The Wolff-Parkinson-White Syndrome in a Holstein-Friesian Cow

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ABSTRACT. A case of Wolff-Parkinson-White (WPW) syndrome in a Holstein-Friesian cow aged 10-year-old was examined in detail. In electrocardiogram (ECG), the P-wave was the same configuration in both the normal and abnormal ECG. The PR-interval shortened from 0.2 to 0.1 second and the duration of the QRS-complex prolonged from 0.1 to 0.12 second compared with normal ECG. The delta wave, characterized in WPW syndrome, could not be recognized. In echocardiogram, notches were recognized at the early stage of ventricular contraction in the interventricular septum. This cow was, therefore, diagnosed as type B WPW syndrome. The abnormal ECG disappeared by the administration of procainamide. It was strongly indicated that the ventricular contraction showing abnormal ECG was generated only by the stimulation through an accessory pathway in this cow.—KEY words: accessory pathway, electrocardiogram, pre-excitation, WPW syndrome.


The Wolff-Parkinson-White (WPW) syndrome is characterized as follows [9]; (1) the shortening of PR-interval in electrocardiogram (ECG), (2) the extension of QRS-complex and the appearance of a delta wave in ECG, and (3) recurrent attacks of tachycardia.

Many reports concerning the WPW syndrome have been published, however, in the veterinary field, particularly in large animals, there have been only a few reports for this syndrome. Spörri first reported this syndrome in two cows in 1953 [5]. Until now six cases of this syndrome in cows have been reported [6, 8].

In this report, the WPW syndrome in a cow was examined using various cardiological skills and equipment. Then the therapeutic drugs for the WPW syndrome were administered and the mechanism of occurrence of the WPW syndrome was discussed according to changes of ECG by the administration of the drugs.

MATERIALS AND METHODS

Animals: A 10-year-old female Holstein-Friesian cow weighing 550 Kg was referred to the Veterinary Teaching Hospital in Rakuno Gakuen University because of chronic inflammation of a wound on June 10, 1987. From ECG, characteristic abnormalities were recognized. The cow was diagnosed as WPW syndrome and was examined in detail compared with normal cows. This cow is still alive and its ECG is continued to be examined. This cow was calved down, and the abnormal ECG has not yet recognized in this calf.

Five female Holstein cows (2- to 10-year-old, weighing 400 to 700 kg) were used as control.

Examination: The ECG was recorded by use of A-B lead, four limb lead and unipolar chest lead. For four limb lead, electrodes were placed on forelimbs and hindlimbs. By the use of four limb lead, lead I, II, III, aVR, aVL and aVF were recorded. Unipolar chest lead was recorded at 150 different
points in chest. Vector-cardiogram (VCG) was sketched from these recordings according to the methods used by Van Arsdel and Bogart [8]. The cardiac electrical axis was examined based on the VCG. Phonocardiogram, blood pressure and echocardiogram were recorded according to the ordinary methods.

Procaniamide, propranolol and digitalis were injected intravenously 4 times every 10 minutes. Their doses were 0.5, 10, 1.25 mg, respectively. One mg of isoproterenol and 5 mg of atropine sulfate were injected subcutaneously. Atropine was administered 4 times every an hour. Twenty g of quinidine were administered orally 3 times every 2 hours for. Electrocardiogram was recorded before and after administration of each drug.

RESULTS

Electrocardiography: Figure 1 represents the ECG obtained by the A-B lead in WPW syndrome cow. The peculiarities of the ECG were as follows; (1) alternate appearance of the normal and abnormal ECG, (2) shortened PR-interval from 0.2 to 0.1 second, (3) prolonged QRS-complex from 0.1 to 0.12 second, and (4) constant PP-interval in both normal and abnormal ECG. In addition, the frequency of appearance of the abnormal ECG was higher. The definite rhythm of appearance of the abnormal ECG was not recognized. The abnormal ECG was not converted into the normal by the ocular cardiac reflex or 10-minute fast walk.

The ECG obtained by four limb lead is shown in Fig. 2. The configurations of QRS-complex varied markedly each other.

![Electrocardiogram by A-B lead in WPW syndrome cow.](image1)

![Electrocardiogram by four limb lead.](image2)
The QRS-complex of the abnormal ECG showed greater positive potential and longer duration than that of the normal one. The contrary deflection of the T-wave in the abnormal ECG was recorded compared with the normal one.

The VCG is illustrated in Fig. 3. The mean QRS vector of the abnormal VCG deviated toward the left and its area was larger than those of the normal one. There was a slowing down of the writing of the QRS loops pointing away at the sagittal and horizontal phases during the early ventricular depolarization of the abnormal VCG. No change of the VCG in the normal ECG was observed.

**Phonocardiography:** The first sound in the abnormal ECG was louder and more echoed than that in the normal. That of normal ECG commenced near the middle of the QRS-complex. The interval between the onset of the R-wave and the first sound was 0.04 second in the normal. On the other hand, the first sound in the abnormal ECG commenced near the terminal point of the QRS-complex and its interval between the R-wave and the first sound was 0.08 second.

**Blood pressure:** The aortic pressure (AOP) and the right ventricular pressure (RVP) are shown in Fig. 4. At the onset of the left shoulder of the RVP in abnormal ECG, there was an upward-slr. And a small dip between the main peak and the right shoulder was presented in the normal ECG but not in the abnormal ECG. The first positive wave in the RVP of the abnormal ECG began earlier than that of the normal ECG. In contrast, it began later in the AOP of the abnormal ECG than that of the normal ECG.

**Echocardiography:** The enlarging rates of the left ventricle at the left ventricular posterior free wall were 15.0 cm/sec in the abnormal ECG and 6.0 cm/sec in the normal ECG. The onset of the systole of the left ventricular free wall was later in the abnormal ECG than in the normal ECG. In the movement of the interventricular septum, there appeared a notch in the abnormal ECG at the early stage of ventricular contraction (Fig. 5).

**Administration of Drugs:** By the administration of procainamide, the abnormal ECG disappeared (Fig. 6). The abnormal ECG disappeared completely at 20 minutes after the administration. No fusion form of both ECG was recognized. No changes were recognized in control cows.

![Diagram of Vectorcardiogram](image)

*Fig. 3. Vectorcardiogram of abnormal and normal wave. X(+) left, X(-): right, Y(+): caudal, Y(-): cranial, Z(+): ventral, Z(-): dorsal*
Fig. 4. Aorta pressure (AO) and right ventricular pressure (RV) in WPW syndrome cow. Electrocardiogram was recorded by A-B lead.

By the administration of isoproterenol, heart rates of the WPW syndrome and control cows increased markedly and RT-intervals became short. The changes of RT-interval of the WPW syndrome and control cows were almost identical. Although PR-interval of the control cows shortened with the increase of the heart rates, that of the WPW syndrome cow remained unchanged (Fig. 7).

The PR-interval of the WPW syndrome cow shortened successively from 0.1 to 0.05 second after the administration of propranolol or digitalis (Fig. 8). No change was observed in the control cows. After the administration of digitalis in the WPW syndrome cow, normal ECG was recognized and its PR-interval was 0.2 second.

By the administration of atropine, no ECG change was observed both in the WPW syndrome and control cows. Quinidine could not convert the abnormal ECG to the normal but induced the deflection of
T-wave and RT-interval increase.

DISCUSSION

The WPW syndrome is now explained by the accessory pathway theory [2]. In general, systolic signals are conducted through atrioventricular (AV) node and Purkinje fibers. However, if another conduction route exists, the signals could be transmitted to the ventricle without the AV nodal delay which is the longest delay in all cardiac conduction system. The signals transmitted
through this route will spread over the ventricle at the normal conductive velocity. On the other hand, the normal cardiac conduction system also transmit the signals of which velocity decreases in the AV node. And the velocity of the signals increases in the Purkinje fibers, resulting in the early depolarization of the ventricle. Consequently, the QRS-complex in the WPW syndrome consists in a fusion wave; an early part of the QRS-complex, called a delta wave, is the depolarization inducing the signal through the accessory pathway, and a latter part is the depolarization inducing the signal from the normal conduction system.

In order to record a delta wave, we carried out extensive electrocardiographic recording at 150 different points in chest with unipolar leads but could not recognize it (data was not shown). In VCG, a slowing down of the writing of the QRS loops (dots are closer together) during the early depolarization seems to be relevant to the delta wave.

The degree of shortened time of PR-interval in this cow was different from human patient with the WPW syndrome. Butterworth et al. [1] reported that the fusion wave in human ECG derived from two different signals for excitation depended on the duration of QRS-complex. Two different signals were action potential through internodal atrial pathway and accessory pathway. If the time required for transmission of action potential from sinoatrial node to ventricle was longer than the period of ventricular excitation by the signal through accessory pathway, the fusion wave was constructed. Briefly, when PR-interval was shorter than the duration time of QRS-complex, the fusion wave was not generated [1]. In this cow, when the activity through the normal cardiac conduction system reached to the ventricles, the ventricles might be already in the absolute refractory period. Therefore, the excitation of the ventricles might be activated by only the stimulation signal through the accessory pathway. And fusion wave (delta wave) might not be recognized.

As shown in the results, the electrical axis of the QRS-complex deviated to the left. Thus the right ventricle might depolarize before the left one. Besides, the first heart sound delayed in the abnormal ECG, and the beginning time of increasing of blood pressure of the right ventricle in the abnormal ECG was earlier than that of the left. Furthermore, echocardiogram showed a notch on the interventricular septum which has been said to appear in the earlier contraction of the right ventricle [3, 7]. From these results, it was strongly indicated that the contraction of the right ventricle began earlier than that of the left. And the cow herein reported seemed to be the type B WPW syndrome [4].

The abnormal ECG of this cow disappeared by the administration of procainamide. In contrast, by the administration of propranolol and digitalis, abnormal ECG did not disappear and produced considerable shortening of the PR-interval of the abnormal ECG. By the administration of isoproterenol, heart rates increased 180 beats per minutes, but PR-interval remained constant in the abnormal ECG. Based on these data, the configuration of ECG in this cow might not be affected by β adrenergic effect. Therefore, the accessory pathway was not controlled by the sympathetic nervous system.

Reports for the WPW syndrome of cows were very few. WPW syndrome in cow reported in this study was characterized as follows; (1) the form of QRS-complex was quite different from normal ECG, (2) delta wave did not appear, (3) PJ-interval (from the beginning of P wave to the end of QRS complex) shortened, and (4) shortening rate of PR-interval was larger than human case. Further investigation should be made to
elucidate the mechanism of transmission of
the signal for excitation.

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要約

ホルスタイン牛のWolff-Parkinson-White症候群の一例：遠藤康浩・田島毅士・黒沢隆・高橋清志・其田
三夫（知農学園大学動物外科学教室）——Wolff-Parkinson-White（WPW）症候群の特徴を示す10歳のホルス
タイン牛に、心電図、心音図、血圧、心エコー図検査および薬物投与試験を行い詳しく検討した。心電図では、P
波は正常波形時および異常波形時とも同様であった。PR間隔は正常波形時0.2秒であったのに対し異常波形時には
0.1秒に短縮した。一方QRS群持続時間は正常波形時0.1秒であったのが異常波形時には0.12秒に延長した。
心電図上Delta波は認められなかったが、心エコー図検査ではB型WPW症候群の特徴と言われているノッチ
が認められた。また、異常波形は塩酸プロカインアミド投与により消失した。以上のことから、この牛の心電図
異常波形時における心室の収縮は副伝導路経由の刺激によってのみ惹起されることが強く示唆された。