Electrocardiographic Changes During Hypothermia and Circulatory Occlusion

By Clarence E. Hicks, M.D., Malcolm C. McCord, M.D. and S. Gilbert Blount, Jr., M.D.

An analysis of the electrocardiograms from 25 patients during reduction of body temperature and total occlusion of circulation reveals marked abnormalities in conduction and rhythmicity. Hypothermia was accompanied by slowing of the heart rate, depression of intracardiac conductivity, and by inhibition of the normal centers of impulse formation with resultant atrial arrhythmias. Circulatory occlusion during hypothermia was associated with a high incidence of ventricular arrhythmias. Changes noted in electric activity of the myocardium tended to return to normal with release of occlusion and warming of the patient.

HYPOTHERMIA and circulatory occlusion are technics recently introduced to permit direct vision, open heart surgery. Observations under these conditions have revealed a high incidence of cardiac arrhythmias, with ventricular arrhythmias in particular constituting one of the major hazards of the procedure. The purpose of this paper is to describe the changes in electric activity of the heart occurring during hypothermia and circulatory occlusion.

Material and Methods

The 25 patients whose electrocardiograms were analyzed ranged in age from 3 months to 36 years. They represented one-third of the total patients undergoing cardiovascular surgery under hypothermia up to February 1955. These patients were selected for study only in that technically satisfactory electrocardiographic records were obtained throughout the procedure. Twenty-four of these patients had congenital cardiovascular disease. The lesions consisted of valvular or infundibular pulmonic stenosis in 13 patients, atrial septal defect in eight patients and ventricular septal defect in three patients. One patient, the oldest, had acquired heart disease consisting of syphilitic aortic insufficiency. Details of the technics of hypothermic cardiovascular surgery have been presented in previous reports from this Institution.1-2 Standard lead II was employed and was constantly monitored by oscillographic observation. Frequent director-writer recordings were made throughout immersion and surgery, with continuous recording during the occlusion period. Anesthetic agents employed were ether, cyclopropane and Penthotal. Medications administered during surgery included curare, succinyl choline and prostigmine. The patients were hyperventilated throughout the operative procedure. Two of the patients were receiving digitalis and one quinidine at the time of surgery.

Results

The electrocardiographic changes observed during the hypothermia and circulatory occlusion are considered under the following categories.

Changes in Cardiac Rate

Progressive slowing of the cardiac rate occurred with decreasing body temperature. This relationship of cardiac rate to temperature is shown in figures 1 and 2. A steep decline in heart rate is demonstrated in both patients in the early phase of temperature reduction, with a more gradual slowing of rate with further decrease in temperature. The rate frequently increased abruptly, coincident with a change in cardiac rhythm, particularly atrial fibrillation; however, with persistence of the newly established rhythm the rate again gradually decreased with further temperature reduction.
Changes during Hypothermia and Circulatory Occlusion

Marked bradycardia often occurred during occlusion of circulation as is illustrated in line 2 of figure 6. An evaluation of the magnitude of the rate changes is afforded by consideration of the average rates in the 25 patients. Prior to immersion in ice water, the average heart rate was 122, prior to circulatory occlusion 55, and during occlusion 32 beats per minute. The heart rate progressively increased as the body temperature was returned toward normal.

Changes in Conduction Patterns

The P-R interval progressively increased with decreasing temperature levels and concomitant slowing of heart rate. This relationship of the P-R interval to temperature is demonstrated in figures 2 and 3. In figure 3 the P-R interval increased from 0.10 to 0.22 second during the temperature decrease, while the heart rate fell from 120 to 56 beats per minute. The QRS interval was considered in relation to temperature only during maintenance of a supraventricular pacemaker and showed progressive widening with decreasing temperature levels. The average increase in QRS duration in all patients was 72 per cent. The relation of the QRS time to temperature is shown graphically in figure 2. Figure 3 demonstrates the increasing QRS interval as it occurred in the electrocardiogram with a change from 0.05 second at 38° C. to 0.11 second at 23° C. The Q-T interval also widened with decreasing temperatures as is again shown in figures 1 and 3. This Q-T interval increase could not be solely attributed to slowing of the heart rate. A comparison of the Q-T interval to the R-R interval by means of Bazett’s formula demonstrated an increasing C (constant) value with decreasing temperature levels.4

Fig. 1. The heart rate and Q-T interval are shown in relation to temperature during the preocclusion period of hypothermia in a 12 year old boy with a ventricular septal defect.

Fig. 2. The heart rate, P-R and QRS interval are shown in relation to decreasing temperature in a 4 year old boy with infundibular pulmonic stenosis.

Moderate increases in heart rate were noted following mechanical stimulation of the heart during manipulation. The addition of circulatory occlusion to the hypothermic state resulted in further slowing of the heart rate, frequently to the range of 10 to 20 beats per minute.

Fig. 3. Isolated complexes from the tracings of a 4 year old patient show P-R, QRS, and Q-T lengthening during stages of 15° C. temperature decrease. The numbers at the bottom of each panel are degrees centigrade.
Configuration Changes

The only consistent changes in the QRS were the appearance of a Q wave and the development of an R' or notched R wave (fig. 4). The R' or notched R appeared in 10 of the 25 cases. The amplitude of R and depth of S waves varied considerably but with no consistent pattern. There was a tendency to a decrease in QRS amplitudes during immersion. The amplitude and direction of the T waves changed frequently with no consistent pattern. Figure 4 illustrates T-wave inversion with return to the original upright pattern with no relation to surgical manipulation or interference with circulation. The S-T segments showed frequent displacement that could not be correlated with any single factor.

Alterations in Rhythm

A sinus mechanism was observed in all patients at the onset of the procedure. During the induction of anesthesia a supraventricular tachycardia frequently ensued which reverted to a sinus mechanism soon after cooling was begun. There then tended to be a progressive depression in the site of impulse formation with decreasing temperature. In 18 instances the first alteration in rhythm was the appearance of an ectopic atrial focus of impulse formation with a 1:1 atrioventricular response. The site of the pacemaker appeared to vary from the sinus node to an ectopic atrial focus or to a nodal focus. There was occasionally a constant ectopic focus in one of the previously mentioned sites but for convenience of grouping this is also included under the term wandering pacemaker. The temperature level at which a sinus rhythm was supplanted by a wandering pacemaker was related to the age of the patient. Younger patients maintained a sinus mechanism during a greater reduction of temperature than did older patients. A sinus rhythm was maintained throughout the entire preoclusion period of hypothermia in three patients under 8 years of age. In contrast, a wandering pacemaker replaced a sinus rhythm after less than 3°C. temperature decrease in three of the five patients over 15 years of age. This relationship of temperature, age and site of origin of impulse is shown diagrammatically in figure 5. The 7 month old infant, S. M., who showed a transition from sinus rhythm to nodal rhythm after only minimal temperature decrease had demonstrated a nodal rhythm at room tem-

Fig. 4. The numbers at the bottom of each panel are: to the left, the time, and to the right the temperature in centigrade. From low and upright at the beginning the T waves invert at 27°C. then at 24°C, still prior to occlusion, they become upright again. QRS and Q-T lengthening, S-T segment deviation and the appearance of a prominent Q are shown in the tracings of this 12 year old patient.

Fig. 5. The preoclusion rhythm changes are shown with the 25 patients arranged according to age. For simplicity three instances of atrial flutter and two instances of idioventricular rhythm were not included. Note particularly the rhythm changes with minor degrees of hypothermia in the older patients to the right and the persistence of sinus rhythm to low temperature levels in the younger patients to the left.
temperature on the day preceding surgery and thus represented no change in impulse mechanism by hypothermia.

The wandering pacemaker was supplanted by a rapid ectopic atrial focus consisting of atrial fibrillation in 12 patients and atrial flutter in three patients. A correlation was again afforded between the patient's age, the temperature level, and the occurrence of atrial fibrillation. Figure 5 demonstrates the appearance of atrial fibrillation predominantly in the older patients. A further depression in pacemaker to a ventricular level was observed in two patients prior to circulatory occlusion. This change was characterized by a regular ventricular rhythm, widening of the QRS time and further slowing of the rate.

Occlusion of circulation for a period varying from two to nine minutes was associated with marked alterations in the existing rhythms. Occlusion was accompanied by further changes in supraventricular rhythm consisting of a wandering pacemaker in three instances and atrial fibrillation or flutter in five instances. The striking abnormality in rhythm occurring during occlusion was the appearance of ventricular arrhythmias. Occlusion resulted in an idioventricular rhythm in 10 instances, cardiac standstill of 10 seconds or more in 13 instances and ventricular fibrillation in three instances. Examples of the type of idioventricular rhythms occurring during circulatory occlusion are presented in figures 6 and 7. Most of the patients demonstrating these rhythms had either considerable cardiac enlargement, cyanotic forms of congenital heart disease, or underwent surgical incision of the right ventricle. The ventricular rhythms occurring during circulatory occlusion were replaced by a supraventricular rhythm immediately following re-establishment of circulation in all but two patients. Operative deaths occurred in these two patients and were attributable to the arrhythmias per se.

Warming of the patient following the re-establishment of circulation resulted in changes in the pacemaker in the reverse order noted with decreasing temperature. A summary of
the preocclusion and occlusion rhythm changes is presented in table 1.

**DISCUSSION**

There have been many reports of electrocardiographic changes in animals during hibernation or artificial hypothermia. However, the electrocardiographic changes in humans during hypothermia have not been reported in significant numbers. The observations of the group of 25 patients reported at this time afford a basis for comparison with these previous studies in animals. Slowing of conduction and depression of higher centers of rhythmicity have been consistently reported by all observers in this field. The major difference that appears in the present study as compared with observations on animals is the severity of alterations in rhythms and the temperature levels at which they occur. Thus, atrial fibrillation occurs commonly in cooled adult human subjects but only rarely in cooled animals.

Since the alterations in the electric activity of the heart constitute a major hazard of surgical procedures carried out under hypothermia and circulatory occlusion, an appraisal of the factors responsible for these changes is of critical importance. The slowing of heart rate with decreasing temperature is considered to result from a depression of the rhythmicity of the centers of impulse formation. Decreased total body metabolic activity may also contribute to this decreased heart rate. Electric conduction through specialized cardiac tissue

![Fig. 7. Four panels recorded during an eight minute circulatory occlusion period at 27 C. in a 4 year old boy with a ventricular septal defect are shown. There was a return to sinus rhythm following re-establishment of circulation.](image-url)
was consistently slowed with decreasing temperature and was manifest by an increase in the P-R, QRS, and Q-T intervals. This slowing is in accordance with the known effect of cold on the chronaxie of conduction tissues. However, there are additional factors, particularly the potassium level, that may affect conductivity. Swan and associates have shown in experimental animals and in a few patients in this series, that the serum potassium decreases and the pH rises during hypothermia. However, in the tracings analyzed in this study the T-wave configuration was quite variable, at times being very high and peaked and showing little resemblance to classic hypokalemic patterns. This lack of correlation with the potassium level is further supported by the report of Bigelow who, found, using different technics of surgery, a Q-T prolongation with a rise in serum potassium. The widening of the Q-T interval described in our patients with decreasing temperature levels could also be attributed to the slowing in cardiac rate. However, calculation of the Q-Tc by means of Bazett's formula showed a progressive rise as temperature decreased. Thus the major factor resulting in depression of cardiac conductivity appears to be decreased temperature.

The progressive change in the location of cardiac impulse formation followed a similar sequence in all patients. There appeared to be a progressive inhibition of the higher centers of impulse formation with decreasing temperature. The rate of discharge of the sinus node was gradually decreased until rhythmicity was reduced below that of lower centers in atrial or nodal tissue. At this time the rhythm changed from a sinus mechanism to slower supraventricular rhythms with varying foci of impulse formation. To this point, the major effects of the cold were slowing of rhythmicity and conduction; however, with further reduction in temperature, atrial fibrillation or flutter supplanted the slow supraventricular rhythms. Atrial fibrillation was noted to begin with cardiac manipulation in some instances. The replacement of slowly discharging ectopic atrial foci by a rapid mechanism as atrial fibrillation or flutter presents a complex problem. This phenomenon may occasionally result from mechanical stimulation; however, it is considered in most instances to represent increased irritability of the atrial myocardium resulting from metabolic alterations related to decreased temperature. The studies by Penrod and by Bing on the ability of the hypothermic heart to extract oxygen suggest that hypoxia does not play a part in these rhythm changes.

Circulatory occlusion resulted in more profound disturbances of rhythm than those accompanying hypothermia alone. Only two patients exhibited ventricular rhythms prior to circulatory occlusion, suggesting that little ventricular irritability was produced by hypothermia alone. Eighteen additional instances of ventricular rhythms occurred during the period of circulatory occlusion and manipulation of the heart. It is considered that hypoxia of the ventricular myocardium, resulting from the total circulatory occlusion, increases the irritability of the ventricles and is the major factor leading to the development of ventricular arrhythmias. The incidence of ventricular rhythms has been shown to be decreased by the alkalosis resulting from hyperventilation and by the use of cholinergic agents such as prostigmine.

The S-T segment deviations, QRS-configuration changes and T-wave changes did not follow a definite pattern. It was not possible to relate these changes to current of injury, hypoxia, cardiac manipulations or drug administration. The pattern of T-wave inversion with deep hypothermia as reported by Bigelow and Hook was not seen; however, the lowest temperature in the present series was 21.4 C.

**Summary**

An analysis is presented of the electrocardiographic changes recorded in 25 patients during reduction of body temperature and total occlusion of circulation. A wide range of abnormalities was noted in the electric activity of the myocardium under these circumstances. Many of these changes were inconstant and difficult to evaluate; however, there were certain electrocardiographic patterns that
appeared consistently in the majority of the patients. These dominant patterns are summarized as follows.

(1) Cardiac rate and conductivity as reflected by increased R-R, P-R, QRS and Q-T intervals were uniformly and progressively slowed with decreasing temperature.

(2) There was a progressive inhibition of higher centers of rhythmicity with decreasing temperature, with the lower centers of inherent rhythmicity assuming control of pacemaking activity as these higher centers were depressed. In two instances ventricular foci controlled the rhythmicity, thus representing complete depression of the higher centers of rhythmicity.

(3) The reduction of body temperature below a critical level, apparently related to the patient’s age, resulted in an increase in the irritability of the atrial myocardium. Thus, atrial fibrillation or flutter was noted in 50 per cent of the patients in this series.

(4) Ventricular arrhythmias occurred infrequently during the period of temperature reduction. This low incidence of serious arrhythmias during hypothermia alone is considered to result from the production of alkalosis by hyperventilation and from the use of prosthigmine.

(5) Ventricular arrhythmias frequently occurred during the period of total circulatory occlusion. Myocardial hypoxia and trauma incident to cardiac manipulation appear to be major factors responsible for the occurrence of these ventricular arrhythmias.

**SUMMARIO IN INTERLINGUA**

Es presentate un analyse del alterationes electrocardiographic registrate in 25 patientes durante reduction del temperatura corporee e occlusion total del circulation.

(1) Frequentia cardiac e conductivitate cardiac, in tanto que reflectite per le augmento del intervallos R-R, P-R, QRS, e Q-T, esseva uniforme- e progressivemente reducere con le reduction del temperatura corporee.

(2) Con le reduction del temperatura corporee il occurreva un progredente inhibition del centros superior de rhythmicitate. In tanto que le activitate de iste centros superior esseva deprimite, le centros inferior de rhythmicitate inherente usurpava le function de governar le rhythm del corde. In duo casos, focos ventricular governava le rhythm durante que le centros superior de rhythmicitate esseva completely deprimite.

(3) Le reduction del temperatura corporee a infra un certo nivello critic—un nivello que esseva apparentemente relationate con le etate del paciente—resultava in un augmento del irritabilitate del myocardio auricular. Assi fibrillation o flutter auricular esseva notate in 50 pro cento del patientes de iste serie.

(4) Arrhythmias ventricular esseva de in-frequente occurrence durante le periodo del reduction del temperatura. On pote supponer que iste basse occurrence de serie arrhythmias durante hypothermia sol resulta del production de alkalose per hyperventilation e del uso de prosthigmine.

(5) Arrhythmias ventricular esseva de frequente occurrence durante le periodo de total occlusion circulatori. Il pare que hypoxia myocardial e trauma resultante del manipulation del corde es major factores in le production de iste arrhythmias ventricular.

**REFERENCES**


CHANGES DURING HYPOTHERMIA AND CIRCULATORY OCCLUSION

13 Bing, R.: Personal communication.
Electrocardiographic Changes During Hypothermia and Circulatory Occlusion
CLARENCE E. HICKS, MALCOLM C. MCCORD and S. GILBERT BLOUNT, JR.

Circulation. 1956;13:21-28
doi: 10.1161/01.CIR.13.1.21
Circulation is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
Copyright © 1956 American Heart Association, Inc. All rights reserved.
Print ISSN: 0009-7322. Online ISSN: 1524-4539

The online version of this article, along with updated information and services, is located on the World Wide Web at:
http://circ.ahajournals.org/content/13/1/21

Permissions: Requests for permissions to reproduce figures, tables, or portions of articles originally published in Circulation can be obtained via RightsLink, a service of the Copyright Clearance Center, not the Editorial Office. Once the online version of the published article for which permission is being requested is located, click Request Permissions in the middle column of the Web page under Services. Further information about this process is available in the Permissions and Rights Question and Answer document.

Reprints: Information about reprints can be found online at:
http://www.lww.com/reprints

Subscriptions: Information about subscribing to Circulation is online at:
http://circ.ahajournals.org//subscriptions/