The Evidence of Transmural Unidirectional Block by Experimental Current Induced Ventricular Tachycardia in the Canine Heart

HIROMU HAMAMOTO, M.D., EJI KINOSHITA, M.D., HARUO TOMODA, M.D. and YUICHIRO GOTO, M.D.

Under normal circumstances, excitation of the heart spreads from the endocardial site to the epicardial site via the Purkinje fiber. The purpose of this study is to observe whether the transmural unidirectional block exists near the current induced local clamp during sustained ventricular tachycardia. Twelve open chested dogs were used and the local transmural electrograms were taken by three plunge electrodes consisting of two pairs of bipolar ones which were located at the epicardial site and the endocardial site respectively. Evidence of the unidirectional block was detected by means of excitation order between the endocardial site, the epicardial site and the conduction direction. We used the concept of intrinsic deflection for documentation of the conduction direction, i.e., an opposite change of the initial deflection of the local electrogram indicates a reversal of direction. Ventricular tachycardia was induced by a current with 3-5 mA to the midwall of the myocardial muscle. Results showed that 1) the change of conduction order and the reverse direction of conduction were seen simultaneously, and 2) ventricular tachycardia was converted to ordinary rhythm by the incision at the epicardial site of myocardium. These results may prove that the unidirectional block is the major qualification for the detection of re-entry.

RE-ENTRY is an primary means of initiation and sustenance of an abnormal rhythm, particularly ventricular fibrillation. Slow conduction and unidirectional block have been shown to be the prerequisites of re-entry. Conduction can be slowed significantly by ischemia, although, the relative effects of ischemia on conduction in the epicardial and endocardial halves of the myocardium have seldom been studied in the same model. Recent studies on the effect of ischemia on myocardial conduction have been mostly limited to the epicardium. The significance of endocardial conduction has rarely been taken into consideration, although its significance has been suggested by intra cardiac mapping studies.

We investigated the transmural conduction time in the acute stage of experimental myocardial infarction, which revealed a significant disparity between the conduction of premature impulses at the endocardium and at the epicardium. The conclusions pointed to the possible existence of re-entry by means of the conduction disparity but the unidirectional block. Although the mapping technique has improved the observation of unidirectional block it is not totally satisfactory yet.

ORIGIN OF THE STUDY

Excitation of the heart proceeds from the endocardial site to the epicardial site via the Purkinje fibre. Under certain pathophysiological conditions, such as during the ventricular tachy-
Fig. 1. Schema of the conduction direction and the excitation order through the transmural myocardium.

Fig. 2. Position of the electrodes at a superficial (left side) and a transmural (right side) view.

MATERIAL AND METHOD
Twelve mongrel dogs weighing between 10 and 20 Kgms underwent the protocol preparations. After general anesthesia with pentobarbital, i.e., 1% 1-2 ml intravenous infusion, the dogs were ventilated with a Harvard respirator. The chest was opened via left lateral thoracotomy through the 5th intercostal space, the lung was retracted and the heart was suspended in a pericardial cradle.

Two kinds of transmural plunge electrodes were constructed a recording electrode and a stimulating electrode. The recording electrode consisted of four silver electrodes with a surface area of 0.1 mm² each, set 0.5 mm apart with an outside needle diameter of 0.1 mm. Thus, each electrode contained an epicardial bipole and an endocardial bipole. The center of the recording bipoles were set 7 mm apart. The stimulating electrode consisted of a pair of electrodes with a surface area of 1 mm² each and set 0.5 mm apart.

Japanese Circulation Journal Vol. 51, February 1987
This pair of electrodes was used for delivering current which was positioned at the midwall of the myocardium.

The second figure indicates the positions of the electrodes with a superficial view and a transmural view. Three recording electrodes were inserted into the free wall of the left ventricular muscle in a triangular pattern, i.e., 1.5–2.0 cm apart and two of the delivering electrodes were also inserted to the myocardium. One of the stimulating electrode was positioned in the center of the triangle initialed (I), and another one was also inserted to the myocardium (O) 4–5 cm away from the (I). Current was delivered alternately between (I) and (O) electrodes.

Thus, the reversal of excitation order and the change of conduction direction were observed. In order to confirm the evidence of a re-entrant pathway, we made an incision at the epicardial site of a recording electrode where the re-entrant pathway during ventricular tachycardia induction is most likely to exist, and observed whether the ventricular tachycardia converted to ordinary rhythm.

RESULTS

1. Transmural conduction time from the endocardial site to the epicardial site was approximately 15–20 msec/6–7 mm.

2. Current requirement for ventricular tachycardia was about 3–5 mA, and the overdose current induced ventricular fibrillation.

3. Reproducibility of ventricular tachycardia induction. There was a high correlation between current requirement and the unidirectional block location, i.e., if the first ventricular tachycardia was induced by 4 mA current and the unidirectional block location was seen at the C electrode, the same phenomena occurred following the second and third induction.

4. Evidence of transmural unidirectional block by the ventricular induction at the center of the triangle. Figure 3 shows the existence of unidirectional blocks at each electrode simultaneously. The upper panel indicates the real recording at the three electrodes, i.e., top recording indicates the ordinary ECG and follows local electrograms at the three points of each endocardial and epicardial site simultaneously.

The lower panel indicates the schematic representation of conduction direction and the time difference between the endocardial and the epicardial sites of the electrodes.

According to the figure, regular conduction order and direction between endocardial site and epicardial site are seen at the first beat at the three electrodes followed by abrupt disorders of both conduction order and direction start from the second beat, i.e., ventricular tachycardia.
Fig. 4. Ventricular tachycardia induction at the outside of triangle.

Fig. 5. Ventricular tachycardia conversion by a incision at the epicardial site of myocardium.

starts. There are no evidences of unidirectional block on the three electrodes even though there is ventricular tachycardia. At the fourth beat there is a sudden appearance of unidirectional block, surrounded by ovals in the figure, at the three electrodes simultaneously, succeeding to

the next beat, i.e., at A and C electrode but C.

Another type of unidirectional block was seen at only one electrode and followed by a shift to the next electrode with ventricular tachycardia shape change on the surface ECG. The conclusion is that the multiple types of unidirectional block pathway exist at the current inducing clamp.

5. Fall of transmural unidirectional block proof by the ventricular tachycardia induction at the outside of the triangle. Figure 4 shows no evidence of the unidirectional block even though ventricular tachycardia was present. Ventricular tachycardia induction was made 4-5 cm away from the triangle which was outside the area of the clamp. Ventricular tachycardia starts from the second beat with disorder of the conduction direction at each electrode. There is neither change of the disorder nor evidence of the unidirectional block during the ventricular tachycardia. This is probably due to the fact that each electrode is set far the ventricular tachycardia focus; furthermore, evidence of constant rhythm indicates that a certain pathway to keep ventricular tachycardia will exist. Combined with the previous figure, there exist multi unidirectional blocks at the border of clamp but inside where there will exist entrance block. This might be essential for explain the formation of a re-entrant pathway.

6. Ventricular tachycardia conversion to ordinary rhythm by cutting at the epicardial site of the myocardium. According to Fig. 5, ventricular tachycardia was induced by increasing the current flow; the ventricular tachycardia starts from the 6th beat with a current flow of 6 mA, and was maintained with continuous induction of 6 mA. At the 19th beat an incision was made at the epicardial site followed by the ventricular tachycardia conversion to ordinary rhythm.
rhythm but with continuous current. Ventricular tachycardia conversion occurred three times in the twelve dogs. This phenomenon is one proof of the re-entrant pathway cut-off.

7. **Clamp size** build by current induction seems to be around 2–3 cm diameter of globular shape which surrounds the induction electrode. According to our data, local existence of globular clamp with depolarized status is enough to cause the reentrant pathway.

**DISCUSSION**

Local conduction disparity, which is occasionally induced by ischemia, and the unidirectional block are major conditions for the existence of re-entry. Recently, the evidence of local unidirectional block was observed by mapping at the superficial of the epicardial site but poor through transmural myocardium.

The aim of this study is to document the unidirectional block by means of a change in conduction direction and the reversal of local excitation order, i.e., transmural disorder of the excitation between the endocardial site and the epicardial site of the myocardium. The concept of intrinsic deflection was used to explain the change in conduction direction.

We created current-induced ventricular tachycardia from the midwall of the myocardium where the local clamp will be constructed. Thus, when current is applied, the local clamp is formed around which the re-entrant pathway results. When the current is off the clamp is also released and is followed by ventricular tachycardia conversion to regular rhythm. This fact will be explained that the current induced entrance block through the clamp leading to induce the unidirectional block. This is a due to the prolongation of depolarization, i.e., keeping the local clamp area of refractory period which results in the inability of the next conduction to get through. With the current off the clamp also disappears and the vanished area of the clamp will become normal, resulting in the blocking off of the next excitation.

As we mentioned, the local formation of the unidirectional block does not exist inside the clamp but near the clamp border; furthermore, the numbers of unidirectional block exist multiply. According to our data, the current requirement for ventricular tachycardia induction and maintenance was about 3–5 mA with a clamp size of around 3 cm diameter of globular shape surrounding which the plural numbers of re-entry pathway will exist. In case of excessive current, we occasionally induced ventricular fibrillation which presumably results from multiple unidirectional block to ventricular fibrillation.

It is said that ischemia reduces the conduction velocity to 95% at most, so that the re-entrant size to maintain ventricular tachycardia is 2–3 cm of diameter which was discussed from the viewpoint of refractory period.

The last figure shows the schematic representation of the time course change of ventricular tachycardia induction resulting in conversion to ordinary rhythm by the incision at the epicardial site of the myocardium. In general, conduction proceeds from the endocardial site to the epicardial site (Fig. 6-1) and induction starts with the gradual increase of the current flow and conduction direction at the epicardial site reversing at the endocardial site (Fig. 6-2). Thus, the unidirectional block occur (Fig. 6-3), at this point, if the current is stopped, ordinary conduction occurs (Fig. 6-3'). Ventricular tachycardia will be maintained under the current flow of 3–5 mA at the midwall (Fig. 6-4). In order to prove the evidence of the unidirectional block, an incision was made at the epicardial site and the ventricular tachycardia converted to ordinary rhythm with the clamp still existing (Fig. 6-4').

From the anatomical point of view, the arrangements of the myocardial cell are complicated, especially the transmural component of the myocardial cells through which the conduction propagate. There, the transverse resistance of the membrane is higher than that of the longitudinal resistance. It can be assumed that the disparity in the arrangement of the myocardial cell makes it easy to produce the unidirectional block.

**REFERENCES**


10. WEIDMANN S: Shortening of the cardiac action potential due to a brief injection of KCl following the onset of activity. J Physiol 132: 157, 1956