The Anesthetic Management of Patients with Heart Disease

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In this era of direct investigation in man a number of measurements have been made of the hemodynamics of congestive heart failure, mitral stenosis, various types of congenital heart disease, hypertension and coronary artery disease. Cerebral, coronary, renal and hepatic blood flow have been measured, as have cardiac output and venous right auricular and pulmonary vascular pressures. Similar studies of the circulatory actions of anesthetic agents have been begun. Ideally a discussion of the anesthetic management of patients with heart disease should relate these two groups of data. Thus, for example, if a fundamental property of a particular drug were the production of pulmonary artery hypertension, this substance might be contraindicated when the pressure was elevated by disease. Unfortunately, only fragmentary analyses have been completed and it is not yet possible to be certain on the basis of physiologic data that one form of anesthesia is the best in a given situation.

This paper represents primarily the personal opinions of the authors. It is not to be regarded as a review of the literature. The circulatory effects of the more commonly used anesthetic agents and technics will be briefly outlined. Comments on our procedure for evaluation of patients will be presented. The management of certain groups of patients will be discussed in the light of our clinical experience. A final section will deal with the cause, prevention, therapy and significance of cardiac arrhythmias noted during anesthesia and operation.

I. Anesthetic Agents and Technics

1. Cyclopropane. This substance is an extremely useful anesthetic agent because of its potency, smoothness and rapidity of induction and emergence, and rapid controllability. Certain alterations in the circulation may follow its use. These must be understood and evaluated before cyclopropane can be assigned its proper place in anesthesia. The drug may cause any or all of the following changes:
   a. A rise in arterial pressure during anesthesia.
   b. A rise in central venous and right auricular pressure.
   c. A decreased arteriovenous oxygen difference.
   d. A decline in blood pressure below preoperative levels at the conclusion of anesthesia. When marked this response has been termed "cyclopropane shock."
   e. Abnormalities of cardiac rate and rhythm.

The factors responsible for these alterations are still not clearly defined. Certain speculations can be made, however. The first three items suggest that the rate of blood flow is more rapid under cyclopropane and that blood is shifted more promptly from the arterial to the venous side of the circulation. Arteriovenous shunts may be opened. Redistribution of blood may occur so that certain areas no longer receive as much of a supply of blood as they normally do. The rise in central venous pressure may be responsible for the rise in
cardiac output which has been reported by some workers. This in turn may be responsible for the rise in arterial blood pressure. Increase in central venous pressure has also been attributed to partial incompetence of the heart, a possibility which deserves further investigation. The clinical significance of these alterations of the circulation to the patient with cardiovascular disease is not at once apparent unless under exceptional circumstances the increase in blood pressure or cardiac output were such as to overstrain a damaged heart. Clinically, this must be a most unusual circumstance.

Postcyclopropane hypotension may pose a problem to the individual with low cardiac reserve, particularly to the patient with coronary insufficiency. At least one factor responsible for this postanesthetic decline in blood pressure is the respiratory acidosis which may accompany underventilation with any agent, be it cyclopropane, pentothal, curare or any other respiratory depressant. This syndrome is therefore largely preventable. It involves maintenance of adequate respiratory exchange during anesthesia. Clinicians have learned to minimize this disorder by maintaining a more normal respiratory exchange during anesthesia.

The abnormalities of cardiac rate and rhythm are of two types. The first are slow rhythms, caused by a downward displacement of the pacemaker and probably related to parasympathetic stimulation and increased vagal tone. These slow rhythms are of little clinical importance. The second are more rapid rhythms caused by increased irritability of the myocardium. There may be ventricular extrasystoles, ventricular tachycardia or, very rarely, ventricular fibrillation.

A great deal of experimental work has been reported on the underlying mechanism of this increased irritability. The ability of cyclopropane to produce increased myocardial irritability and abnormalities of rate and rhythm is not peculiar to this agent alone but is characteristic of a large group of hydrocarbons such as butane, gasoline, methane, carbon tetra-chloride, DDT, xylocaine and many others. The action of any one of these substances alone, however, is rarely sufficient to produce an evident arrhythmia. Apparently a trigger mechanism is necessary, the most easily demonstrable of which is that initiated by epinephrine.

Ventricular tachycardia and fibrillation can be produced by the injection of epinephrine into animals or man receiving cyclopropane anesthesia. If one avoids injecting epinephrine, the only other source of epinephrine would be endogenous. Presumably epinephrine could be mobilized by anoxia, pain, increased carbon dioxide and so forth. In this way a sufficient amount might be liberated to precipitate an arrhythmia in the presence of cyclopropane.

We grant that ventricular tachycardia may not be uncommon during cyclopropane anesthesia if this drug is used alone. An incidence of 10 per cent has been suggested by some. If, as is done by most clinicians, ethyl ether is added in small amounts, the incidence of ventricular tachycardia is significantly reduced. Furthermore, clinical experience indicates that ventricular tachycardia is relatively rarely followed by ventricular fibrillation on the operating table. Many instances of ventricular tachycardia are reported in the literature. Authentic reports of ventricular fibrillation occurring during cyclopropane anesthesia are extremely rare. Sudden circulatory collapse may occur during cyclopropane as with any other powerful depressant. Such a catastrophe cannot be assumed to be due to ventricular fibrillation unless an electrocardiogram was being taken at the time or unless the heart were visualized directly and seen to be in fibrillation. The most common cause for such a calamity is overdose of the anesthetic agent.

We have anesthetized many patients with serious cardiac disease with cyclopropane and small amounts of ether. It is our belief that the opponents of this agent have somewhat exaggerated the possibilities of harm following its use and have overlooked the advantages of smooth induction, relatively prompt recovery and controllability. Many of the circulatory alterations which have been discussed can be prevented, minimized or treated successfully. The mortality rate following cyclopropane is difficult to assess but in our clinic it does not appear to be excessive. The comparative incidence of postoperative circulatory complications is even more difficult to define in terms of
different anesthetic agents. Again our experience indicates that serious sequelae have been no more frequent with cyclopropane.

Since so many agents and technics are available to the modern anesthesiologist, it is probably wise to avoid use of this agent in the presence of thyrotoxicosis or other instances in which myocardial irritability is known to be increased.

2. Ethyl Ether. There are many characteristics which suggest the use of this anesthetic agent for patients with cardiovascular disease. There is a wide margin between the concentration of the drug required to produce respiratory failure and that required to produce circulatory failure. During anesthesia, fluctuations of blood pressure are much less common than with other anesthetics, other things being equal.

A great many misconceptions about ether have arisen because of the very safety of the agent which permitted individuals of varying skill to administer the drug without obvious harm to the patient. The unskilled administration of ether is followed by increased secretions in the respiratory tract, swallowing of ether-laden mucus, by anoxia and carbon dioxide retention. The latter are particularly likely to occur if excessive amounts of gauze are used on the mask or if the gauze becomes soaked with water vapor during an open drop anesthesia. We believe that one of the disadvantages of ether is the prolonged period of induction occasionally required. A hurried induction may be accompanied by cough, excessive secretions, respiratory obstruction and excitement. These sequelae, however, need not invariably occur and are less commonly seen when the drug is administered by capable personnel.

The work of Zweifach, Chambers and associates suggests that there may be a disintegration of the circulation during anesthesia with ether when an animal is subjected to hemorrhage. Compensation appears to be less adequate than with cyclopropane and a vasodepressor substance seems to be liberated. If this work is confirmed for man, there may be significant clinical implications. At the moment, however, ether stands as an exceptionally valuable anesthetic agent.

3. Nitrous Oxide. This relatively weak anesthetic agent cannot be administered by itself for major operations without the deliberate production of some degree of anoxia unless the patient is in shock or debilitated. The drug causes little postoperative disturbance of function and is therefore of great value when its potency is increased by the concomitant use of some other central nervous system depressant such as an opiate or barbiturate. If muscular relaxation is not required for an operation, such a combination may be extremely useful in that recovery from anesthesia is usually prompt and uneventful.

4. Pentothal. The outstanding advantage of this substance for the patient with cardiac disability is the smooth induction afforded. Excitement is absent and there is relatively little strain. If larger doses are used, however, and if the anesthetist attempts to obtain a deeper plane of anesthesia, tachycardia and hypotension are not infrequent.

It should be recalled that the barbituric acid derivatives are not analgesics. They do not block sensory pathways as readily as do most narcotics, so afferent impulses from the operative site can readily reach the cerebral cortex. In response to surgical manipulations, reflex spasm of the vocal cords with respiratory obstructions and anoxia, reflex spasm of abdominal muscles, or actual movement of the patient may occur.* In attempting to prevent or treat these reactions the anesthetist may believe it necessary to add more and more Pentothal until dangerous degrees of depression result. To combat this pharmacologic weakness, the analgesic action of nitrous oxide is often used in a complementary fashion, as has been described.

It should be recognized that Pentothal cannot produce muscular flaccidity with any degree of safety. If Pentothal or Pentothal and nitrous oxide are selected for operations in which muscular relaxation is required, the addition of a curare compound is often essential. This is not without hazard, although in the

*We do not believe that Pentothal "sensitizes" the larynx, but merely fails to depress its normal irritability.
last few years substances have been developed which block conduction at the nerve muscle junction without lowering blood pressure. Nonetheless, profound degrees of respiratory depression in the postoperative period result occasionally from any of these substances. In our opinion their indiscriminate use will be followed by a higher mortality and morbidity.

5. **Spinal Anesthesia.** Spinal anesthesia, which produces superb muscular relaxation and affords ideal working conditions for the surgeon during intra-abdominal operations, exerts a profound effect upon the circulation. Two major physiologic alterations occur. There is a decrease in peripheral resistance as the result of interruption of vasoconstrictor impulses to arterioles. There is also a pooling of blood on the venous side of the circulation probably secondary to postarteriolar dilatation. As a result of either or both of these factors, venous return to the heart is diminished and cardiac output declines. Undoubtedly coronary blood flow is diminished. If the coronary vessels are sclerotic and are unable to adjust to this diminution in flow, coronary insufficiency may result.

On the other hand, it should be pointed out that during spinal anesthesia the heart is required to do less work because of the diminution in peripheral resistance and output. Thus, despite a reduction in coronary artery flow caused by the drop in blood pressure, there may be adequate oxygenation of the myocardium at the lower blood pressure since the heart requires less oxygen. The supply is less but the demand has also been reduced. This point deserves further investigation since, if proved correct, it may extend the use of spinal anesthesia.

6. **Regional or Local Anesthesia.** Superficially it might appear that isolation of a discrete area of the body by block of individual nerves with local anesthetic agents would disturb the general circulation least of all. Pain relief during operation could thus be obtained for the seriously ill individual without strain from the anesthesia. This stand must be qualified, however, for the following reasons. Intra-abdominal manipulation during block of the abdominal wall may, for example, be attended by a reflex decrease in blood pressure and by such unpleasant subjective reactions as pain, nausea, retching and dyspnea. The emotional upset involved is significant and supplemental general anesthesia is often required. If hypotension, therefore, has resulted either from traction or from absorption of excessive amounts of the local anesthetic, one's purpose in sparing the patient has been defeated since one has to resort in addition to general anesthesia. Epinephrine is frequently added to solutions of local anesthetic drugs to prolong the action of the latter. Epinephrine is a powerful cardiac stimulant and the increased amount of cardiac work resulting from its administration may be beyond the capacity of the coronary arteries to supply blood. Anginal pain or more serious evidences of coronary artery insufficiency may result. Such reactions are often erroneously interpreted as being due to the anesthetic agent. It is our opinion that local anesthesia is useful for operations on the extremities and body surface. Where less superficial operations are involved supplementation with Pentothal and nitrous oxide or cyclopropane is indicated to minimize hypotension and subjective discomfort.

**II. Evaluation of the Patient**

The patient with cardiac disease who is facing anesthesia and operation should be evaluated by three individuals: cardiologist, anesthesiologist and surgeon.

The cardiologist is best qualified to take a complete history, to make an accurate physical examination, and to interpret laboratory data and the results of special tests of the cardiovascular system. He can establish the pathologic diagnosis and can determine the degree of functional disturbance in each patient. He can decide whether the heart is in the best possible condition or whether further bed rest, the use of diuretics, coronary vasodilating drugs or digitalis would improve the patient's chances for a successful surgical convalescence.

We do not believe that the cardiac consultant should suggest the type or method of anesthesia if he is associated with a specialist in this latter field of medicine. Few cardiologists accompany their patients to the operating room, watch the problems of induction and maintenance, realize
the difficulties posed by the body habitus of the patient, or appreciate the requirements of individual surgeons. Until they have practical, first hand experience with the conduct of anesthesia, it is unwise for them to insist upon a particular course. If a cardiologist is asked to evaluate a patient for operation in a hospital where technician-anesthetists are employed, it is equally unwise for him to do more than suggest a plan of management. It is undoubtedly safer for the patient to receive that anesthetic agent with which the technician is most familiar than to have him attempt a theoretically safer procedure in which he is less skilled.

The anesthetist approaches the patient from a different standpoint. He must convince the patient that he is aware of the problem at hand, and is capable of managing it successfully. The apprehension of such patients is understandably acute. They must be reassured. The anesthetist studies the body habitus. He is concerned over the short, thick necked individual who snores readily. During the unconsciousness associated with general anesthesia such a person will suffer from respiratory obstruction unless carefully observed and treated. The obese patient with a large abdomen experiences a reduction of lung volume in the supine position as the diaphragm is pushed up. In such a patient, furthermore, spinal anesthesia may rise rapidly and to excessive levels. Previous experience with anesthesia is reviewed, as is previous experience with such drugs as morphine, Demerol, scopolamine. The number of pillows required by the patient when sleeping is of interest. It may also be advisable for the anesthetist to test in advance the patient’s reaction to the position required for operation, for the circulatory abnormalities associated with the Trendelenburg or laterally flexed positions may be great.

The surgeon must assess the degree of muscular relaxation required, the estimated length of the procedure, the position desired. He must consider the necessity of limiting the extent of the operation because of the patient’s reduced reserve. Fluid and electrolyte balance must be evaluated, together with the hazard of circulatory overload during intravenous fluid therapy. He must realize and accept the fact that the orthopneic patient may fare well in certain positions on the operating table.

III. SPECIFIC DISEASE ENTITIES OR CONDITIONS

1. Pregnancy with Emphasis on Mitral Stenosis as a Complicating Factor. The circulation is considerably altered during pregnancy. At term cardiac output is increased by approximately 25 per cent, plasma volume by about 40 per cent. Of even greater interest is the effect of labor itself on the circulation. The variations in blood pressure are of greater magnitude than in any other clinical conditions which we have studied by intra-arterial pressure measurements. Pulse pressure may more than double within 10 seconds of the onset of a uterine contraction; a rise in systolic pressure of 60 to 80 mm. Hg is not uncommon. The least rise accompanies a uterine contraction which is not felt by the patient; a greater rise is noted during a contraction which the patient recognizes; the greatest hypertension follows a combination of uterine contractions and voluntary bearing down. The effect of this increased stress on the heart has obvious implications for the woman with a diminished cardiac reserve.

For two reasons we believe that spinal or caudal anesthesia may be useful for pregnant patients with cardiac disability, particularly if vaginal delivery is elected. The administration of any type of anesthesia tends to minimize the wide swings of blood pressure noted above but the smoothest curves have been achieved during spinal anesthesia. Under this method variations in blood pressure are reduced to alterations which cause little concern. This result should be the same during caudal anesthesia. The second reason involves the type of cardiac disease most frequently seen.

About 90 per cent of pregnant patients with cardiac disease have had rheumatic fever. The majority of valvular lesions are of the mitral valve. For those women who, during pregnancy, have experienced episodes of acute pulmonary edema often associated with hemoptysis, spinal or caudal anesthesia offers significant safeguards. Both methods produce a circulatory effect which might be termed a bloodless phle-
botomy. Vasomotor fibers to arterioles and venules are blocked in increasing numbers as the level of anesthesia rises. Venous pressure falls and blood is pooled in the periphery. This is of advantage to the patient with incipient pulmonary edema. Indeed, spinal anesthesia has been used in the treatment of pulmonary edema because of the circulatory adjustments produced.

The position of the patient is important. By definition the orthopedic patient cannot lie flat comfortably, and there are patients in whom pulmonary edema is precipitated by assumption of the supine position. It is strange how frequently this is overlooked in surgical practice. Particularly is this true when general anesthesia is administered and the patient cannot complain vocally of dyspnea. The semi-Fowler position with the head and chest elevated is essential for individuals with incipient heart failure. This is all the more important when cardiac weakness is combined with the elevated diaphragm and respiratory embarrassment of a full-term pregnancy.

For less seriously ill patients, nitrous oxide anesthesia together with various types of local block affords a useful combination.

2. Hypertension. Benign essential hypertension, almost regardless of the degree of elevation of blood pressure, causes relatively little concern to the anesthesiologist. If symptom free and particularly if the hypertension has persisted for years, these patients go through anesthesia and operation exceedingly well. It is only when the elevated blood pressure is associated with obvious damage to the heart, kidneys or cerebral circulation that such patients cause concern. Even under these circumstances it is surprising how well such patients withstand the stress of operation. Extensive sympathectomy and/or adrenalectomy have recently offered the anesthesiologist the opportunity of anesthetizing many seriously ill patients whose hypertension is complicated in a variety of ways. This experience has strengthened the belief that patients whose physical examination and laboratory data are discouraging indeed, may nevertheless pass through surgical procedures successfully.

Our original approach to patients whose hypertension was complicated by cardiac, cerebral or renal damage was based more on logic than on experimental evidence. It appeared reasonable to assume that if the hypertensive individual’s chief problem was that of previous congestive failure, one was justified in permitting mean arterial pressure to decline somewhat during anesthesia and operation rather than to attempt to maintain it at preoperative levels by excessive intravenous fluid therapy or use of vasopressor substances, both of which might overtax the weakened myocardium. If, on the other hand, the patient’s history suggested that the primary weakness was in cerebral or renal tissues, an effort was made to maintain an adequate blood pressure during operation, even if this involved a constant infusion of such a drug as, Neosynephrin, norepinephrine or the like. General anesthesia was used for all patients. Efforts were made to avoid excitement during the induction of anesthesia, although it was interesting that when arterial pressure measurements were made continuously even during relatively strenuous bouts of excitement, systolic blood pressures rarely rose more than 30 mm. Hg. Induction with sodium thiopental was not infrequently used in an attempt to put the patient to sleep as quietly as possible. Maintenance was then carried on, usually with ethyl ether. As long as the patient continued to be warm and dry, parenteral fluid therapy with glucose in water (rather than saline) or whole blood was continued cautiously. If the skin became cold, clammy or dusky, with peripheral vasoconstriction evident, the rate of infusion was increased significantly and a pressor drug whose primary action was central rather than peripheral was employed, for example, ephedrine. It has seemed logical to believe that if reflex peripheral constriction in response to hypotension were already maximal there was little indication for a drug whose primary site of action was peripheral. Perhaps this was fallacious.

The most recent change in attitude towards the anesthetic management of patients with hypertension (and actually other patients as well) is the concept of deliberately produced
and controlled hypotension. Though still unsettled, the arguments involved are of great interest to internists.

During the past several decades various individuals have suggested that a decrease in the blood pressure of a patient undergoing an operation may not be hazardous, but may even be useful, particularly from the viewpoint of minimizing blood loss and decreasing thereby the operative time. For example, neurosurgeons were not averse to permitting a decline in blood pressure during difficult craniotomies in order to obtain hemostasis.

Recently a number of workers have reopened the question. Gillies, in Scotland, has administered spinal anesthesia, avoiding the prophylactic injection of a pressor drug, and has attempted to produce a relatively bloodless operating field by a deliberate reduction in blood pressure. The metabolism of the patient is lowered by a minimal degree of general anesthesia. Oxygen is administered in concentrations above that in room air, and the patient is placed in such a position as to pool blood in the lower extremities. Goffen and colleagues, in this country, have used epidural anesthesia for the same purpose, as part of the anesthetic management of patients undergoing a fenestration. Gardner, Hale and others have developed a technic of controlled arterial bleeding, with provision for immediate reinfusion of the blood decreasing the hazard of the procedure. Drugs which depress the circulation directly have also been administered purposely during operation, hexamethonium bromide being the drug used most often.

Under what circumstances is such an approach justified? As already pointed out, one tends to worry much less about hypotension if the patient’s skin is warm, dry and of good color, if the pulse is full, of reasonable rate and if there is a reasonable pulse pressure. If the patient is conscious, the presence of mental acuity, the absence of restlessness, anxiety, air hunger, chest or arm pain taken together suggest adequate cerebral and coronary blood flow. These functions are more difficult to evaluate in the unconscious patient. One can readily admit to having observed patients whose absolute level of blood pressure has been as low as 70 mm. Hg systolic but who have appeared to be otherwise in good condition. One must also admit, however, that other patients with similar levels of blood pressure have experienced lesions of the brain, heart, liver or kidneys due either to an inadequate flow of blood through patent vessels, or to an actual occlusion of vessels by thrombosis or spasm. It is perhaps difficult to prove that the low blood pressure caused the complications, but the burden of proof is on those who deny the likelihood.

All of the factors which determine whether the body can tolerate hypotension are not known, but the following must be of significance:

a. The duration of the hypotension.
b. The degree of the hypotension.
c. The previous condition of blood vessels, particularly those supplying the heart, brain, kidneys and liver. If these vessels are sclerotic or otherwise abnormal, changes in their caliber in response to hypotension may not occur as readily as with normal vessels, and an insufficiency of blood may result.
d. The metabolic needs of the tissues during hypotension, again with particular emphasis on the heart and brain. The lower the metabolism, the greater the reduction in supply of blood which can be tolerated. Thus, the cellular depression associated with general anesthesia may be a relative safeguard during periods of low blood pressure, since the tissue requirements are presumably lowered at the same time. Likewise, if the hypotension be produced by spinal anesthesia, the heart may be better protected than one might imagine at first glance, because it is being called on to do less work against the lessened peripheral resistance. The coronary blood supply may be curtailed, but so may the demand for this blood.
e. The liberation of specific depressant substances from ischemic tissues. Shorr's demonstration that a vasodepressor material may be formed in the liver of shocked animals, or a liver subjected to poor blood flow, has widespread implications. This may only be the first of a group of toxic products so formed, the
impact of which on the body as a whole may be serious.

The successful use of deliberately produced arterial hypotension will depend entirely on whether tissue nutrition to the heart, brain and liver can be maintained during the period of low blood pressure. The kidney apparently will survive reasonable insults. The clinician electing this procedure must therefore be able to predict which patient will tolerate it. Most investigators will agree that young, healthy subjects will withstand reduced blood pressure satisfactorily. Most will agree that aged, sclerotic subjects with previous histories of coronary, cerebral or renal insufficiency will constitute poor candidates. Who, however, can safely select the patient midway between these two extremes?

3. Coronary Artery Disease. There is no single satisfactory approach to the anesthetic management of the patient with coronary artery disease, particularly if an intra-abdominal operation is planned. The following suggestions are recommended:

a. Assurance through personal contact that the anesthetist is aware of the patient's particular problem. This relieves a great source of anxiety on the part of the patient who frequently worries lest those concerned with his care be not completely familiar with his cardiac disability.

b. Adequate sedation prior to operation. The mental stress of a trip to the operating room is understandable and attacks of angina pectoris or coronary insufficiency may be precipitated by such an emotional crisis if proper sedation has not been achieved.

c. Smooth induction of anesthesia. A stormy induction with the likelihood of struggling, respiratory obstruction, anoxia, and retention of carbon dioxide is hazardous for a heart whose blood supply is marginal. Since this blood supply in all probability cannot be increased in the normal fashion by coronary artery dilation, further demand for blood secondary to increased cardiac work should be avoided.

d. Maintenance of blood pressure. A reduction in mean arterial pressure will reduce the amount of blood flowing through the coronary arteries. Although, during hypotension, cardiac work may be reduced, since the heart will be opposing less peripheral resistance, one cannot be certain that the demand for nutrients will be sufficiently lowered to be satisfied by the decreased supply.

e. Administration of oxygen. The value of this is self-evident.

Regional anesthesia is thought by some to be the safest form of pain relief for these patients. If an intra-abdominal operation is planned, we prefer general anesthesia with ether or cyclopropane to unsupplemented regional anesthesia for several reasons. Under local anesthesia the patient is conscious and aware of the sight, sounds and smells of an operating room. This is not calculated to provide mental calm unless large amounts of sedatives are provided. Furthermore, despite satisfactory anesthesia of the abdominal wall, intra-abdominal manipulations are often accompanied by severe pain, nausea, vomiting, dyspnea and a lowered blood pressure. These signs and symptoms are the result of traction reflexes and are noted as intraperitoneal structures are placed under tension with grasping forceps or retractors. The sequence of events is poorly understood, although the fall in blood pressure has been ascribed to a reflex constriction of the portal venous system with diminished venous return to the heart and a lowered cardiac output. The pain, frequently substernal, and the subjective feeling of shortness of breath have been attributed to a reflex reduction in coronary blood flow, although evidence on this point is not conclusive.

If there is virtue in urging the patient with coronary artery disease to live quietly, avoid stress and strain, and adjust his ways to a calmer existence, it seems unwise to subject this same individual to the mental and physical upset occasioned by an operation performed under local anesthesia, when a properly administered general anesthetic can abolish the mental disquiet and minimize the untoward effect of intra-abdominal traction.

4. Congestive Heart Failure. In our experience surgical emergencies have been rare in patients during acute episodes of congestive heart failure. Regional anesthesia has been employed in the few instances requiring immediate opera-
tive intervention. The patient has been kept with the head and thorax elevated. If oxygen therapy has been required prior to operation, this is continued during the surgical procedure. No parenteral fluid therapy is given. Venous tourniquets and a slow intravenous drip of theophylline ethylenediamine may be indicated in the presence of impending or actual pulmonary edema. Theoretically, the hemodynamic effect of spinal anesthesia suggests this method of pain relief in such patients. We have administered spinal anesthesia to patients with acute pulmonary edema as the result of sudden overloading of the circulation with intravenous fluid therapy. The results have been difficult to evaluate, however. If an elevated central venous pressure is needed to maintain cardiac output in certain patients, this reasoning may be incorrect.

5. Congenital Heart Disease. Because of the increase in the number of congenital abnormalities being studied by surgeons, a discussion of all of the problems involved in these patients would be too lengthy for this paper. Suffice it to say that most patients with a patent ductus arteriosus, tetralogy of Fallot, or coarctation of the aorta withstand anesthesia and operation exceptionally well. Since many of the surgical approaches to these diseases involve direct manipulations of the heart, we shall present some discussion of the utility of cardiac depressant drugs in the next section.

IV. CARDIAC ARRHYTHMIAS DURING ANESTHESIA AND OPERATION

Disorders of cardiac rate or rhythm are not infrequent in the anesthetized patient. There are certain circumstances under which one anticipates such abnormalities. Direct laryngoscopy and the introduction of a tube into the trachea is almost always followed by a rise in blood pressure and tachycardia. These changes may be marked if the depth of anesthesia is minimal. Manipulations near the hilum of the lung, stripping of an adherent pericardium, direct approaches to the heart chambers may all be accompanied by gross irregularities. Occasionally arrhythmias appear to occur “spontaneously.”

Some of the mechanisms responsible for such occurrences have been considered in the section on cyclopropane. Those incident to increased vagal activity (occasionally sufficient to cause cardiac standstill) are best treated by atropine administered in large doses intravenously. As a rule their clinical importance is less than disorders indicative of increased myocardial irritability. Under these conditions the anesthetic agent itself appears to have set the stage. The trigger may take the form of increased circulating epinephrine following direct injection, surgical manipulation or because of carbon dioxide accumulation or anoxia. Ectopic foci appear and gross disorders of rhythm result.

The prevention of these arrhythmias is based logically on the preceding information. Direct laryngoscopy and intubation of the trachea should be carried out under reasonably deep planes of anesthesia. Circulatory alterations are minimized under these conditions. Adequate ventilation throughout the period of induction and maintenance is essential. Not only must the hemoglobin be well oxygenated and the patient be of good color but the more insidious and the more difficult to recognize accumulation of carbon dioxide must be prevented. Recent work indicates that respiratory acidosis may be the common accompaniment of anesthesia for major operative procedures, both in the abdomen and in the chest. Since ventricular rhythms are increased in incidence and severity in the presence of carbon dioxide accumulation, prevention of this physiologic abnormality is the obvious answer. The injection of all sympathomimetic amines should be prohibited unless absolutely essential. If the need is great, neosynephrine or methoxamine should be used. These are not associated with the production of ventricular arrhythmias.

One then comes to a discussion of the use of drugs specifically designed to depress cardiac irritability by a direct action on the heart or intended to minimize cardiac abnormalities by blocking sympathetic inflow to the heart. The group of cardiac depressants most commonly used includes three substances: quinidine, procaine and procaine amide. All three of these can be given intravenously and two, quinidine and procaine amide, can be given by mouth.
Their pharmacologic action is intricate and complex.

As a generality, it is safe to say that the beneficial results following the action of any of these drugs depend upon a general cardiac depression. In this very action, of course, lies the possibility of harm. All three drugs can cause serious degrees of hypotension. All can produce flabby, dilated hearts if the dose is excessive and all can produce ectopic foci in the ventricles, the most severe and serious manifestation of which is ventricular fibrillation. They are undoubtedly of real use clinically but the dosage needed to provide protection and to avoid any of the toxic reactions is difficult to predict in advance. A great deal remains to be learned about their ultimate role.

The substances which block out or reduce the effect of sympathetic activity on the heart include ethyl ether and Dibenamine. As already indicated ether is almost always given with cyclopropane, small amounts of the former serving to produce the type of "functional decerebration" which appears to cut the heart off from impulses arising from the sympathetic center in the hypothalamus. Both ether and Dibenamine can be shown to reduce the incidence and severity of ventricular rhythms arising as a result of cyclopropane anesthesia. How useful they may be in minimizing other disorders of rhythm is less certain.

To illustrate the way in which an anesthesiologist faces the problem of arrhythmia, one can outline the regime employed when a multifocal ventricular tachycardia develops during anesthesia. Since these rhythms are more likely to occur under deep anesthesia, the anesthetic concentration is reduced. Since they may result from inadequate ventilation, every attempt is made to insure adequate oxygenation and removal of carbon dioxide. If ether had not been added, a small amount is usually administered. If none of these measures is successful, one of the cardiac depressant substances may be injected intravenously. Either quinidine lactate, procaine hydrochloride or procaine amide may be administered in doses of from 50 to 100 mg. per injection. Such an injection may be repeated several times. In the majority of instances these measures suffice. Less commonly, ventricular tachycardia may continue only to stop abruptly for no apparent reason. Clinical experience indicates that the great majority of such patients show no abnormality during anesthesia, operation or the postoperative period which indicates that their circulatory system had been seriously taxed. One dislikes seeing such abnormalities of rate and rhythm but one learns to live with them, in a sense, in the belief that ultimate damage will be minimal.

It is unfortunate that anesthesiologists do not know more about cardiology and that cardiologists do not have time to spend in the operating room. A great deal could be learned about cardiac arrhythmias, their mechanisms, significance, prevention and treatment if these two groups of medical specialists were to pool their information.

V. Conclusion

Teamwork between cardiologists, surgeons and anesthesiologists has increased the likelihood of survival of patients whose surgical disease is complicated by decreased cardiac reserve. Two main fields should be further explored if the reduction in mortality and morbidity is to continue. More must be learned of the basic actions of anesthetic agents and techniques on the circulation of man. Greater efforts should be made to collect and analyze data on the actual course of patients with heart disease anesthetized in different ways and subjected to different operations.