THE CONTROL OF CARDIAC ARRHYTHMIA DURING SURGERY * †

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ANESTHESIOLOGISTS have almost completely mastered the control of respiration; they have improved the technic of blood replacement but control of the heart itself has not been attained. The same control that has been obtained over the lung will not be extended to the more fickle heart for some time. That control will, no doubt, first be extended with drugs, then taken over completely by the extracorporeal pump during periods of intracardiac operation and various cardiac crises.

These are ambitious dreams and they will be realized, but it is necessary to plot our *present* position. This is extremely difficult not only because of the quantity of information that is available but especially because it is necessary first to separate the paucity of fact from the plethora of opinion. There is an endless gamut of fancy, presumption, and half-truth opposed by only a few pertinent and salient facts.

The first bearing one must take in order to correct cardiac dysrhythmia is to determine its exact nature: is it simple extrasystole, a wandering pacemaker, ventricular tachycardia, auricular or ventricular fibrillation or an actual cardiac arrest? Any of these irregularities may occur. Distinction is often impossible by simple auscultation or even by direct observation; electrocardiographic tracings may be required. To apply what appears today to be rational therapy, the abnormal rhythm must be properly identified.

The second bearing consists of identifying the cause of the arrhythmia. Some factors that can be responsible for abnormal cardiac rhythm are:

I. Reflex mechanisms initiated at points away from the heart (not peculiar to cardiac surgery) such as:
   A. Position of the patient

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B. Intratracheal, intrabronchial or intrapulmonic stimulation (1, 2) as may be associated with
   1. Intubation
   2. Aspiration of secretions
   3. Rapid lung inflation
   4. Prolonged positive pressure
C. Chest wall manipulation, especially rapid rib spreading
D. Sudden alterations in blood volume

II. Chemical changes in the blood stream, particularly
A. Hypoxia
B. Toxicity or sensitivity to drugs, including anesthetic agents (3)

III. Direct cardiac and intracardiac manipulation producing
A. Pericardial, epicardial or myocardial stimulation
B. Dislocation of the heart from its position of optimum, function (2, 4).

The existence of intrinsic heart disease may lower the threshold of reaction to any of these. An abnormal heart may not only be more susceptible to unusual stimuli but may overreact to them. Local conditions such as an exposed and dry epicardium may also exaggerate the response to these various factors.

In the majority of cardiac arrhythmias seen in the operating room, the cause is immediately evident, particularly if it is mechanical. The common irregularities (ventricular and auricular extra systoles, tachycardia, foci of ectopic beats, shifting pacemaker) cease when the offending indirect or direct manipulation or dislocation is discontinued. Such disturbances are called benign. It must be remembered, however, that these may be the precursors of the malignant states of fibrillation or arrest.

The heart may be protected by a number of prophylactic measures aimed at making the organ less irritable to extrinsic stimulation. General protective measures include regulation of fluid and electrolyte balance. We are learning to restrict water and sodium intake before major operations in elderly patients and those having poor cardiorenal reserves. The proper use of digitalis may lower the incidence of disturbances in rhythm. The preoperative administration of quinidine is meeting with increasing favor (5). It is usually not employed in cases of long-standing auricular fibrillation lest there be sudden reversion of rhythm. Quinidine is effective in decreasing the excitability of the myocardium, in quieting ectopic pacemakers and therefore in reducing the danger of auricular or ventricular fibrillation. It also slows the heart rate either by its action on the sinus node or on the vagus nerve.

During surgical procedures on the heart itself, we have come to lean heavily on procaine to prevent arrhythmia. Procaine raises the threshold of the heart to extrasystoles and fibrillation. It is readily

absorbed from the pericardium and topical use results in a direct effect on the myocardium. The local anesthetic effect may play a part in its action (5). Another important feature is that a 1 per cent solution may be used liberally to keep the epicardium moist during prolonged exposure without ill effect. The erroneous impression has been gained in certain quarters that we, Burstein (6) and Harken and Zoll (4), observed defibrillation of the heart through intravenous procaine injection during cardiac operations in our chest center in the European Theater of the recent war. We did not. We pointed out certain uses of intravenous procaine but we did not see defibrillation. Systemic administration of procaine diminished irritability of the conducting system or myocardium or both and caused reversion of a shifted pacemaker to the sinus node. Intravenous doses of 40 mg. of procaine have been given three times in two hours for this purpose.

In anticipation of the occurrence of arrhythmia, there should be a definite plan of action appropriate to the emergency. These emergencies may fall into the following categories:

I. *Prefibrillation states,* that is, auricular or ventricular extrasystoles, shifting pacemaker, ventricular tachycardia, changes in A-V conduction, A-V block
II. *Ventricular fibrillation*
III. *Cardiac standstill*

There are logical routines to follow in each situation but dogmatism should not conceal the fact that there is much room for improvement in these therapeutic programs.

The prefibrillation rhythms often cease spontaneously or with removal of the stimulus. If the prefibrillation arrhythmia persists, the use of 20 to 30 cc. of 1 per cent procaine repeated up to three times in two hours will generally bring about a normal sinus rhythm. Such procaine administration depresses the conduction system and myocardium sufficiently that ventricular fibrillation is unlikely to develop. This is consistent with our own experience and the experimental work of Wiggers and Wegria (7), Mautz (5), and Mautz and Beck (8). Beck (16) has used procaine given directly into the right ventricle in a similar fashion with cessation of premature beats during surgery. The experience of anesthesiologists with cyclopropane-epinephrine fibrillation makes further warning unnecessary. Certainly, epinephrine should not be used with cyclopropane. So much for the "benign arrhythmias" or prefibrillation states.

The malignant forms of arrhythmia are ventricular fibrillation and cardiac standstill. They are often difficult to differentiate and present only the crises of circulatory arrest. The differentiation and course of intervention is conditioned by whether or not the chest is open when the crisis arises. The anesthesiologist's impression of a circulatory crisis may be immediately confirmed if the chest is already open in a

thoracic procedure. It may be confirmed by subphrenic cardiac palpation if the abdomen is open. Differentiation is more difficult when the chest is closed. Once the existence of fibrillation or cardiac standstill has been established, the chest must be opened. Then the problem resolves itself into discovering the type of crisis and instituting proper treatment.

Paroxysmal ventricular tachycardia or ventricular flutter may be confused with ventricular fibrillation on inspection and one may falsely ascribe a reversion to normal rhythm to therapy that in fact is of no value to the fibrillating heart.

In the event of *cardiac standstill,* the restoration of an effective pump again depends upon prompt action and good teamwork in carrying out a prior plan. Blood oxygenation and propulsion must be accomplished immediately and simultaneously. A high concentration of oxygen must be actively delivered through a patent airway. Circulation must be maintained mechanically by cardiac massage. This may provide the necessary stimulus for restoration of action.

Conventionally, cardiac massage is carried out with the heart cupped in the hand, apex directed toward the base of the palm. The fingers are placed on the posterior ventricular surface, the thumb on the anterior surface and rhythmic compression effected thirty to forty times a minute. Compression is forceful and regular and relaxation is abrupt to augment cardiac filling. Massage is interrupted at regular intervals to provide an opportunity for the heart to take over its own function. The heart may assume a normal, regular beat but the contractions may be feeble; this is perhaps the only condition in which epinephrine should be employed for cardiac resuscitation. Administration of 0.5 cc. of 1/1000 epinephrine directly into the chamber of the right ventricle may serve to increase the strength of the beat. This drug should not be used to *initiate* cardiac contraction as this may result in ventricular fibrillation. Barium chloride (1 or 2 cc. of 0.5 per cent solution) injected directly into the heart may also be used to restore myocardial tone (10, 11). Throughout cardiac massage, the heart should be kept moist with warm saline solution or 1 per cent procaine.

To increase blood flow through the coronary arteries, the aorta may be compressed just above the coronary ostia during the resuscitation (12). It is possible that the limited heart output may be made adequate to sustain the brain by clamping the aorta distal to the left carotid artery during the period of cardiac massage. It appears to us that certain aortas might be fractured or damaged by this maneuver, particularly if the clamp was not properly selected.

In short, resuscitation of the heart from asystole involves: (a) adequate oxygenation; (b) removal of the stimulus; (c) cardiac massage, and (d) reinforcement of the beat.

Conversely, if the circulatory arrest is due *to ventricular fibrillation* or ventricular fibrillation has developed in the course of resuscitation

from cardiac standstill, another previously laid plan must be carried out. This involves direct procaine injection, massage and electric shock.

The sequence for abolishing ventricular fibrillation by electric shock was developed in the laboratory by Hooker (13), Wiggers (14, 15) and Beck (16) and supported by the work of Fauteaux (11) and Beck (8). It consists of the following three steps: (1) 5 to 10 cc of 1 or 2 per cent procaine is injected into the right side of the heart or intravenously; (2) cardiac massage is briefly applied to distribute the drug throughout the myocardial bed; (3) then an instantaneous stimulus to the heart is given by passing an ordinary 110 volt alternating current with 1.5 amperes through the heart between two large electrodes. This shock results in simultaneous contraction of all the myocardium, and after a momentary standstill supraventricular rhythm may develop. A series of such shocks may be required to accomplish defibrillation. The electrocardiogram, if connected or immediately available, will confirm the diagnosis and allow close observation of the results of therapy.

Beck (17) reported the first successful case in a human being of defibrillation by electric shock and procaine administered into the right auricle. There was complete recovery after prolonged ventricular fibrillation. This method is not infallible. It has been successful.

Lampson has recently described a case of ventricular fibrillation in which recovery was accomplished by the use of massage and intravenous procaine, without electric shock (18). Although this particular case report is adequately supported and documented, it certainly does not establish procaine as a satisfactory and dependable defibrillating agent. Incidentally, it must be recognized that cases of spontaneous recovery from ventricular fibrillation have been reported by Robinson (19) and Zindahl (20).*

To repeat, the action of procaine is due to protoplasmic depression of conductivity in cardiac muscle, thus permitting the restoration of normal sinus activity in the presence of certain ectopic arrhythmias. Review of electrocardiographic tracings taken during various cardiac operations showed that intravenous procaine did no more than reduce the incidence of ectopic beats and depress wandering pacemakers; many instances were found in which such changes had occurred either spontaneously or concomitantly with cessation of the irritating stimulus. Previously we had thought that procaine gave us greater control by actual slowing of sinus tachycardia.

* We have recently had a patient develop cardiac standstill coincident with or within a few minutes after giving 40 mg. of procaine. Massage by bimanual technic was followed by ventricular fibrillation. This paradoxical situation of a "procainized" heart in frank ventricular fibrillation called for an unorthodox solution. Neosynephrin was injected into both ventricles and vigorous bimanual massage followed. The fibrillating ventricles reverted to dissociated rhythm, then nodal rhythm, and then sinus rhythm within a few minutes. All changes have been documented by continuous electrocardiographic tracings. This will be reported in detail separately.

Further demonstration of the effect of procaine on abnormal foci has been brought to our attention by Paul Zoll (21).

A patient on whom electrocardiographic tracings revealed a prolonged series of ventricular extrasystoles, with no normal sinus beats in sequence, was given intravenous procaine (1 Gm. per liter) slowly. The patient was fully conscious. During the period of therapy there was noticeable depression of ectopic beats, with intervals as long as two minutes of normal sinus rhythm. A similar reversion was obtained by 1 Gm. of procaine injected intramuscularly in a second patient. Although it was effective, the results were transitory.

A great deal of investigation as to the pharmacologic activity of procaine is now somewhat belatedly in progress. Other agents closely related may be shown to possess greater and more selective effects in the prophylaxis and treatment of cardiac dysrhythmia (22).

Such is the present position. There is much to be learned and the technics need improvement. Every surgeon should be equipped to apply the methods outlined since they appear to be the most effective available. We must appreciate our deficiencies, however. There must be much objective evaluation of the different maneuvers. Better defibrillating drugs are needed. Certainly, more reliable defibrillating equipment must be devised. When these objectives are attained, the heart will be literally in our hands.

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AMERICAN ASSOCIATION OF BLOOD BANKS ANNOUNCES SITE OF THIRD ANNUAL MEETING

The Third Annual Meeting of the American Association of Blood Banks is being planned for October 12, 13, and 14, at The Stevens Hotel in Chicago. The central location was chosen so that the meeting will be more accessible to members from all points of the country. The program which is being planned will be one which will attract blood bank personnel, hospital executives, pathologists, clinicians, surgeons, and other people interested in the procurement, preservation, and administration of blood and blood derivatives. For further information write the Office of the Secretary, 3301 Junius Street, Dallas 1, Texas.