Case Report

An unusual disappearance of right bundle branch block by digitalis in a child with cretinism is described. A transient occurrence of right bundle branch block pattern in V1 and left bundle branch block pattern in V2 and V3 is also observed in the same case, and the possible mechanisms behind these findings are discussed.

Low voltage of the QRS complexes, flattening or inversion of the T waves and low or flat P waves have been commonly noted in myxedema. Arrhythmias are only occasionally observed. Korth and Schmidt noted right bundle branch block (RBBB) in all of 10 myxedematous females as well as the changes usually described. There are considerable data indicating that electrocardiographic changes observed in myxedema may revert to normal following thyroid therapy. However, it has never been previously reported, to our knowledge, that RBBB in myxedema was disappeared after digitalization. It is the main purpose of this report to describe such unusual case. In addition, a complex arrhythmia, possibly a transient posterior hemiblock, was also observed in this case.

Case Report

A 3-year-old girl was referred to the Yamato City Hospital for evaluation of physical and mental retardation. The mother's pregnancy and delivery was normal. The birth weight was 3200 grams. Exchange blood transfusion was performed because of severe hyperbilirubinemla of the newborn. The child was observed at another hospital from birth to the time of referral, but definitive hormonal diagnostic studies were never performed. A brother is also suspected to have almost the same physical and mental retardation.

Physical examination revealed a poorly developed and poorly nourished girl, who was 69 cm in height and 7370 grams in weight. The circumferences of the chest, the head, the abdomen, the upper limb and lower limb were 43.5 cm, 44 cm, 38.5 cm, 12 cm and 15 cm, respectively. All these measurements are equivalent to those of a 6-or-7-month-old baby. There were an enlarged and protruding tongue, and an infantile naso-orbital configuration. The skin was dry and cool. The cervical veins were moderately extended. No cyanosis or clubbing was observed. The heart was enlarged to the anterior axillary line and a Grade 2/6 soft, ejection, systolic murmur was heard at the second, left intercostal space; there was no pericardial friction rub. The abdomen was large and protuberant; the edge of the liver was felt 3 cm from the costal margin.

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below the right costal margin. Head control, rolling over, speaking, sitting alone, and walking alone were considerably disturbed. The temperature was 37.2°C, the puls 94 and the respiration 56. The hematocrit was 35 per cent, and the white-cell count 16,000. Serum electrolytes were normal. This child was suspected to have moderate degree of congestive heart failure complicated by infection of the upper respiratory tract. Then, penicilnine was given intramuscularly and digitalization was initiated.

An electrocardiogram (Fig. 1), recorded at admission, revealed sinus arrhythmia at an average rate of 90 per minute, with an axis of +190 degrees. In the first and the last one third of the tracing of V1, V2 and V3, the QRS complexes showed RBBB pattern, though those in V2 and V3 looked atypical as RBBB. In the middle one third of the tracing, there was a sudden change in QRS configuration in V2 and V3, showing deep and slurred S-waves. However, the QRS deflections in V1 remained almost unchanged, though the R waves were only slightly decreased in amplitude. After a run of this arrhythmia the QRS complexes returned to the previous RBBB pattern. In V4, V5 and V6, the dominant QRS configuration revealed the RBBB pattern, while the 4th and 5th beats were thought as supra-ventricular premature beats with aberrant ventricular conduction.

Fig. 2 showed an electrocardiogram on the third hospital day, when digitalization was considered to be fully saturated and a maintenance dose of digitalis was being administered. As can be seen in this tracing, RBBB patterns were completely disappeared with an average rate of 60 per minute. The QRS axis was not changed. The chest leads revealed sinus tachycardia and incomplete A-V dissociation in the presence of second degree of A-V block. This combination of arrhythmias are not uncommon as a consequence of digitalis therapy. Digitalis was discontinued just after the second ECG tracing was taken, because the arrhythmias as mentioned above were considered as a manifestation of digitalis intoxication. Fig. 3 was an electrocardiogram on the 13th hospital day; the QRS configurations were essentially the same as those in Figure 2, while the heart rate was much faster.

During the period of this ECG study, thyroid therapy was not initiated because no confirmative laboratory data could not be obtained. Soon after the third ECG recording, the child was diagnosed as cretinism on the basis of clinical manifestations and the following laboratory data; the total cholesterol was 380 mg, the PBI 0.4 µg, and T4 1.1 µg per 100 ml; T3 was 15.8 per cent. The clinical status of this child has been remarkably improved with thyroid medication and has still been under our observation.

**Discussion**

Advanced myxedema is commonly associated with cardiac disorders such as cardiac enlargement, heart failure and electrocardiographic abnormalities. Among electrocardiographic abnormalities, low voltage of the QRS complexes, flattening or inversion of the T waves and low or
flat P waves are the most constant features. Cardiac arrhythmias are occasionally observed in some patients; sinus bradycardia, intraventricular conduction defect and heart block are counted with higher incidence. Unfortunately, less is known about the pathogenesis of all these electrocardiographic abnormalities, but their rapid regression to normal following thyroid therapy may indicate, to lesser or greater extent, a direct relationship to the myxedema itself. Heart block and intraventricular conduction defect has been attributed to mucinous infiltration of the bundle of His. At admission, this patient was suspected as having congestive heart failure and then digitalization was performed. During digitalization, hepatomegaly was improved and urine volume

Fig. 2. ECG's were taken on May 14, 1971. This tracing was recorded when this patient had been on digitalis for three days since admission. The heart rate is about 60 per minute. The frontal axis is about +190 degrees. Note that RBBB pattern is not seen in these tracings. Sinus tachycardia and incomplete A-V dissociation are seen in the presence of second degree A-V block.

was increased. Electrocardiogram recorded at admission revealed RBBB, and a complex arrhythmia, which will be discussed later. The ECG, recorded three days after the initiation of digitalization, showed complete disappearance of RBBB and the arrhythmia. These findings are not clearly understood from the general knowledges of myxedematous heart. The pharmacological effects of digitalis are generally believed not to improve mucinous infiltration of stimulus conduction system caused by myxedema. As can be seen in Fig. 1 and 2, the heart rate was faster before digitalization than after digitalization. At this moment, the rapid heart rate was simply thought as the cause of RBBB in this case. Of particular interest, however, it was demonstrated in the third ECG tracing (in Fig. 3) that RBBB pattern was not found and the heart rate was faster than in the first tracing (in Fig. 1). Consequently, “heart rate dependent RBBB” can safely be eliminated from the possible pathogenesis.
Fig.3. ECG's were taken on May 24, 1971, when this patient had been on thyroid therapy and no digitalis medication was administered. The heart rate is about 136 per minute. No RBBB pattern is seen in any leads.

The rapid disappearance of RBBB after reversal of the heart failure by digitalis would suggest that digitalis might play an important role in the improvement of the conduction system. If the intra-cellular edema in the conducting system was, if not totally, involved in disturbances of impulse transmission, it is not certain but possible that digitalis reduced the intra-cellular edema and regained the normalization of impulse transmission through the conducting system.

Another focus of the present study was, as shown in Fig. 1, a sudden change in QRS pattern. In V1, the configuration of QRS complexes during the run of this arrhythmia was essentially the same as that of the dominant QRS complexes, though the amplitude of these QRS complexes was slightly lower. On the contrary, in V2 and V3, the main QRS deflection during this arrhythmia was shifted in an opposite direction to that in the dominant rhythm, representing left bundle branch block (LBBB). Unfortunately, since this arrhythmia was not seen in other leads, detailed analysis of this arrhythmia was greatly limited. With this lack of information in mind, however, an attempt was made in a search for a reasonable interpretation of this arrhythmia.

The R-R interval and the P-R interval were almost the same through the first ECG tracing. It would appear, therefore, that the whole rhythm was the sinus origin and also, the heart rate might have no etiological relationship with the occurrence of these QRS complexes on question. If the posterior branch of the left bundle of His was blocked (LPH), the posterior part of the left ventricle would be delayed in activation. In the presence of RBBB, the activation of the right ventricle would be also delayed. When both RBBB and LPH coexisted, it can be easily estimated that the activation of the left ventricle governed by the posterior branch of His would precede, simultaneously occur with, or follow that of the right ventricle. With the above mentioned possibility in mind, many different patterns of mixture of RBBB and LBBB could be expected. Therefore, a pattern such as RBBB appearing in V1 and LBBB in V2 and V3, could be possibly expected, when the coexistence of RBBB and LPH was suspected in this case. Quite recently, Rosenbaum and his associates reported the syndrome of RBBB with intermittent left anterior and posterior hemiblock and also they described a variety of ventricular trifascicular blocks. Some of their cases look very similar to this case. Unfortunately, since no information was obtained about the QRS axis in this case when the chest lead ECG’s were recorded, no conclusive interpretation of this arrhythmia is suspended.

REFERENCES