CHANGES IN THE EXCITABILITY OF THE HEART DURING THE ACUTE PERIOD OF BURN DISEASE

(UDC 616-001.17-036.11-07:616.12-008.313-07)

A. N. Medelyanovskii and V. B. Troitskii

Pathology Laboratory, Order of Lenin Central Institute of Hematology and Blood Transfusion, USSR Ministry of Health (Presented by Academician N. A. Fedorov)

Translated from Byulleten' Éksperimental'noi Biologii i Meditsiny, Vol. 59, No. 6, pp. 32-35, June, 1965

Original article submitted January 18, 1964

Up to the present time the changes in the functional status of the heart during the development of burn shock have been studied very little. The functional insufficiency of the heart in burn shock, with corresponding EKG changes, according to a number of investigators are connected with the disruption of the coronary blood circulation [1, 2, 3, 6, 8, 15, 16]. In the initial stages of burn shock an increase in cardiac sensitivity is noted, and then resistance to adrenaline [14]. The heart "situated" in the sinocarotid zone of the burned animal, responds with a stronger tachycardia, which paradoxically increases after the injection of acetylcholine [13] than does the intact heart of this animal.

The aim of our investigation was to study the cyclical changes in the excitability of the cardiac ventricle to different stages in the development of the reaction to burn trauma.

METHODS

The experiments were performed on 22 dogs of 12-26 kg in weight. In 17 experiments, several hours before the start of the experiment, after intravenous injection of 0.7-1.2 ml of intranarcone (2% solution) and under etheroxygen anesthesia, the heart was exposed (incision in the fourth intercostal space with a sagittal resection of the pericardium) and bipolar silver electrodes in organic glass mountings were sutured to the surfaces of the right and the left ventricles, to be used both for recording the electrocardiogram and the application of the stimulus. The electrode supports were lead out to the surface, the wound closed, air removed from the thoracic cavity. Burns of one-quarter the skin surface were produced with heated alcohol applied for three minutes. Study of the cyclical changes in cardiac excitability (application of test direct current impulses at successive precise points in the cycle) using an FRK-61 cardiosynchronizator were begun after complete normalization of the intrathoracic pressure and removal of the animal from the anesthetized state. In five animals, operated upon seven to 15 days prior to the experiment, the operative intervention was limited by the extraction (under novocaine anesthesia) of the leads which had been fixed subcutaneously. The effect of stimulation was followed on the screen of FRK-61, BEKS-01 and UO-1 oscilloscopes and was simultaneously recorded with time markers and stimulation markers in three standard and two precordial leads on an eight-channel ink-recording "Kaiser" polygraph apparatus. In order to follow changes in the excitability in the limits of the cycle within the interval used, the cycle was divided into 16 equal parts. For more precise study the refractory period was related not to the duration of the entire cycle but only to the QT which was also divided into 16 equal parts. In this connection, from left to right on the abscissa were inscribed the points of division of systole, for which the threshold values of excitability had been obtained, and then after an interval, the diastolic portion of the whole cardiac cycle, divided into 16 equal parts. More detailed description of the methods are given in other articles [4, 5].

RESULTS

The most striking displacement of all elements in the curve of cyclical changes in excitability were observed during the first hour after the burn was experienced. The characteristic feature of this period, for both ventricles,

TABLE 1. Change in the Absolute Refractoriness of the Left (L) and Right (R) Ventricles During Burn Shock

Nature of change	Time after i		infliction of b		urn (in min)	
J	L	R	L	R	L	R
Increase Decrease $n \cdot \dots \cdot \dots + m \cdot \dots \cdot P \cdot \dots$	14 21 13,0 <0,05	15 18 9,60 <0,001	8 15 12,9 >0,05	8 11 13,4 <0,05	9 16 19,9 >0,05	10 13 11,7 <0,01

TABLE 2. Change in the Diastolic Excitability Threshold of the Left (L) and Right (R) Ventricles During Burn Shock

Nature of change	1 h after burn		4-6 h after burn		Total period of observation	
	L	R	L	R	L	R
Increase Decrease n ± m P	11 13 9,9 <0,001	$ \begin{array}{c} 13 \\ 17 \\ 13,6 \\ < 0,02 \end{array} $	9 17 12,2 >0,05	20 22 $6,1$ $<0,001$	21 9,8 <0,01	15 21 9,8 <0,01

and in particular the right, was a shortening of the period of absolute refractoriness (Table 1). For the right ventricle this shortening was clearly expressed throughout the entire first hour following infliction of the burn.

In subsequent stages of development of burn shock (more than 60 min) mainly shortening of the refractory period of the right, and lengthening of the refractory period of the left ventricle was observed (see figure).

The acute shortening of the absolute refractory period (by 50-75% or more for one or both ventricles) is of particular interest. This was observed in six animals during shock. Similar profound fall in the refractoriness was observed in five cases in the right (tendency toward shortening) and in three cases in the left ventricle.

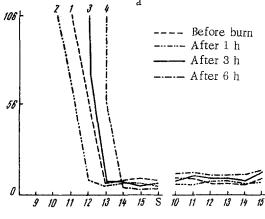
Other pathologic changes in cardiac excitability which arise during shock and predispose to the development of fibrillation and other grave arrhythmias are the considerable lowering of the threshold against a background of its sharp increase during the period of relative refractoriness (dip-phase of Brooks) and in the period which directly follows the relative refractory period.

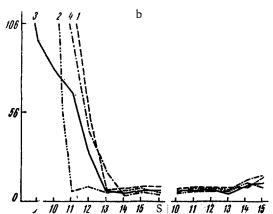
The diastolic threshold of excitability in shock is decreased for the right ventricle and increased for the left one.

The lowering of the threshold of diastolic excitability of the right ventricle (relatively initial) was noted throughout the shock interval. An elevation of the excitability threshold of the left ventricle was distinctly expressed at the end of the first hour of observation; at subsequent stages of shock valid changes in the diastolic excitability were observed only in the right ventricle (Table 2).

During the entire investigatory period a progressive decrease in the excitability threshold for the 12th point was noted for the right ventricle in 15 animals. In this number of instances during the development of shock an increase in the diastolic excitability threshold is noted for the left ventricle. In three cases (or each group), against the background of the main direction of changes in threshold values there were observed temporary deflections of an opposite nature. In nine experiments an increase in the excitability thresholds for the left and a decrease for the right developed at the same time, indicating the development of dissociation of the electrophysiological status of both ventricles. In some of the experiments during the shock period a disappearance of the diastolic plateau of excitability threshold – a sharp variation in it was observed which was expressed as a decrease of the threshold in the early part of diastole and its increase in the presystolic or terminal part of diastole.

The term "shock" is used here to signify the reaction of the organism to burn injury, by analogy with other (distinct in mechanism) reactions in extreme stimulation. The data obtained indicate that during burn shock dissociation of the state of the right and left ventricles develops, expressed in opposite changes in the value of diastolic threshold and prolongation of the refractory period. This dissociation, evidently, may be explained by disturbance in the hemodynamics of the greater and lesser circulations and in the related overexertion and alterations in the ventricles. The lengthening of the refractory period and the increase in diastolic excitability threshold is noted as an effect of adrenalin and increase of functional load. Similar changes in left ventricular indices during shock in combination with roentgenologic data on the post-burn, profound changes in the contractile activity of the left ventricle, and the dissociation of hemodynamic indices of the left and right sides of the heart under these conditions permit us to hypothesize that one of the main reasons for these disturbances is the prolonged work of the left side of the heart against an increased load under conditions of toxemia and hyperadrenalinemia.





Changes in the curve of cyclic variation of ventricular excitability during the development of the reaction of the organism to lethal burn trauma. a) Left ventricle; b) right ventricle; I) before burn; 2, 3, 4) after burn (by respectively, one, three and six hours. On abscissa: Left: points of proportional division into 16 equal parts of electrical systole (QT; points 9 to 16), Right: Points of similar division of the entire cardiac cycle (points 10 to 15). On the ordinate 'tension (in volts) of the threshold (which produces electrical systole) direct stimulus of duration three msec.

A marked shortening of the absolute refractory period, mainly in the late stages of shock, leads to death as also noted in myocardial infarction, and is related to a number of biologically adverse disruptions in protective inhibitions which lead into a state of cardiac excitability at a time of the most tension and "vulnerability" in the cardiac cycle. The above also leads to an abrupt lowering of thresholds during and after the end of the relatively refractory period characteristic for the altered heart [5] and in particular during increase in sympathetic effects.

The smallest deviations in the cyclic curve of excitability in the early periods of the development of shock may be credited to nonspecific neuro-reflex and neuro-mediated influences. Subsequent progression of disruption in the activity of the right and left ventricles reflects the formation of a complex of hemodynamic, biochemical and structural mechanisms which accompany burn shock [7, 9, 11, 12, 17].

LITERATURE CITED

- 1. T. Ya. Ar'ev, Klin med. No. 3, (1962), p. 7.
- 2. N. D. Voitsekhovich, Voenmed. Zh. (1947), No. 12. p. 17.
- 3. E. G. Kalinovskaya, Vrach. delo, No. 7, (1961), p. 15.
- 4. A. N. Medelyanovskii, O. I. Kiselev, and E. V. Bogdanova, In book: Phase methods of study and control of the functions of the cardiovascular system [in Russian], Moscow., (1963), p. 64.
- A. N. Medelyanovskii, Pat. fiziol., No. 5, (1963),
 p. 68.
- 6. N. A. Fedorov and S. V. Skurkovich, Thesis reports of the 34th plenary session of the Scientific Council of the Central Institute for Hematology and Blood Transfusion [in Russian], Moscow, (1955), p. 36.
- 7. L. J. Buis and F. W. Hartman, Am. J. clin. Path., 11, (1941), p. 275.
- 8. H. A. Bozzard In book: Research in Burns. Philadelphia, (1962), p. 109.
- 9. J. Han, P. Garcia de Jalon, Fed. Proc., 22, (1963), N. 2, Pt. 1, p. 288.
- 10. J. D. Hardy et al., Surg. Gynec. Obstet., 101, (1955), p. 94.
- 11. H. N. Harkins, The Treatment of Burns. Springfield, (1942).
- 12. J. P. Gilmore and S. W. Handford, J. appl. Physiol., 8, (1956), p. 393.
- 13. A. Nana, C. Mircioin, and E. Neumann, In book, Stud. Cercet. Med. (Cluj), 12, N 2, (1961) p. 223.
- 14. I. H. Page, Am. J. Physiol., 142, (1944), p. 366.
- 15. D. W. Jr. Richards, Harvey Lect. (1943-1944), 39, (1944), p. 217.
- 16. S. Sevitt, Burns. Pathology and Therapeutic Applications. London, (1957).
- 17. K. H. Zinck, Verh. dtsch. Ges. Kreisil.-Forsch., Tag. II, S. 263. (1938).