EFFECTS OF INJURY TO THE RIGHT VENTRICULAR CONDUCTING TISSUE OF CANINE HEARTS ON EPICARDIAL ACTIVATION SEQUENCE AND ELECTROCARDIOGRAMS

MIYOSHI OHNO, M.D., JUNJI TOYAMA, M.D., TATSUO KOHBE, M.D.
SHINOBU ISOMURA, M.D., ITSUO KODAMA, M.D.
AND KAZUO YAMADA, M.D.

The distal right ventricular conducting tissues of 45 canine hearts were experimentally interrupted in various degrees by a transmural incision of the right ventricular free wall, a trans-section of the lateral branches of the right bundle or by injury to the endocardial Purkinje network. Right ventriculotomy caused a slight activation delay (less than 10 msec) of the right ventricle which was restricted to the distal area from the incision, but the delay was not long enough to cause a significant prolongation of the QRS duration in the limb lead ECG. In one experiment, a vertical incision in the middle region induced an exceptional, marked alteration of the right ventricular activation sequence and an apparent prolongation of the QRS duration (16 msec) indicating an incomplete RBBB. However, anatomical analysis revealed that the lateral branches were nearly completely interrupted by the incision. An extensive injury to the Purkinje network extending toward the right ventricular outflow tract by a blunt scalpel caused a local activation delay in the outflow tract, without producing any serious delay resulting in an ECG pattern of RBBB.

These results may suggest that right ventriculotomy would induce the RBBB pattern of ECG if lateral branches are extensively injured by the surgical procedure, and that the injury to the Purkinje network extending toward the outflow tract does not play a primary role of genesis of RBBB pattern in ECG.

SURGICAL correction of congenital heart diseases such as tetralogy of Fallot (TF), ventricular septal defect (VSD) or pulmonary stenosis (PS) is frequently followed by the electrocardiographic

pattern of the right bundle branch block (RBBB). This RBBB pattern has been explained by the operative trauma in the main right bundle branch (RBB) during the repair of VSD¹⁻⁴ and by the injury to the more distal right ventricular conducting tissues due to the right ventriculotomy⁵⁻⁷ or to the infundibular resection at the pulmonary conus⁸. Among these explanations, the trauma in the proximal portion of the

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Division of Circulation and Respiration, Research Institute of Environmental Medicine, Nagoya University, Nagoya, Japan

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Address for reprints: Miyoshi Ohno, M.D., Division of Circulation and Respiration, Research Institute of Environmental Medicine, Nagoya University, Furocho, Chikusa-ku, Nagoya 464, Japan

Fig.1. Arrays of 43 electrodes for recording the epicardial electrogram. Blank circles indicate the position of electrode placed on the right ventricle and left ventricle. RV: Right ventricle, LV: left ventricle

main RBB was confirmed to play an important role in the genesis of RBBB by many clinical and experimental studies^{1-4,9-14}. However, there is little available evidence supporting the etiologic significance of the injury to the more distal right ventricular conducting tissue or Purkinje network. In the present study the distal right ventricular conducting tissues of canine hearts were experimentally interrupted in various degrees by transmural incision of the right ventricular free wall, by trans-section of the lateral branches of the right bundle or by injury to the endocardial Purkinje network extending toward the pulmonary conus. The effects of these surgical interventions on the epicardial activation sequence as well as on the electrocardiographic pattern were investigated in order to elucidate the precise mechanism responsible for the surgically induced RBBB pattern.

METHODS AND MATERIALS

Forty-five mongrel dogs weighing 8 to 15 kg were anesthetized with thiamylal sodium (30 mg/kg, intravenously). Under artificial ventilation the heart was exposed through midsternal thoracotomy. Then, the heart surface was completely covered with a flexible nylon mesh sock, to which 43 silver-wire electrodes were attached for recording epicardial potentials. As illustrated

in Fig. 1, 29 out of the 43 electrodes were positioned equidistantly on the surface of the right ventricle, and the remaining 14 electrodes were similarly placed on the left ventricle.

The epicardial potentials which were recorded as unipolar electrocardiograms with respect to the Wilson's central terminal, were amplified 50 fold and fed into a microcomputer processing system for the body surface map (HPM 5100, Chunichi Electric Corp.). The conventional limb lead ECGs (I, II and III) were also recorded using the same system. As reported previously,¹⁵ this body surface mapping system can simultaneously input the data of 96 channel electrocardiograms (ECGs) for 250 msec at a sampling interval of 1 msec. All the epicardial electrograms processed simultaneously by this system were displayed as a digital format on a matrix for the following measurements. The onset of ventricular activation was defined at the instant when at least 5 epicardial electrograms showed a sequential potential change exceeding the noise level of the recording system. The arrival time of activation at each lead point was determined by measuring the time required from the onset of ventricular activation to the instant showing steepest potential change of the electrogram, and then the epicardial activation sequence was constructed from these values.

After the control measurements, the follow-

TABLE I CHANGES OF THE LATEST ACTIVATION TIME OF RIGHT VENTRICLE AND QRS DURATION FOLLOWING VERTICAL INCISION

Experiment No.	Latest activation time of right ventricle			QRS duration		
	Control	After incision	Difference	Control	After incision	Difference
<i>Incision along atrioventricular groove</i>						
1	28	32	4	34	36	2
2	28	34	6	32	35	3
3	25	30	5	35	35	0
4	30	33	3	33	35	2
5	34	37	3	40	40	0
Mean $\pm$ SD	29.0 $\pm$ 3.0	33.2 $\pm$ 2.3	4.2 $\pm$ 1.2*	34.8 $\pm$ 2.8	36.2 $\pm$ 1.9	1.4 $\pm$ 1.2
<i>Incision on middle region of right ventricular free wall</i>						
6	26	33	7	30	35	5
7	29	44	15	32	48	16
8	28	34	6	32	35	3
9	27	36	9	36	38	2
10	30	38	8	38	40	2
Mean $\pm$ SD	28.0 $\pm$ 1.4	37.0 $\pm$ 3.9	9.0 $\pm$ 3.2*	33.6 $\pm$ 2.9	39.2 $\pm$ 4.8	5.6 $\pm$ 5.3
<i>Incision along anterior interventricular groove</i>						
11	34	37	3	37	40	3
12	26	28	2	37	37	0
13	28	31	3	32	34	2
14	30	32	2	35	37	2
15	33	34	1	40	40	0
Mean $\pm$ SD	30.2 $\pm$ 3.0	32.4 $\pm$ 3.0	2.2 $\pm$ 0.7**	36.2 $\pm$ 2.6	37.6 $\pm$ 2.2	1.4 $\pm$ 1.2

*: Significantly different from the value of control at $p < 0.01$; **: significantly different from the value of control at $p < 0.025$

ing three kinds of surgical intervention were made to injure the distal right ventricular conducting tissues: 1) transmural incision of the right ventricular free wall and the outflow tract (ventriculotomy), 2) trans-section of the lateral branches of the right bundle, and 3) injury to the endocardial Purkinje network extending toward the right ventricular outflow tract. The ventriculotomy was performed in a transverse or vertical direction by using a small sharp scalpel. The hemorrhage from the cut wound was immediately stanchied by a running suture. Trans-section of the lateral branches of the right bundle was accomplished by cutting all the false-tendons, using a scalpel for tonsillectomy which was trans-atrially introduced into the right ventricular cavity. To cut the false-tendons, the tip of the

scalpel was moved blindly toward the anterior papillary muscle. Injury to the endocardial Purkinje network extending toward the outflow tract was produced by scratching with the tip of the Kocher's forceps inserted into the right ventricular cavity through the free wall near the outflow tract. The effects of these surgical procedures on the activation sequence and on the limb lead ECG were evaluated after a recovery period during which the ST-segment in epicardial electrograms deviated by the myocardial injury was returned to their control levels.

When all the measurements were completed, the heart covered with the mesh sock was isolated, and the position of each electrode was confirmed. Then, the heart was examined grossly with Lugol stain to localize the anatomic lesions.

Fig.2. Epicardial activation sequence and limb lead ECG before (A) and after (B) vertical incision along the atrioventricular groove. The ventricular activation time is indicated by the accompanying graph. The solid line depicted on the right ventricle in panel B shows the line of incision (Experiment No.1).

Fig.3. Epicardial activation sequence and limb lead ECG before (A) and after (B) vertical incision on the middle region of the right ventricular free wall (Experiment No.9).

Statistical analysis of measured values was performed by Student's t-test, and P values of less than 0.05 were considered significant for differences.

RESULTS

Normal Right Ventricular Activation Sequence

We investigated the activation sequence of dog hearts as well as limb lead ECG by using a micro-computer processing system for body surface map. This system enabled us to examine the epicardial activation sequence much more extensively than other recording methods employed previously^{16,17}

Under the control condition, all the 45 dogs examined showed an activation sequence more or less similar to those reported by previous investigators^{14,16-18} As shown in Figs. 2-10 (Panel A),

the initial epicardial breakthrough appeared in the anterior lower portion of the right ventricular free wall 7 to 10 msec (average $\pm$ SD, 8.3 ± 0.9 msec) after the onset of ventricular activation, then the activation wave front spread radially over the entire epicardial surface of the right ventricle. The latest region to be activated in the right ventricle was the right ventricular outflow tract. It took 27 to 34 msec (average $\pm$ SD, 30.2 ± 3.0 msec) from the onset of ventricular activation until the completion of the right ventricular epicardial activation. The QRS duration in limb lead ECG was 34 to 40 msec (average $\pm$ SD, 36.7 ± 2.3 msec).

Right Ventriculotomy

- 1) Vertical incision along the atrioventricular groove
Effects of vertical incision along the atrioven-

Exp. No11

Fig.4. Epicardial activation sequence and limb lead ECG before (A) and after (B) vertical incision along the anterior interventricular groove (Experiment No.11).

tricular groove were examined in 5 dogs. Representative results are illustrated in Fig. 2. After the incision (Fig. 2B, solid line), an activation delay by several milliseconds occurred at the lateral area from the incision. On the contrary, the appearance time and the region of epicardial breakthrough were not affected. The latest region to be activated (32 msec from the onset of ventricular activation) was in the outflow tract. The configuration and the duration of QRS complex of the limb lead ECG were virtually unaffected by the incision.

Table I summarizes the results obtained from the 5 experiments. The latest activation time of the right ventricle was slightly but significantly increased after the incision, whereas no significant prolongation was observed in the QRS duration.

Fig.5. Epicardial activation sequence and limb lead ECG before (A) and after (B) transverse incision in the outflow tract (Experiment No.18).

2) Vertical incision on the middle region of the right ventricular free wall

Fig. 3 shows a typical experiment in which vertical incision was made on the middle region of the right ventricular free wall (solid line in Fig. 3B). After the incision, activation time over the inflow tract (lateral area from the incision) was apparently delayed, but the appearance time and the region of the breakthrough were almost the same as observed under the control condition (Fig. 3A). The latest region to be activated on the right ventricle was in the inflow tract (36 msec after the onset of ventricular activation).

The results obtained from 5 dogs were summarized in Table I. The latest activation time of the right ventricle was significantly delayed compared with the control values. Although

TABLE II CHANGES OF THE LATEST ACTIVATION TIME OF RIGHT VENTRICLE AND QRS DURATION FOLLOWING TRANSVERSE INCISION

Experiment No.	Latest activation time of right ventricle			QRS duration		
	Control	After incision	Difference	Control	After incision	Difference
<i>Incision in outflow tract</i>						
16	33	39	6	37	42	5
17	29	32	3	33	36	3
18	28	32	4	38	38	0
19	26	28	2	33	33	0
20	36	41	5	40	43	3
Mean $\pm$ SD	30.4 $\pm$ 3.6	34.4 $\pm$ 4.8	4.0 $\pm$ 1.4*	36.2 $\pm$ 2.8	38.4 $\pm$ 3.7	2.2 $\pm$ 2.0
<i>Incision in middle of right ventricular free wall</i>						
21	33	34	1	40	40	0
22	31	40	9	42	44	2
23	33	38	5	40	40	0
24	29	30	1	32	34	2
25	30	34	4	36	38	2
Mean $\pm$ SD	31.2 $\pm$ 1.6	35.2 $\pm$ 3.5	4.0 $\pm$ 3.0	38.0 $\pm$ 3.6	39.2 $\pm$ 3.2	1.2 $\pm$ 1.0
<i>Incision along posterior interventricular groove</i>						
26	33	34	1	40	40	0
27	33	37	4	40	42	2
28	33	35	2	36	38	2
29	28	30	2	33	35	2
30	31	32	1	35	35	0
Mean $\pm$ SD	31.6 $\pm$ 2.0	33.6 $\pm$ 2.4	2.0 $\pm$ 1.1**	36.8 $\pm$ 2.8	38.0 $\pm$ 2.8	1.2 $\pm$ 1.0

Symbols are the same as in Table I.

some experiments showed a concomitant increase in QRS duration in limb lead ECG, such a change was statistically insignificant.

Only the experiment Number 7 (Table I), which showed the most remarkable activation delay of the right ventricle after the incision, was accompanied by the alteration of the appearance time as well as the region of breakthrough, resulting in an activation sequence quite similar to those observed after the transsection of lateral branches (Fig. 8). Anatomical examination of this case revealed that the lateral branches happened to be interrupted almost completely by the procedure of incision.

3) Vertical incision along the anterior interventricular groove

One of the 5 experiments, in which a vertical

incision was made along the anterior interventricular groove, is illustrated in Fig. 4. As shown in the left side of panel B (solid line), the right ventricular free wall was incised vertically from base to apex along the anterior interventricular groove. Activation delay caused by this incision ranged from 5 to 15 msec and was restricted to the region between the line incised and the anterior interventricular groove. The epicardial activation sequence in the other area of the right ventricle or in the left ventricle was not influenced by this incision. No serious change was observed in QRS complexes of the limb lead ECG. More or less similar results were obtained from the remaining 4 dogs as presented in Table I, and the latest activation time of the right ventricle was slightly but significantly increased after the incision. However, the procedure caused no

Fig.6. Epicardial activation sequence and limb lead ECG before (A) and after (B) transverse incision in the middle region of the right ventricular free wall (Experiment No.21).

significant change in the duration of the QRS of limb lead ECG.

4) Transverse incision in the outflow tract

In 5 dogs, the right ventricular outflow tract was incised transversely. A typical experiment is illustrated in Fig. 5. As shown in panel B, the activation of the outflow tract distal to the line incised was delayed by several milliseconds after the incision. In contrast, the epicardial breakthrough and the subsequent activation sequence in the right ventricular free wall as well as in the left ventricle were virtually unaffected by the incision. The S-deflexion of QRS complexes in leads II and III was augmented a little after the incision.

The results obtained from the 5 dogs are summarized in Table II. The latest activation time of the right ventricle was delayed significantly after the incision. In most experiments,

Fig.7. Epicardial activation sequence and limb lead ECG before (A) and after (B) transverse incision along the posterior interventricular groove (Experiment No.27).

the duration of the QRS complex in limb lead ECG was also prolonged slightly, but the change was statistically insignificant.

5) Transverse incision in the middle region of the right ventricular free wall

Fig. 6 shows an experiment in which the middle region of the right ventricular free wall was incised transversely. The pattern of activation sequence in the right and left ventricle as well as the configuration of QRS complex was virtually unaffected by the surgical procedure despite the long incision. Although some experiments showed a slight delay in the latest activation time of the right ventricle or a slight increase in the duration of QRS complex of limb lead ECG (Table II), these changes were statistically insignificant.

6) Transverse incision along the posterior inter-

TABLE III CHANGES OF THE LATEST ACTIVATION TIME OF RIGHT VENTRICLE AND QRS DURATION FOLLOWING TRANS-SECTION OF THE LATERAL BRANCHES OF THE RIGHT BUNDLE

Experiment No.	Latest activation time of right ventricle			QRS duration		
	Control	After incision	Difference	Control	After incision	Difference
31	33	44	11	35	46	11
32	32	48	16	40	51	11
33	30	47	17	32	50	18
34	38	51	13	40	54	14
35	36	50	14	41	52	11
Mean $\pm$ SD	33.8 $\pm$ 2.9	48.0 $\pm$ 2.4	14.2 $\pm$ 2.1*	37.6 $\pm$ 3.5	50.6 $\pm$ 2.7	13.0 $\pm$ 2.8*

Symbols are the same as in Table I.

Fig.8. Epicardial activation sequence and limb lead ECG before (A) and after (B) trans-section of the lateral branches of the right bundle (Experiment No.31).

Fig.9. Epicardial activation sequence and limb lead ECG before (A) and after (B) injury to the endocardial Purkinje network extending toward the right ventricular out-flow tract (Experiment No.36).

ventricular groove

Fig. 7 illustrates one of the experiments in which a transverse incision was made along the posterior interventricular groove. As shown in

the left side of panel B, the incision caused an activation delay of 6 to 8 msec in the restricted region between the line incised and the posterior interventricular groove. The epicardial break-

TABLE IV CHANGES OF THE LATEST ACTIVATION TIME OF RIGHT VENTRICLE AND QRS DURATION FOLLOWING INJURY TO THE ENDOCARDIAL PURKINJE NETWORK EXTENDING TOWARD THE RIGHT VENTRICULAR OUTFLOW TRACT

Experiment No.	Latest activation time of right ventricle			QRS duration		
	Control	After incision	Difference	Control	After incision	Difference
<i>RBBB (-)</i>						
36	29	32	3	36	37	1
37	35	36	1	35	35	0
38	29	35	6	36	36	0
39	24	30	6	33	34	1
40	25	28	3	33	33	0
41	33	40	7	42	44	2
42	27	30	3	37	37	0
Mean $\pm$ SD	28.9 $\pm$ 3.7	33.0 $\pm$ 3.9	4.1 $\pm$ 2.0*	36.0 $\pm$ 2.8	36.6 $\pm$ 3.3	0.6 $\pm$ 0.7
<i>RBBB (+)</i>						
43	27	63	36	34	65	31
44	29	59	30	32	64	32
45	33	63	30	36	65	29
Mean $\pm$ SD	29.7 $\pm$ 2.5	61.7 $\pm$ 1.9	32.0 $\pm$ 2.8*	34.0 $\pm$ 1.6	64.7 $\pm$ 0.5	30.7 $\pm$ 1.2*

Symbols are the same as in Table I.

through and activation sequence in the remaining part after the incision were almost the same as observed under the control condition. The incision did not influence the configuration of the QRS complexes of limb lead ECG.

In all the 5 dogs studied, the incision caused a slight but significant delay in the latest activation time of the right ventricle. Although some dogs showed a slight concomitant prolongation of QRS duration, it was statistically insignificant (Table II).

Trans-section of the lateral branches of the right bundle

In this set of experiments, the lateral branches of the right bundle were dissected blindly by using a scalpel introduced trans-atrially into the cavity of the right ventricle. Representative results are presented in Fig. 8. After the trans-section of the lateral branches, the activation sequence of the heart was seriously altered in relation to that of the control. Thus, the epicardial breakthrough appeared in the apex of the left ventricle at around 17 msec after the onset of the ventricular activation (9 msec later than

control), then the activation wave fronts proceeded toward the base of the right and the left ventricles. On the right ventricle, V-shaped isochronous lines were depicted, and the uppermost region of the inflow tract was activated at last (44 msec after the onset of ventricular activation). The trans-section also caused a concomitant change in the configuration of QRS complexes of limb lead ECG. As shown in the bottom of Panel B, the S-deflexion in leads II and III after the trans-section was remarkably augmented when compared with that of control.

Table III summarizes the results obtained from 5 experiments. All the dog hearts in which the trans-section was undertaken showed a marked delay of the latest activation time of the right ventricle as well as a prolongation of QRS deflexion by longer than 10 msec from the value of control. These results agreed with those obtained by Moore et al.¹⁹

The prolongation of QRS duration after trans-section of the lateral branches corresponded to about half of that after cutting of the main RBB in dogs, as reported previously.¹⁴ The conduction disturbance observed in this set of experi-

Fig.10. Epicardial activation sequence and limb lead ECG before (A) and after (B) injury to the endocardial Purkinje network extending toward the right ventricular outflow tract (Experiment No.43).

ments may, therefore, be comparable to the incomplete RBBB in dogs.

Injury to the endocardial Purkinje network extending toward the right ventricular outflow tract

In the third set of experiments, the endocardial Purkinje network extending toward the right ventricular outflow tract was injured indiscriminately by scratching with the tip of the Kocher's forceps which is introduced through the free wall of the right ventricular outflow tract. Two representative results are presented in Figs. 9 and 10. Table IV summarizes the results obtained from 10 experiments. In 7 out of the 10 experiments, the injury caused an activation delay limited to the outflow tract of the right ventricle as illustrated in Fig. 9. The activation sequence in the remaining area of ventricles or configuration of QRS complexes in limb lead ECG of these experiments was unaffected by the injury.

As summarized in Table IV, all of these 7 cases showed a slight but significant delay after the injury in the latest activation time of the right ventricle. However, no significant prolongation was induced by the injury in the QRS duration of limb lead ECG.

In the 3 experiments, the activation sequence of the right ventricle was completely altered after the injury. Representative results are shown in Fig. 10. Activation wave fronts of this dog proceeded from the epicardial breakthrough, which appeared in the apex of the left ventricle at around 16 msec from the onset of ventricular activation, toward the base of the right and left ventricle. The entire surface of the right ventricle was covered with dense and parallel isochronous lines indicating a serious activation delay of the whole right ventricle. An area from the uppermost region in the inflow tract toward the posterior margin of outflow tract was activated at last (at around 65 msec from the onset of ventricular activation). QRS complexes in limb lead ECG after the injury were characterized by a deep and wide S-wave. As indicated in Table IV, all of these 3 experiments showed a marked delay in the latest activation time of the right ventricle as well as a prolongation of QRS duration of over 30 msec. These alterations in epicardial activation sequence and QRS complex in limb lead ECG correspond with complete RBBB in dogs which was produced experimentally by cutting the main RBB¹⁴

DISCUSSION

There have been few experimental studies reported in which the effects of right ventriculotomy on cardiac activation sequence and ECGs are investigated.^{20,21} According to the experiments by Genender et al²¹ on dog hearts, right ventriculotomy did not affect the epicardial activation sequence of the right ventricle if the surgical incision was made either medial to the arborization of the free-running Purkinje fibers or through their arborization on the free ventricular wall. QRS of ECG was likewise unaltered. When the incision was made lateral to the area of the arborization, it caused an activation delay of over 10 msec in the area lateral to the incision, but QRS durations were unaltered even in these dogs. Our results are partially in agreement with above authors' findings. Thus, in most dogs examined (29 out of 30 experiments), right ventriculotomy caused a slight activation

delay (less than 10 msec) which was restricted to the distal area of right ventricle from the incision; but the delay was not long enough to cause a significant prolongation of QRS duration of limb lead ECG. No apparent difference was observed between the incisions in the lateral and medial area of the free wall. The activation delay tended to be somewhat more appreciable only when the vertical incision was made in the middle region of the free wall that corresponded to the area of arborization of the free-running Purkinje fibers in the experiments by Genender et al.²¹ This discrepancy between our results and those of Genender et al. on right ventriculotomy might be attributed to the different recording methods of epicardial activation and to the different extent of injury to the lateral branches of the right bundle.

Our anatomical analysis after the electrical measurements revealed that some of the lateral branches were interrupted by the incision when it was made on the middle area of the free wall in the vertical direction. In contrast, any other types of vertical or transverse incision did not cause such an interruption of the lateral branches. The most serious interruption of the lateral branches by the incision was observed in experiment Number 7, which is the only experiment showing an exceptional, marked alteration of the right ventricular epicardial activation sequence and an apparent prolongation of QRS duration (16 msec) after the right ventriculotomy. These findings suggest that right ventriculotomy would induce the ECG pattern of RBBB, provided that lateral branches are extensively injured by the surgical procedure. This assumption was supported by the experiments in which the lateral branches were selectively transsected by the scalpel introduced trans-atrially. When the lateral branches were transsected by the scalpel nearly completely, quite similar alterations to those observed in experiment Number 7 were induced in the epicardial activation sequence as well as in QRS complex of limb lead ECG. Thus, the disappearance of the right ventricular epicardial breakthrough, the activation delay of the right ventricle ranging from 11 to 17 msec, and the prolongation of QRS duration by 11 to 18 msec were appeared.

In clinical reports, the right ventriculotomy was followed by not only an incomplete but also a complete RBBB pattern of ECG.^{6,7} Nevertheless, we could produce only the incomplete pattern of RBBB with a prolongation of QRS

duration less than 54 msec, even when all the lateral branches were interrupted. This might be explained by the different anatomical architecture of conducting tissue of the right ventricle between men and dogs. The RBB of the dog's heart divides into anterior, lateral and posterior branches at the base of the anterior papillary muscle on the septum near the apex.²² Accordingly, even if all the lateral branches are interrupted, the remaining anterior and posterior branches could prevent the serious alteration of activation sequence resulting in a complete RBBB pattern of ECG. On the contrary, the RBB of human hearts courses down along the septum, and on the moderator-band as a bundle without branching.⁵ Therefore, the incision of ventriculotomy applied to the area around the arborization of the moderator-band, could induce the complete interruption of activation mediated by the RBB.

Recently, Horowitz et al.⁸ reported that the ECG pattern of RBBB was routinely observed after trans-atrial repair of TF even though no ventriculotomy was performed. Hence, they stressed the etiologic significance of the damage to the Purkinje network during the infundibular resection. In this study, an extensive injury to the Purkinje network extending toward the right ventricular outflow tract by the tip of the Kocher's forceps induced a local activation delay restricted to the area of the outflow tract which is far from the serious delay resulting in a characteristic ECG pattern of RBBB, provided that the intervention did not cause a concomitant injury to the main RBB. Accordingly, the RBBB pattern of ECG following the infundibular resection should probably be attributed to some alternative mechanism such as an injury to the hypertrophied cardiac muscle in the outflow tract or the anomalous conducting tissue of the right ventricle.

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