IT IS widely appreciated that the risk of death for cardiac patients undergoing major surgery is considerably less today than was the case 50 years ago. Greater surgical skill and improved cardiac management deserve much credit for this decrease in mortality. Of perhaps equal importance, however, have been the contributions from other areas. These include advances in anesthesia, the development of adequate blood bank facilities, improved understanding of fluid and electrolyte balance, and the effective use of chemotherapeutic agents. Finally, the fact that patients with clinical coronary artery disease can undergo major surgery with an additional mortality hazard of less than 4 per cent raises the value of attention to small details and of the institution of specific precautions to forestall catastrophe before, during, and following surgery.

The problem of cardiac risk during surgery has been a special pre-occupation of the medical service of the Beth Israel Hospital since the introduction of total thyroidectomy for the treatment of intractable forms of heart disease in 1930. This paper represents an attempt to develop from this accumulated experience of the past 30 years a working guide to the management of cardiac patients requiring major surgery.

The physician confronted with a cardiac patient needing surgical intervention must answer the following questions. Is organic heart disease actually present? What is the nature of the heart disease? What is the relation of the heart disease to the surgery? By what means before, during, and after surgery can the risk be reduced?

Problems in the Diagnosis of Organic Heart Disease

It is disconcerting to realize how many patients have been told they have heart disease that cannot be subsequently substantiated by complete cardiologic study. Among 631 patients referred to the Work Classification Unit at Bellevue Hospital in New York because of employment difficulties attributed to heart disease, 175 (28 per cent) were found on careful examination to have no recognizable cardiac pathology. In most instances the erroneous diagnosis of heart disease had been based on a faulty interpretation of symptoms or signs that had been present for some time.

Fatigue and dyspnea should be carefully evaluated before being accepted as evidence of congestive failure. These symptoms may be found in patients with pulmonary disease, with malnutrition, or with anxiety. Precordial pain need not be cardiac in origin, but may be caused by neuritis, arthritis, or by the "effort" syndrome. Palpitation frequently is unrelated to heart disease. Essential

From the Medical Research Department of the Yamina Research Laboratories, Beth Israel Hospital, and the Department of Medicine, Harvard Medical School, Boston, Massachusetts.

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hypertension uncomplicated by demonstrable cardiac involvement, pulmonary embolism, and latent syphilis may each lead to an erroneous diagnosis of heart disease. Arrhythmias per se are not necessarily indicative of myocardial pathology: sinus arrhythmia, bradyarrhythmia, premature contractions, paroxysmal atrial tachycardia, fibrillation, or bundle-branch block may occur in the absence of demonstrable organic heart disease. Many systolic murmurs may be transient and innocent. Faint murmurs at the apex or base that do not radiate widely or louder murmurs that disappear on expiration are usually of no significance. In the presence of marked anemia or thyrotoxicosis, grade-III systolic murmurs and even diastolic murmurs may also be on a functional basis. Similarly, disturbances in heart sounds, such as accentuation of the first sound at the apex, a split in the second sound at the pulmonic area, or a third heart sound may be of no clinical significance. Many additional examples could be given of errors in the interpretation of other signs as well as of laboratory aids such as the electrocardiogram or the chest roentgenogram. It is perhaps sufficient to state that care and judgment are required to determine that heart disease is actually present before one can assess the added risk it may bring to a surgical procedure.

Nature, Extent, and Significance of Heart Disease

Once a diagnosis of heart disease has been established, its importance from the viewpoint of surgery can best be appraised by classifying the cardiac defect according to the nomenclature of the New York Heart Association. Particularly in the older age groups the etiology frequently will be multiple. Perhaps the most significant aspect of the heart disease, insofar as it involves surgery, is the degree of incapacity suffered by the patient. This interpretation of incapacity involves not only an estimate of what the patient can do, but also what the patient ought to do.

The relation of the heart disease itself to the contemplated surgery is too often overlooked. Occasionally the surgical problem concerns the cardiac pathology itself. Constrictive pericarditis, atrial septal defect, and mitral stenosis are common examples. In the presence of constrictive pericarditis, congestive heart failure becomes an indication rather than a contraindication for surgery. Not infrequently heart disease may masquerade as a "surgical" condition. Thus, acute myocardial infarction may simulate acute cholecystitis or peptic ulcer. Such possibilities must be seriously considered and excluded before undertaking any surgical procedure. The converse is also true, and many patients are denied necessary surgery for an acute abdominal catastrophe because of an erroneous diagnosis of acute myocardial infarction.

Relation of Congestive Heart Failure and Cardiac Pain to Cardiac Risk

After it has been determined that organic heart disease does exist, and after its nature, extent, functional significance, and relation to the surgical problem have been defined, it is possible to evaluate adequately the additional risk to the cardiac patient for whom surgery is contemplated. In 1928, Marvin stated, "... for purposes of anesthesia and operation, a heart that is damaged but that is carrying on an adequate circulation under normal conditions of life is the equivalent of a normal heart." This statement, made over a quarter of a century ago, is still largely true today with the following notable exceptions: patients with angina pectoris or myocardial infarction, patients with aortic stenosis or regurgitation, and patients with atrioventricular block. These exceptions must be recognized because, while they may permit an adequate circulation under normal conditions, they may also lead to sudden death. The existence of a considerably increased risk must, therefore, be recognized in these patients even though none of the usual contraindications is present.

Other than these situations, the preoperative reduction of cardiac risk resolves itself,
MAJOR SURGERY IN THE CARDIAC PATIENT

from a practical viewpoint, into the appropriate treatment of congestive heart failure and the proper evaluation of cardiac pain.

Congestive Heart Failure

Preoperative treatment may be strikingly successful in converting even those patients presenting with gross congestive failure into less formidable risks. In addition to the underlying cardiac lesion, all factors that may have precipitated or aggravated the heart failure must be considered. These factors act by increasing the disparity between myocardial blood supply and demand. Some of these factors may be intracardiac, others extracardiac; several, moreover, may be functional in that they are not caused by organic disease (table 1). Recognition of these entities is particularly important, since several of them such as arrhythmias, anemia, and thyrotoxicosis are entirely reversible with appropriate therapy before surgery.

Angina Pectoris and Myocardial Infarction

In the evaluation of cardiac pain, the presence of stable angina pectoris per se is no deterrent to necessary surgery. The recent onset or recent aggravation of pre-existing angina pectoris, however, almost invariably indicates some change or imbalance in the coronary circulation. Under such circumstances the stress of operation may precipitate myocardial infarction. The recent onset or increase in angina may itself be the pro-drome of an episode of acute myocardial infarction. A recent myocardial infarction is a clear contraindication to surgery. As a general rule, elective procedures should not be undertaken until 3 to 6 months have elapsed after an infarction. Although the acute inflammation subsides by the third or fourth week, when the patient is usually ambulated, firm scar tissue is not present until the third or fourth month. Necrotic muscle and the outpouring of polymorphonuclear leukocytes are at their height during the first week (fig. 1). Although evidence of repair, manifested by the proliferation of pigment cells, plasma cells, lymphocytes, the removal of necrotic tissue, and the ingrowth of blood vessels and

Table 1

Precipitating and Aggravating Factors in Congestive Heart Failure

<table>
<thead>
<tr>
<th>Intracardiac</th>
<th>Extracardiac</th>
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<tbody>
<tr>
<td>Acute myocardial infarction</td>
<td>Pulmonary embolism</td>
</tr>
<tr>
<td>Acute rheumatic fever</td>
<td>Infection</td>
</tr>
<tr>
<td>Bacterial endocarditis</td>
<td>Asthma</td>
</tr>
<tr>
<td>Tachycardia*</td>
<td>Thyrotoxicosis</td>
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<tr>
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<td>Obesity</td>
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<td>Pregnancy</td>
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<td>Trauma</td>
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<td>Anemia</td>
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<tr>
<td></td>
<td>Exertion*</td>
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<tr>
<td></td>
<td>Emotion*</td>
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<tr>
<td></td>
<td>Malnutrition</td>
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<tr>
<td></td>
<td>Anoxia</td>
</tr>
<tr>
<td></td>
<td>Cessation of cardiac therapy</td>
</tr>
</tbody>
</table>

*Functional

new connective tissue, occurs during the second, third, and fourth weeks after the onset of infarction, these processes are still going on for many weeks after the initial insult. If necrotic material and active healing are still present, foci of irritability remain that may set off a fatal arrhythmia or precipitate fresh necrosis under the stress of a surgical procedure. These facts offer the best anatomic justification for prolonged bedrest during the first 3 to 4 weeks after an acute myocardial infarction and for delay in surgery until a firm scar has developed.

Other data, obtained from a study of cardiac rupture, have shown that an acute myocardial infarction continues to change in extent for some time after its inception. Although the clinical picture indicates the onset of a myocardial infarction at a particular time, the entire infarct is not all of that same age. Rather, infarction is a continuous and progressive process with the simultaneous repair of old areas, the development of fresh necrosis, and the recovery of ischemic muscle.

Surgery and the Prognosis of Heart Disease

One of the problems that frequently arises in deciding on surgery in cardiac patients is the estimation of life expectancy with regard to the cardiac lesion itself. While it is reasonable to treat uterine prolapse or an abdominal

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hernia by nonsurgical means in many cardiac patients, necessary surgery should never be denied because of a poor statistical prognosis of the heart disease itself. Although, in general, the average duration of life among patients with angina pectoris is from 2 to 8 years in different series, some individuals survive 15 or 20 years after the onset of cardiac pain. Similarly, patients may live many years after the onset of congestive heart failure.

The mortality curves of patients with angina pectoris and congestive heart failure admitted to the Beth Israel Hospital have demonstrated that the statistical prognosis is particularly somber in the early course, but less so in the later years of survival. The difficulty that confronts the physician is that he cannot predict early in the course of angina pectoris or congestive heart failure which patient will survive for long periods of time. Consequently, angina pectoris and congestive heart failure are not adequate reasons, in themselves, for denying patients necessary surgery.

The Urgency of Surgery

The degree of cardiac risk accepted by a physician is often dependent on the urgency of the surgery under consideration. On the basis of our experience, elective surgery has been recommended for cardiac patients without angina pectoris, myocardial infarction, congestive heart failure, aortic regurgitation, aortic stenosis, atrioventricular block, or other serious arrhythmias (table 2). Cardiac enlargement, hypertension, or organic defor-
withies of the heart valves are not in themselves contraindications to elective surgery. For urgent surgery, such as carcinoma or repeated attacks of biliary colic, attempts should be made to control congestive failure, to stabilize angina pectoris, and, if possible, to delay surgery for weeks to months after acute myocardial infarction. In patients with aortic valve disease and arrhythmias, decisions concerning the time and extent of surgery must always be individualized. There are, of course, no absolute contraindications when lifesaving surgery is indicated such as in mesenteric thrombosis.

Cardiac Complications and Their Causes

The cardiac patient rarely, if ever, gets into difficulties from the surgery itself, but rather because of the complications attending the operative procedure. The four major cardiovascular catastrophes that may result from surgery are congestive failure, acute myocardial infarction, cardiac arrhythmias, and thromboembolism. In some instances latent rheumatic fever may become activated. The two commonest causes of these major cardiac disasters during or immediately following surgery are hypotension and anoxia. Usually they result from anesthetic difficulties or blood loss; each may aggravate the other, and either may produce myocardial ischemia. Hypotension can be prevented or promptly treated by adequate blood replacement at the time of blood loss, the avoidance of anoxia, the maintenance of an adequate blood volume by intravenous fluids, and the prompt use of appropriate sympathomimetic amines if the blood pressure begins to fall. Anoxia can be prevented by maintenance of a patent airway, adequate oxygenation, and an effective arterial pressure.

There are other less frequent complications that may also precipitate cardiac embarrassment. Fever and excitement increase cardiac work; dehydration may predispose to the development of thromboembolic phenomena; hypervolemia can cause pulmonary edema; hypoglycemia can precipitate myocardial infarction in patients with coronary artery obstruction; abdominal distention may interfere with venous return; and malnutrition may cause hypoproteinemia and vitamin B<sub>1</sub> deficiency, thereby precipitating or aggravating congestive failure.

Specific Measures That Reduce Cardiac Risk

Heart disease is usually a chronic affliction, and cardiac patients frequently have coexisting noncardiac pathology. Failure to recognize renal, hepatic, or pulmonary insufficiency, a bleeding diathesis, hypothyroidism, or latent diabetes may place additional burdens on the already compromised heart and thereby negate the efficacy of a surgical procedure. In addition to careful cardiovascular study, a comprehensive clinical evaluation is essential for the recognition of other diseases that may further embarrass the heart during surgery. There are, moreover, specific measures that may significantly reduce the hazards of surgery.

Drugs Prior to Surgery

In view of the increasingly wide variety of potent chemical agents now available for the treatment of disease, it is important that the administration of any such medications be known to the physician prior to surgery. Some patients may be using drugs regularly and not volunteer this information to the doctor. Many of these agents, such as rauwolfia and chlorpromazine, may potentiate the effect of preoperative medication. Failure to appreciate the significance of anti-epileptic

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**Table 2**

<table>
<thead>
<tr>
<th>Cardiac status</th>
<th>Type of surgery</th>
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<tbody>
<tr>
<td>Congestive failure</td>
<td>Elective * Controlled 0†</td>
</tr>
<tr>
<td>Angina pectoris</td>
<td>Stable 0</td>
</tr>
<tr>
<td>Recent infarct</td>
<td>3-6 months 0</td>
</tr>
<tr>
<td>Aortic valve disease</td>
<td>± 0</td>
</tr>
<tr>
<td>Atrioventricular block</td>
<td>± 0</td>
</tr>
<tr>
<td>Arrhythmia</td>
<td>±†</td>
</tr>
</tbody>
</table>

* Surgery contraindicated.
† No contraindication to surgery.
‡ Recommendation for surgery based on specific findings in individual patient.
medication, thyroid, pilocarpine, quinidine, digitalis, antihistamines, hypotensive or hypoglycemic agents, anticoagulants, or steroids may complicate or lead to the fatal outcome of an otherwise benign surgical procedure.

The problem of steroid therapy warrants special emphasis. Patients receiving corticosteroids must be given even greater amounts of these drugs during and following surgery than is generally recognized. Patients who have received steroids in the past may, after an interval of even 6 to 12 months, develop hypotension while undergoing surgery if corticosteroids are not administered during the operative procedure. If minimal amounts were given 4 to 6 months previously, little or no medication may be necessary. In contrast, patients who have received doses large enough to produce hypercorticism, may have continued suppression of the pituitary-adrenal axis for as long as a year following the cessation of therapy and may develop circulatory collapse on being subjected to the stress of surgery. Such patients, therefore, should receive intramuscular cortisone, 200 mg. daily beginning 48 hours before and on the morning of surgery and this drug should be continued postoperatively in decreasing amounts for 5 to 8 days. Hydrocortisone succinate should be available for intravenous use should hypotension occur during surgery. In the immediate postoperative period, hourly blood pressure measurements should be recorded to guard against cardiovascular collapse in these patients. It must also be recognized that patients receiving corticosteroids are very sensitive to morphine, so that it should be used cautiously, if at all.

Preoperative Medication

Every patient should be interrogated to determine whether or not he has ever had any idiosyncrasy to previous medication. Barbiturate excitement, for instance, may precipitate congestive failure in a susceptible individual. It is important to inquire carefully into any unusual reactions that the patient may have experienced in the past, especially from any of the medications contemplated before and after surgery. Moreover, noncardiac conditions such as emphysema, cirrhosis, or myxedema often require modifications in the preoperative use of barbiturates and opiates. Although this advice is more often honored in the breach, many untoward reactions would be avoided if all drugs that might be used while the patient was in the hospital were tested before operation, particularly if they had never been administered previously to the patient.

There may be unpredictable delays in the absorption of a preoperative sedative, and a large amount of such a drug given the evening prior to or even several hours before surgery may suddenly become effective during anesthesia, thereby producing undue depression. It is thus advisable to use light sedation and to administer immediately before surgery an adequate amount of morphine or meperidine to produce the desired degree of relaxation.

The digitalis glucosides are indicated preoperatively in the treatment of congestive heart failure, atrial fibrillation, and in some patients with a history of paroxysmal atrial tachycardia or atrial flutter. Somewhat more digitalis should be given to patients with atrial fibrillation or flutter than the minimal amount necessary to control their ventricular rates at rest. A given stress, such as exercise, causes a much greater rise in ventricular rate in patients with atrial fibrillation than in those with normal sinus rhythm. This disproportionate rise can be greatly reduced by full digitalization leading to a slightly lower ventricular rate of approximately 70 at rest. The use of digitalis cannot be justified solely on the basis of the patient’s age, blood pressure readings, or the mere presence of organic heart disease.

In patients with frequent angina pectoris, who are about to undergo surgery, the use of sublingual nitroglycerin prophylactically, 20 to 30 minutes before surgery, may be helpful. Atropine is useful in lessening bronchial and upper respiratory secretions. It is not always appreciated, however, that, through its inhibitory effects on the vagus, an undue
increase in ventricular rate, especially in patients with atrial fibrillation, may result. It is desirable to anticipate this by the administration of test doses of atropine prior to surgery.

Reassurance

Anxiety may increase cardiac work. It is important to recognize that the majority of cardiac patients have a special apprehension toward impending surgical procedures. Such anxiety may frequently be reduced if the patient’s own physician visits the patient in his room prior to the administration of the preoperative medication. Further reassurance can be provided if the physician can tell the patient that he will also be present at the operation.

Anesthesia

The choice of an anesthetic agent and of anesthetic technic for the cardiac patient should be considered carefully. “Ideal” anesthesia is an illusion, and attempts to establish fixed rules for the selection of anesthetic agents lead only to disaster. The selection of a program of anesthetic management should be based on the cardiac status as delineated by the cardiologist, the needs of the surgeon, the skill and experience of the anesthesiologist, and, wherein possible, the wishes of the patient. The following considerations are, however, helpful in reaching wise decisions in this area.

When feasible, local or regional field anesthesia should be considered because it causes the least increase in cardiac work. Aside from the possibility of vasomotor collapse or convulsions, particularly with cocaine and its derivatives, local anesthesia is associated with the least risk, if anxiety, discomfort, and pain can be avoided. The possibility of convulsions can be minimized by premedication with barbiturates. It is particularly important that no epinephrine be included in the local anesthetic mixture. Local anesthetic drugs are frequently packaged by pharmaceutical firms with epinephrine to provide a bloodless operative field through local vasoconstriction. It is the obligation of the physician to insist that, in cardiac patients, the local anesthetic be free of such sympathomimetic agents which, if absorbed systemically, can cause sudden death through cardiac arrhythmias in patients with pre-existing coronary disease.

Spinal anesthesia has certain advantages in patients with congestive failure from valvular disease. Under such anesthesia orthopnea usually disappears. The attendant risk of hypotension in patients with diastolic hypertension and coronary artery disease, however, is significant though it can be prevented and treated by the intramuscular injection of vasopressor substances.

General anesthesia should not be undertaken without providing for an unobstructed airway. An endotracheal technic in certain types of surgery provides many safeguards for the cardiac patient. Chloroform is a direct myocardial toxin, may produce fatal arrhythmias, and should no longer be used. Cyclopropane provides smooth, rapid induction, a pleasant recovery, and good relaxation, if it is supplemented, when necessary, by a muscle relaxant. Epinephrine is contraindicated at any time during the use of cyclopropane. The principal hazard of cyclopropane is the production of arrhythmias. These may possibly be diminished by the addition of a trace of ether to the cyclopropane anesthesia. Arrhythmias from cyclopropane are particularly prone to occur during induction. Cyclopropane anesthesia, therefore, may be initiated with thiopental sodium (Pentothal). In general, cyclopropane should be avoided in cardiac patients prone to arrhythmias and in patients with thyrotoxicosis.

Although Pentothal may depress the rate and depth of respiration and predispose to laryngospasm, it is an excellent drug for cardiac patients, if hypoxia is avoided and if relaxation is not required, as in the reduction of fractures or the incision and drainage of an abscess. Ethyl ether is an effective drug for patients with cardiovascular disease except when extreme relaxation is needed. Fluctuations in blood pressure are minimal at light and intermediate levels of anesthesia. To avoid hypotension from deep levels of
anesthesia, a light plane of ether anesthesia together with a muscle relaxant provides a good means of obtaining excellent relaxation.

Nitrous oxide alone cannot produce the desired amount of relaxation, particularly for abdominal surgery, without undue lowering of the oxygen content of the gas mixture. It is only safe and effective when combined with Pentothal and a relaxant such as succinylcholine. If muscle relaxants are used, adequate ventilation must be carefully maintained. This combination of anesthetic agents can be highly recommended for cardiac patients.

In practice, the experienced anesthesiologist by minute-to-minute observation and care can provide effective and safe anesthesia for the patient with heart disease. He can do this by inducing anesthesia quietly and without struggle, by maintaining anesthesia evenly and with adequate pulmonary ventilation, and by using anesthetic drugs in quantities that produce minimal changes in the patient and still provide adequate surgical exposure.

Cardiac Arrhythmias during Surgery

The physician should be prepared for the development of various arrhythmias during surgery. Some of these are of short duration and require no therapy. Many are directly related to hypotension or anoxia and, although responsive to pressor amines, are corrected only by eliminating the cause of the disturbance in pathologic physiology. If serious arrhythmias persist despite maintenance of arterial pressure and of oxygen saturation, drugs may be helpful in reestablishing a normal sinus rhythm. Supraventricular tachycardia and atrial flutter and fibrillation may be treated by carotid sinus pressure, intravenous lanatoside C (if the patient is not already receiving digitalis), or intravenous procaine amide (Pronestyl). Frequent ventricular beats or ventricular tachycardia may be eliminated by intravenous Pronestyl, beginning with a rate of 50 mg. per minute and gradually increasing the dose under direct electrocardiographic monitoring.

Cardiac arrest leading to death is one of the major accidents occurring particularly in patients with aortic stenosis, conduction defects, and coronary artery disease. Cardiac standstill is the usual mechanism of cardiac arrest during anesthesia. Ventricular fibrillation may occasionally be responsible. Preventive measures include the administration of atropine and the avoidance of overdigitalization, as well as the prevention of anoxia and hypotension. Constant observation of the heart by the anesthesiologist, using an oscilloscope or another monitoring device, is imperative in order to institute therapy immediately. Pounding the chest wall and needle puncture of the heart to stimulate cardiac action are indicated. The use of external stimulation of the heart through electrodes placed on the chest wall has been shown to be effective in the treatment of ventricular standstill. Impulses at 60 per minute and 3 milliseconds in duration are applied at 50 to 150 volts. All cardiac patients should have a cardiac monitor and an external electric pacemaker applied prior to the induction of anesthesia. Appropriate precautions must be taken to safeguard the patients from the hazards of electrical apparatus during anesthesia. If the patient has been monitored by oscillographic control and the mechanism is seen to be ventricular fibrillation, external counter-shock with an A.C. current at 250 volts or more for 0.15 second is effective. The heart either resumes normal beating or can be aroused from standstill by external electric stimulation. Most recently a simple method of applying pressure to the unopened chest for maintaining circulation until the cardiac beat is restored has been recommended.\(^{18}\) If none of these measures is effective within 60 seconds of arrest, thoracotomy and manual massage of the heart are indicated. To salvage patients who develop cardiac arrest in the operating room, it is necessary that a prophylactic and therapeutic program be available in advance of the accident. Details of such a program have been published by Zoll and his associates.\(^{16,17}\)

Postoperative Drugs

Several drugs frequently used postoperatively may increase cardiac risk. Intestinal
distention occurs following numerous surgical procedures. In cardiac patients this complication may be treated by enemas or decompresing procedures but never by agents such as pitressin. This drug is one of the most potent coronary artery vasoconstricting agents and may cause severe cardiac pain, myocardial infarction, arrhythmias, and sudden death. In addition, oliguria may ensue. Ergot is frequently used to stimulate uterine contractions. It produces the same undesirable side effects as pitressin in patients with coronary artery disease and should certainly be avoided in all diabetic women. Carbon dioxide mixtures are still used empirically for the treatment of intractable hiccup. In concentrations above 5 per cent, cardiac output, rate, and blood pressure are increased and a cardiac catastrophe may be precipitated. It is desirable, therefore, to use carbon dioxide mixtures, if at all, only for short periods of time such as 2 to 3 minutes at 10-minute intervals. Insulin, if given in excess, may produce hypoglycemia: in patients with pre-existing coronary disease, arrhythmias, congestive heart failure, or acute myocardial infarction may result. Therefore, in the treatment of the postoperative diabetic patient with insulin, it is usually desirable to permit a modest glycosuria. The goal of insulin therapy in the immediate postoperative period should be the prevention of acidosis rather than the elimination of hyperglycemia. Epinephrine is frequently used in the treatment of peripheral vascular collapse. This agent may produce severe cardiac pain, arrhythmias, and occasionally shock and death. There are other sympathomimetic amines such as phenylephrine (Neoepinephrine), norepinephrine, and metaraminol (Aramine) that are better suited to the treatment of hypotension because they have less marked cerebral and less adverse cardiovascular effects.

**Fluid and Electrolyte Balance**

In cardiac patients excessive postoperative losses of fluid should be replaced to prevent electrolyte and acid-base imbalance which, if uncorrected, may lead to cardiac arrhythmias, digitalis toxicity, and impaired myocardial metabolism. If renal function is good, adequate postoperative hydration can be maintained by the administration of enough fluid to equal the volume of water lost plus the volume of urine required to prevent nitrogen retention. This goal can be accomplished by the administration of sufficient fluid to produce 1 liter of urine per 24 hours with the use of 5 per cent glucose in distilled water intravenously and by the resumption of oral intake as soon as possible. Large amounts of sodium-containing fluids may lead to congestive failure or pulmonary edema, even though these findings have been absent in the past. In patients with anemia, packed red cells may be preferable to whole blood. From studies of the influence of the rate of administration of intravenous fluids on cardiovascular dynamics in normal subjects, it would appear that 15 ml. of fluids per minute represent a rate of fluid replacement in cardiac patients that should not produce pulmonary edema, unless salt is included.

Some cardiac patients, despite a normal blood urea nitrogen and an adequate preoperative urinary output, may have subclinical renal insufficiency. This state may be so aggravated by anesthesia, blood loss, dehydration, electrolyte imbalance, and congestive heart failure that a significant postoperative reduction in glomerular filtration rate may result. The possibility of such an alteration restricts the margin of safety ordinarily provided by the limited amounts of water recommended above.

**Infection**

Postoperative infections are particularly undesirable in cardiac patients because fever increases cardiac work and because bacterial foci may lead to the development of endocarditis, myocarditis, or pericarditis. Unfortunately, routine prophylactic chemotherapy is not effective in preventing infectious complications, and may, in itself, lead to superinfection with resistant organisms, disturbances in bowel function, and other evidences of drug intoxication. In patients with established vascular disease or other cardiovascular abnormalities predisposing to endocarditis, prophy-
Cardiac Pathology in an Unselected Series of Injected Hearts* (1936-1945)

<table>
<thead>
<tr>
<th>Age group</th>
<th>Normal M</th>
<th>Normal F</th>
<th>Myocardial hypertrophy</th>
<th>M 451-650 Gm.</th>
<th>F 451-650 Gm.</th>
<th>Valvular deformity</th>
<th>M 451+ Gm.</th>
<th>F 451+ Gm.</th>
<th>Coronary artery narrowing:</th>
<th>M</th>
<th>F</th>
<th>M</th>
<th>F</th>
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<td>27</td>
<td>12</td>
<td>12</td>
<td>10</td>
<td>10</td>
<td>10</td>
<td>10</td>
<td>10</td>
<td>17 (253)</td>
</tr>
<tr>
<td>61-70</td>
<td>15 M</td>
<td>19 F</td>
<td>21</td>
<td>12</td>
<td>6</td>
<td>3</td>
<td>9</td>
<td>4</td>
<td>23</td>
<td>21</td>
<td>24</td>
<td>13</td>
<td>15</td>
<td>7</td>
<td>1</td>
<td>1</td>
<td>1</td>
<td>299 (112F)</td>
</tr>
<tr>
<td>71+</td>
<td>7 M</td>
<td>5 F</td>
<td>8</td>
<td>2</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
<td>15</td>
<td>14</td>
<td>21</td>
<td>15</td>
<td>12</td>
<td>10</td>
<td>1</td>
<td>1</td>
<td>1</td>
<td>48 (191)</td>
</tr>
</tbody>
</table>

*Hearts reported as showing coronary obstruction may also have had valvular deformity or myocardial hypertrophy. The reverse was not true.

1M, male; F, female.

2+5 per cent reduction in the cross-sectional diameter; 2+, 50 per cent; 3+, 75 per cent.

§Refers to total occlusion of one or more coronary arteries.
patients is sufficiently great that it cannot be recommended as a routine operative or post-operative procedure even for cardiac patients in whom the likelihood of thrombosis is real. This is not to deny that in the future, anticoagulation may become the rule rather than the exception in the postoperative care of the patient with heart disease.

If anticoagulant therapy is indicated postoperatively, heparin is the drug of choice and intravenous administration the preferred parenteral route. If anticoagulants are required within 72 hours of surgery, heparin may be given every 2 hours by intermittent intravenous injections in amounts that double the clotting time at 2 hours; after that time interval the drug may be given on a 4 hourly schedule. Vein ligation should be limited to the treatment of septic phlebitis and to the management of nonseptic phlebitis when anticoagulants are contraindicated or have failed.

Frequent deep breathing, coughing, and turning in bed minimize the hazards of pulmonary atelectasis. Whether such activity as well as movements of the extremities and early postoperative ambulation prevent thromboembolism has not been established. If such activity causes no harm to the patient, it is to be recommended because it diminishes vascular stasis—one of the factors known to predispose to thrombosis. Bandaging of the leg postoperatively with well-fitted elastic stockings, that do not act as tourniquets nor produce ulceration of the heel, can be recommended as a safe procedure in cardiac patients with an adequate peripheral arterial circulation. Such bandaging increases the rate of flow in the deep veins of the extremities. Whether it actually decreases the incidence of phlebitis has not been established conclusively.

The Distinction between the Cardiac and Noncardiac Patient

As the risk of surgery for the cardiac patient approaches that for the noncardiac, renewed interest will center about the causes of death attending surgery in individuals free of clinical heart disease, particularly over the age of 40. That deaths from heart disease play a significant role among such patients cannot be denied. Necropsy data have revealed a high incidence of coronary artery obstruction among patients without clinical heart disease. In table 3, classified by age and sex, is the cardiac patholology observed among 1,011 unselected hearts examined by a special injection and dissection technique at the Beth Israel Hospital from 1936 to 1945. In the fifth decade alone 60 per cent of male hearts exhibited coronary artery obstruction. Among all hearts from patients over the age of 40, less than one fifth were entirely normal. The majority of these patients died of noncardiac causes and many had no symptoms, signs, or laboratory evidence of heart disease.

The implications of these data take on even broader significance with the realization that under the age of 40 and in patients with morphologically normal hearts, an occasional unexpected death may be cardiac in origin. Sudden cardiac arrest on induction of anesthesia for a diagnostic uterine curettage in an otherwise healthy 22-year-old woman may serve as an example. Since these cardiac difficulties cannot be predicted, the ultimate reduction of cardiovascular deaths in general surgery will be realized only when the precautions recommended for cardiac patients are applied to all individuals subjected to surgery.

References

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“We have good reason,” he writes, “to be diligent in making farther and farther researches; for tho’ we can never hope to come to the bottom and first principles of things, yet in so inexhaustible a subject, where every smallest part of this wonderful fabric is wrought in the most curious and beautiful manner, we need not doubt of having our inquiries rewarded with some further pleasing discovery; but if this should not be the reward of our diligence, we are however sure of entertaining our minds after the most agreeable manner, by seeing in everything, with surprising delight, such plain signatures of the wonderful hand of the divine Architect, as must necessarily dispose and carry our thoughts to an act of adoration, the best and noblest employment and entertainment of the mind.”—Stephen Hales, B.D., F.R.S. Vegetable Statics, 1727.
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STANFORD WESSLER and HERRMAN L. BLUMGART

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