bodily responses to that which has gone before. Among these various things it may be very difficult to discern the deleterious effects of the anesthetic agent or technic.

It is the purpose of this brief review to delineate some of the factors in the total operative experience that may contribute to anesthetic morbidity and mortality. We shall discuss the role of preoperative preparation, of preanesthetic medication, of the anesthetic agent and technic, and the operation itself in the problems that are encountered during anesthesia. An understanding of these matters enables the internist to prepare his patient for anesthesia and operation rationally.

Medical Preparation of the Patient

The very drugs that are employed to bring the patient into the best possible state before operation are often the ones that create difficulty during anesthesia. The use of digitalis is perhaps the best example. It is well known that it is difficult to achieve just the right amount of digitalization in the very ill cardiac patient. Inadequate or "overdigitalization," not previously recognized, will pose considerable problems for the anesthetist. If a patient is on the verge of toxicity, it requires just a little extra stress during induction or maintenance of anesthesia to produce
increasing degrees of heart block, serious ventricular arrhythmias, and even cardiac arrest. Induction of anesthesia may be likened to the effect of any stress, such as exercise, in revealing digitalis toxicity. Despite the best preparation and management as far as anesthesia is concerned, respiratory obstruction, excitement, and breath-holding may precipitate the difficulties enumerated above. Perhaps the most unpredictable patient in this regard is the patient who is “digitalized” just the day before operation. It hardly seems possible that adequate digitalization, enough to withstand the stresses of anesthesia and operation, can be accomplished in such a short period of time.

In the case of inadequate digitalization the prospect of rapid heart rates, cardiac failure, and pulmonary edema looms large. This is one reason why, prior to anesthesia, one tries to counteract anxiety as much as possible to avoid a rapid heart rate that may precipitate pulmonary edema. In the seriously ill cardiac patient who has been given digitalis, electrocardiographic monitoring throughout the course of anesthesia and operation seems almost mandatory. If a tachycardia or arrhythmia is observed, one needs the electrocardiogram for accurate diagnosis and to choose the appropriate drug for treatment.

Although we have written mainly about digitalis, it should be also pointed out that quinidine, procaine amide, the prior use of cortisone, serpasil, chlorpromazine, and anti-hypertensive drugs, all make the administration of anesthesia more hazardous by the production of arrhythmias and hypotension.4,5

Another factor in the development of arrhythmias or hypotension during induction and maintenance of anesthesia is the practice of instituting vigorous diuresis prior to surgery in order to secure a “dry” body weight. Vigorous diuresis may not only lower the blood volume but may lead to electrolyte imbalance, particularly with regard to sodium and potassium,6 so that hypotension and serious cardiac arrhythmias may ensue. The combinations of digitalis, electrolyte imbalance, and the effects of anesthetic agents on the circulation are so interrelated that it is surprising that more arrhythmias and hypotension are not seen.

**Preanesthetic Medication**

It is the purpose of preanesthetic medication to maintain the patient in good condition by allaying apprehension and to counteract some of the undesirable effects of anesthesia, such as increased salivary secretions and un-
tity of atropine needed to block the vagus adequately is often unsafe for the heart patient in that tachycardia and hypotension may be produced. If a patient is satisfactorily digitalized and the heart rate is slow, the administration of atropine may increase the pulse rate to hazardous levels. Large doses of atropine in addition to the muscarinic-blocking on the heart interfere also with ganglionic transmission in the autonomic nervous system. Thus the patient given a large dose of atropine may also evidence postural hypotension.8

In the prevention of tachycardia a possible side effect of meperidine on cardiac rate must be considered. This drug, which is a good analgesic and similar in most respects to morphine, has the additional property of producing vagal block. There have been instances wherein the use of meperidine has precipitated a rapid atrial rate and heart failure when given to patients with slower rates and atrial flutter.9

These details about preanesthetic medication are mentioned to emphasize their possible untoward effects in the cardiac patient who is about to undergo anesthesia. These drugs can and must be used with caution. In moderate dosage the barbiturates are safest for avoidance of apprehension. The interaction of the tranquilizing and anesthetic drugs has already been noted to produce undesirable side effects. Reserpine (Serpasil) because of the slow rate of excretion must be discontinued many days before anesthesia lest hypotension supervene. Atropine, unless employed for the treatment of patients with complete heart block, should be used sparingly for the effect on salivary secretion. The same precautions apply to the employment of morphine or meperidine unless they be needed for the relief of pain.

Anesthetic Agents

All of the potent general anesthetic agents exhibit effects in common on the cardiovascular system. These agents are di-ethyl ether, cyclopropane, chloroform, di-vinyl ether, ethyl chloride, and some of the newer fluorinated compounds. While ether and cyclopropane are used most frequently, it should be understood that all these agents decrease myocardial contractility in direct proportion to the blood level of anesthetic achieved. Ordinarily hypotension is not perceived during anesthesia because of various compensating vascular reflexes. The effect on cardiac output of these agents is best seen in the heart-lung preparation where such reflexes are not brought into play.10 However, the deeper the level of anesthesia the more frequently is hypotension seen; particularly so in the patient with heart disease. Despite opinion to the contrary, overdosage with di-ethyl ether is a frequent cause of hypotension. According to experiments in the dog, were it not due to a coincident reflex release of epi-nephrine and norepinephrine during di-ethyl ether anesthesia, this commonly observed hypotension would be almost universally seen.11

Because the anesthetist knows that hypotension accompanies the use of potent inhalational agents, he has adopted technics that allow him to use these agents sparingly. It is not uncommon in operations on the heart, for example, to administer di-ethyl ether so that the patient is maintained in the first stage of anesthesia, the stage of analgesia. Such light anesthesia avoids the depressant effect on the cardiac output of greater blood levels of anesthetic. The patient experiences no pain and can frequently respond to simple commands. In the postoperative period he will have little recollection for the events during operation. Similarly the use of the muscle relaxants has enabled one to use the weaker general anesthetic agents, nitrous oxide and ethylene, with high concentrations of oxygen for the performance of major surgery. Generally all that is required of anesthesia for a major surgical procedure is a combination of analgesia, amnesia, and muscle relaxation. Analgesia and amnesia usually can be attained by the inhalation of mixtures of 70 per cent nitrous oxide in oxygen combined with the intravenous injection of one of the muscle relaxants, so that the sur-
geon encounters muscle relaxation and has ready access to the field.

Other common effects of the general anesthetic agents involve the peripheral vasculature. A most striking effect after the onset of general anesthesia is the rapid appearance of vasodilatation in the skin. This vasodilatation is shared by skeletal muscle and is equivalent to that which might be obtained from a chemical or surgical sympathectomy. In other words, the blood flow to the extremities increases as much as 4 times early in the course of anesthesia. Were it not for compensatory vasoconstriction in other vascular beds, arterial hypotension would be encountered frequently. When vasodilatation in the extremities is greatest, vasoconstriction in the liver and kidneys has been found. The cerebral and coronary circulations are not involved in this compensatory vasoconstriction. Just why vasodilatation in the skin and muscles takes place is not clear at present. As anesthesia is continued, however, vasodilatation in the periphery disappears and the hepatic and renal circulations are restored toward normal. Appreciation by the anesthetist of these circulatory alterations has done much to eliminate the hazards of general anesthesia.

**Anesthetic Technic**

It may seem trite to say that in the last analysis all patients, especially those with heart disease, encounter trouble during anesthesia because of anoxia. Anoxia, as in waking life, is related either to respiratory or circulatory deficiency. Frequently one cannot distinguish between the two. Failure to give oxygen or to relieve respiratory obstruction during anesthesia is just as hazardous as arterial hypotension. If anoxia is the basic fault, it follows that patients with pre-existing difficulty in coronary arterial perfusion are those who will suffer most under faulty anesthetic management. Consequently, the patient with coronary arterial disease or aortic valvular disease, is the one who will tolerate anoxia least. Similarly patients who are apt to die suddenly in waking life are those who will expire in the advent of anesthetic difficulty. Thus, people with complete heart block and Stokes-Adams attacks, and patients prone to the development of acute tachycardias and arrhythmias are serious problems in anesthesia. Good anesthetic management lies in the understanding of the pathologic physiology of the patient’s heart disease.

Anoxia and hypotension should be avoided during anesthesia in the patients with heart disease. This is sometimes more easily said than done. Oftentimes hypoxia and hypotension intervene despite the best anesthetic management. The discussion that follows will attempt to point out a few of the many factors other than faulty technic that lead to anoxia and hypotension during anesthesia.

The manner in which anesthetic technic may create difficulties for the cardiac patient may be seen in the situations of inadequate pulmonary ventilation, intubation of the trachea, and the appearance of cough. Adequate pulmonary ventilation is necessarily part of good anesthetic technic. Unfortunately practically every anesthetic agent and adjunct interferes in some way with normal respiration. Preanesthetic medication, whether barbiturate or opiate, may depress the response of the respiratory center to carbon dioxide. The general anesthetic agents also depress the response of the respiratory center. Were it not for stimulatory peripheral respiratory reflexes, respiratory depression would be seen even with di-ethyl ether. The muscle relaxants interfere with respiration at the periphery through a blocking effect on neuromuscular conduction. The severity of the respiratory problem may be enhanced by an open thorax or certain operative positions such as the lateral decubitus and Trendelenburg. These factors are noted to illustrate the complexity of the respiratory problem in anesthesia. There are few practical monitoring devices to warn the anesthetist that pulmonary ventilation is inadequate.

The end result of inadequate pulmonary ventilation is always anoxia and carbon di-
oxide accumulation. Oxygenation is more easily maintained than the elimination of carbon dioxide. Consequently, respiratory acidosis has loomed large in the explanation of some of the untoward circulatory effects of anesthesia. Increased blood levels of carbon dioxide decrease myocardial contractility, predispose the heart to vagal influence, and may be the reason for the development of certain ventricular arrhythmias and arterial hypotension seen during or at the conclusion of anesthesia.

If one examines the circumstance during which cardiac arrest occurs most frequently during anesthesia, it will be found that tracheal intubation is often a precipitating event. It has been assumed that stimulation of the trachea gives rise to vagal stimulation with consequent cardiac inhibition. This has been called a vago-vagal reflex. However, this is not the customary response. Ordinarily during intubation of the trachea if there is adequate oxygenation and a satisfactory depth of anesthesia, the response to tracheal stimulation is that of tachycardia and hypertension.15 Seemingly a sympathetic rather than a vagal response is elicited. This can be confirmed by anyone who observes the electrocardiogram during intubation. Nevertheless tachycardia and hypertension may place considerable strain on an already diseased heart. Ideal conditions, technical facility, and the appropriate level of anesthesia can minimize these sympathetic effects.

Coincident anoxia or carbon dioxide retention determines the response to tracheal intubation. Vagal effects similar to those ordinarily seen in other species such as the dog are prone to occur when intubation is performed in man under such circumstances.

Cough and straining in response to irritation of the trachea by an endotracheal tube or catheter may add to the circulatory changes mentioned. The circulatory consequences of the Valsalva maneuver may be reproduced. Closure of the bronchi and a prolonged expiratory effort during cough can lead to hypotension and circulatory collapse through interference with the return of venous blood to the thorax and right atrium. The type of heart disease and the anesthetic agent employed may enhance or minimize the effect of the Valsalva maneuver. Thus, if there is a high central venous pressure, as in mitral stenosis or constrictive pericarditis, the effect of increased intrathoracic pressure on venous return to the heart will be minimized.16 Similarly if an agent such as cyclopropane is used, which is associated with the development of a high central venous pressure in deep anesthesia, the effect of the Valsalva maneuver is not apt to be as great as with other agents. The patient with a poor cardiac output or the patient with diminished coronary arterial blood flow is apt to die suddenly when the Valsalva maneuver is exerted coincident with the circulatory depressant effects of anesthetic drugs. We have seen patients with heart disease die when the trachea was suctioned to remove secretions in the treatment of atelectasis. Similarly we have seen patients with aortic stenosis and aortic insufficiency die during induction of anesthesia when uncontrolled cough ensued.

The internist must have observed events such as those related here to realize that often it is the anesthetic management rather than the choice of anesthetic agent that leads to circulatory difficulties and sometimes death.

A good rule for medical consultant, surgeon, and anesthetist to observe is that the anesthetic procedure ought never to be more hazardous or complicated than the proposed operation. Local anesthesia is a simple technique. Most of the undesirable pharmacologic and technical accompaniments of general anesthesia are avoided with this method. In using local anesthesia, however, one must be sure that apprehension during the course of operation with a consequent endogenous release of sympathomimetic amines, does not increase the work of a diseased heart. A disproportion between the demands of the myocardium for oxygen and the available coronary arterial blood flow may lead to ischemia or infarction. Likewise, the possible hy-
potentiose and myocardial depressant effects of local anesthetic agents should be anticipated. Rapid absorption from the injection site with the development of high blood concentrations gives rise to these quinidine-like effects. These remarks should not negate the value of local anesthesia. Proper preanesthetic preparation, use of minimal quantities of anesthetic, and good technique will eliminate these sequelae.

The versatile anesthetist often elects spinal as a type of local anesthesia for surgical procedures in the cardiac patient. This choice sometimes confounds the medical consultant who is aware of the fact that arterial hypotension is common with this method. Yet spinal is probably the least stressful anesthetic of all from the standpoint of administration. The respiratory, metabolic, and technical disadvantages of general anesthesia are lacking. Muscle relaxation is excellent during operation and hypotension can be controlled by the prophylactic administration of pressor amines. An important consideration in this instance of hypotension is the lesser cardiac work required against a diminished peripheral circulatory resistance. The need for coronary circulation is not so great and the hypotension is less hazardous to the myocardium.

Surgical Procedure

Compounding these anesthetic problems already mentioned is the surgical operation, which in its mere technical aspects poses hazards for the patient with heart disease. We have already mentioned the role that the position of the patient on the operating table might play in the production of hypotension or deficient pulmonary ventilation. Sudden blood loss with the production of hypotension need be mentioned only in passing. Certain surgical manipulations within the thorax or abdomen may produce arterial hypotension, changes in heart rate and rhythm. The best known of these is the abdominal traction reflex that gives rise to arterial hypotension. This reflex has been variously ascribed to traction on the mesentery, to stimulation of the celiac plexus, or to interference with the return of venous blood to the heart by the placement of abdominal packs. Recently it has been shown that mechanical stimulation, particularly to the parietal peritoneum of the upper abdomen, may cause a sudden fall in blood pressure of considerable magnitude.17 The pathways of this reflex have not yet been mapped out but the adequate stimulus has been assumed to be a deformation of the peritoneum with stimulation of Pacinian corpuscles. In association with hypotension thus produced bradycardia is a frequent complication. It is for this reason that the vagus has been implicated in the reflex arc. In order to explain the rapidity of development of hypotension it has been postulated that the vagus exerts an effect on ventricular myocardial contraction despite anatomic evidence to the contrary. That this reflex is not entirely benign has been shown by the postoperative discovery of silent myocardial infarction in patients with these circulatory changes.18

Summary

Anesthesia for the patient with heart disease has become increasingly safer with the passage of years due to a better knowledge of the physiology of heart disease and of the pharmacologic action of anesthetic agents themselves. There are few statistics to support the clinical impression that patients with cardiac disease tolerate anesthesia and surgery well but reports are not lacking. The difficulty in gathering statistics lies in an inability to compare results in comparable groups of patients. Anesthesia is but a small part of the total operative experience. The patient with serious heart disease usually undergoes a relatively long period of preoperative preparation. When ready, he is given preanesthetic medication and is subjected to the administration of anesthetic. The surgical operation is superimposed on these preliminary events. Subsequently, convalescence with its many physiologic adjustments completes the surgical experience.
Anesthetic problems may originate in any of the aforementioned phases of the operative experience. These problems have been briefly reviewed. The most important factor in minimizing the problems of anesthesia itself is the skill and knowledge with which the anesthetic agents are chosen and administered rather than the actual pharmacologic and physiologic effects of the agents themselves. On a background of thorough medical preparation safe anesthesia for the patient with heart disease consists of the careful selection of preanesthetic medication, flawless technic, and use of minimal quantities of anesthetic agents and adjuncts. Despite the attainment of these goals, circulatory and respiratory problems will be encountered. These may be related to the heart disease itself, to medical preparation, pharmacologic side effects, and to a multitude of undesirable respiratory and circulatory reflexes. The latter in turn reside in anesthetic and surgical manipulation. Knowledge of these factors enables the internist to understand the problems of anesthesia and to prepare the patient for the operative experience.

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LEROY D. VANDAM and THOMAS K. BURNAP

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