Cineangiocardiographic Observations on the Asynchronism of Cardiac Contraction during Ventricular Pacing

Hideo Ueda, M.D., Kenichi Harumi, M.D., and Keiji Ueda, M.D.

SUMMARY
Effects of experimental ventricular pacing on ventricular function were studied on 5 anesthetized dogs. The heart was stimulated by bipolar electrodes placed at the right atrial appendage and the epicardium at the apex or base of the left ventricle. Cineangiocardiographic analysis of the left ventricle was made and the sequential change in the shape of the left ventricle during ventricular contraction was compared among each of three stimulation sites.

A systolic expansion of the localized area of electrical stimulation was observed during stimulation at the base as well as at the apex of the left ventricle, but not during right atrial stimulation, and it was consistently reproducible in any given experiment. Correlation of individual cine frames to simultaneously recorded ventricular pressures revealed that the bulge developing in mid-systole reached its maximum size at the end of the ventricular ejection period and disappeared during the period of isovolumic relaxation of the ventricle.

Additional Indexing Words:
Atrial pacing    Systolic bulge of the ventricle    Spread of excitation

Since the study by Wiggers\textsuperscript{1} in 1925, the effect of sequence of ventricular excitation on cardiac hemodynamics has been studied in animals\textsuperscript{2-7} and recently in man.\textsuperscript{8-10} However, hemodynamic differences induced by different pacemaker sites have not been clearly established and a variety of conflicting findings have been reported. In these previous studies, ventricular performance has been analyzed and evaluated mainly from the hemodynamic standpoint, whereas changes in ventricular shape and size during ventricular pacing were not evaluated.

In this regard, the present experiments were designed to observe the morphological changes of the ventricle during experimental cardiac pacing with the use of cineangiocardiography, and to analyze the relation between aberrant ventricular conduction and cardiac function.

From the Second Department of Internal Medicine, Faculty of Medicine, University of Tokyo.
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METHODS

Five mongrel dogs weighing 10 to 13 Kg. were anesthetized with pentobarbital 30 mg./Kg. and the chest was opened in the mid-line and the heart was cradled in the open pericardium under artificial respiration.

Bipolar stimulating electrodes were applied at the right atrial appendage and selected sites of the epicardial layer of the left ventricular myocardium. As shown in Fig. 1, the apical electrode was placed at the apical portion of the left ventricle and the basal electrode near the left atrioventricular sulcus midway between the anterior interventricular sulcus and Margo obtusus.

The sinus node was crushed and the heart was paced from 60 to 240 beats per min. The chest was closed and the body surface scalar electrocardiogram was recorded by McFee's lead system using foam rubber as a volume conductor. In
3 dogs, aortic and left ventricular pressures were recorded through cardiac catheters and a pressure transducer (Statham P 23Db) on an oscillographic recorder (paper speed 10 cm. per sec.).

The dog was positioned in the right anterior oblique position on an X-ray table and 2 to 3 ml. of contrast material (sodium iothalamate) was injected into the left ventricle through a cardiac catheter. Cineangiocardiograms were taken with a 9 inch image intensifier and a 16 mm. motion picture camera with a speed of 40 frames per sec. The contour of the left ventricular cavity on cine films was analyzed frame by frame by tracing with a projector and/or by printing on paper. The distance from the aortic valve to the apex of the left ventricular cavity was measured as the long axis of the ventricle and the width (short axis) was measured on a line perpendicular to the long axis and at the height of the basal pacing electrode in each cine frame. The position of the mitral valve was analyzed on cine film and the timing of the closure and opening of the valve were identified as the onset of isovolumic contraction and the end of isovolumic relaxation, respectively. The duration of ventricular systole was estimated from the time interval between the closure and opening of the mitral valve.

Nineteen experiments of atrial pacing, 14 of ventricular apical pacing and 11 of ventricular basal pacing were performed on 5 dogs. Cineangiocardiographic and hemodynamic findings during ventricular pacing, either apical or basal, were compared with those during atrial pacing at the same rate of pacing.

Results

Changes in the shape of the left ventricle during ventricular contraction induced by each of three different sites of pacing were compared. Results in representative experiments are shown in Figs. 2, 3 and 4.

1) Sequential change in the shape of the left ventricle during right atrial pacing (Fig. 2)

Systolic change of ventricular shape was the same as that of sinus rhythm and shortening of the long axis by 9 to 22% was accompanied by shortening of the short axis by 25 to 55%, depending on the pacing rate.

2) Sequential change in the shape of the left ventricle during apical pacing of the ventricle (Fig. 3)

Short axis was shortened by 29 to 45% during ventricular contraction, which was similar to that observed during right atrial pacing at the same heart rate. The long axis of the left ventricle was elongated during initial phase of ventricular systole and distension of the half-contracted apical portion of the ventricle was observed at 75 to 100 msec. after the closure of the mitral valve. The distended apical portion (apical bulge) progressively increased in its size as ventricular ejection proceeded and reached its maximum size at 125 to 200 msec. after the closure of the mitral valve. The bulge then rapidly disappeared during the subsequent short period (usually 50 msec.) just prior to the opening of the mitral valve. The duration of systole was prolonged
Fig. 2. Sequential change of the left ventricle during right atrial pacing. (Exp. III-1, pacing rate: 180/min.)
Cineangiocardiographic frames are shown from left to right at 50 msec. interval. Electrocardiogram, aortic and left ventricular pressures are shown to indicate the timing of each cine frame.
Fig. 3. Sequential change of the left ventricle during pacing at the apex of the left ventricle. (Exp. III-2, pacing rate: 180/min.) Cineangiographic frames during ventricular contraction are shown from left to right at 50 msec. intervals. Arrows indicate the apical bulge.
Fig. 4. Sequential change of the left ventricle during pacing at the base of the left ventricle. (Exp. III-3, pacing rate: 180/min.) Cine angiocardiographic frames during ventricular contraction are shown from left to right at 50 msec. interval. Arrows indicate the basal bulge.
during apical pacing of the left ventricle, compared to that during right atrial pacing at the same rate of pacing.

The shape of the apical bulge was usually ellipsoidal or cylindrical and approximately 0.8 to 1.0 cm. in width and 1.0 to 1.5 cm. in length. The shape and size of the bulge were nearly identical in a given dog and they were not influenced by the pacing rate in the majority of experiments.

3) Sequential change in the shape of the left ventricle during basal pacing of the ventricle (Fig. 4)

The long axis decreased in its length by 9 to 21% during ventricular contraction, which was similar to that observed during right atrial pacing at the same pacing rate. In early systole, 75 to 100 msec. after the closure of the mitral valve, a small outward convexity of the left ventricular cavity (basal bulge) was observed at the anterior margin of the left ventricular cavity beneath the electrode at the base of the ventricle. The bulge gradually expanded toward the end of the ventricular ejection period and reached its maximum size at 150 to 200 msec. after the closure of the mitral valve, forming a semi-circular bulge with its diameter approximately 5 to 8 mm. The bulge then rapidly disappeared during the subsequent period of 50 msec. prior to the opening of the mitral valve.

The size of the basal bulge was nearly identical in a given experiment and not influenced by pacing rate, but it was smaller than that of the apical bulge produced by apical pacing in the same dog.

The circumscribed distension or bulge of the left ventricle during ventricular systole showed the following characteristic features; i) the bulge was consistently reproducible and distinctly observed only during ventricular pacing, but not during atrial pacing, ii) it developed from mid-systole, progressively increasing in its size toward the end of the ventricular ejection period, and rapidly disappeared during the subsequent period corresponding to isovolumic relaxation, iii) its location was closely related to the site of the pacing electrode on the ventricle.

**DISCUSSION**

A paradoxical, systolic distension of a circumscribed area of the ventricular myocardium near the site of stimulation of the epicardium, which has not been previously observed, may well be a direct effect of asynchronous contraction of the ventricular myocardium during ventricular pacing.

The study of Durrer11) on the spread of excitation during ventricular stimulation demonstrated a slow conduction around the site of stimulation and then faster conduction to the rest of the myocardium. Consequently, the
refractoriness of the site of stimulation and its adjacent area would terminate earlier and relaxation of the myocardium around the site of stimulation may begin earlier than the rest of the myocardium, which may result in the protrusion of a circumscribed mass of myocardium due to the elevated intraventricular pressure. The minor differences in the extent of myocardium involved in the systolic bulge in each dog may be due to individual differences in functional integrity of the conduction system and of the contractile state of the myocardium. These findings may help to clarify conflicting results reported in previous studies\(^1\)-\(^5\) on the relation between the site of stimulation within the ventricle and hemodynamic sequelae.

This study also suggests that the systolic bulge produced by epicardial stimulation of the ventricle may be one of the major causes of relative ineffectiveness of ventricular pacing.\(^6\)-\(^8\)

**REFERENCES**