Two Cases of Bilateral Bundle Branch Block with Prompt Transitions in One and Gradual Transitions in the Other Case

KIMIAKI NAKAMOTO

The cause of heart block is usually obscure. In irreversible cases, it may be due to fibrous, calcific, degenerative, ischemic, congenital, neoplastic or traumatic lesions in the conduction system. In reversible cases, it may be due to neurovegetative, inflammatory, medical, metabolic or toxic effect on the conduction system. Recently, histologic study of LENÈGRE has established that in the great majority of the cases, complete heart block is caused by bilateral bundle branch block (BBB) rather than block in the atroventricular (A-V) node or in the common bundle. Also careful anatomic studies of YATER and associates have shown that in about 20 per cent of the clinical cases of complete A-V block, interruption in the continuity of both branches of the bundle of His could be considered responsible. According to LENÈGRE, however, no particular lesion could be found either in the heart itself or in the main coronary arteries in which the BBB was the site of major lesions. The lesions of the intraventricular conduction pathways were then considered to be “primary or idiopathic”. As to the etiology of BBB, it was reported by LENÈGRE that although half the cases of major lesions of the BBB branches were associated with severe coronary atherosclerosis, these lesions occurred with no apparent cause in a high proportion of cases (30%). His histological examination of the intraventricular conduction pathways disclosed that the A-V node was normal in 52, and the common bundle in 26 of 62 cases examined. However, the right bundle branch was never found to be normal. Among 28 cases of chronic A-V block examined by LENÈGRE, 2 cases had severe muscular dystrophy, 5 cases arterial hypertension and 2 cases diabetes mellitus, but 9 cases had no apparent underlying cardiac cause. Therefore, as he stated, if one considers that isolated arterial hypertension without coronary atherosclerosis, diabetes mellitus and chronic respiratory disease are merely concomitant but not etiologic factors in the lesions of the intraventricular conduction pathways, the proportion of “primary” blocks due to bilateral bundle branch lesions without obvious ischemia and changes of walls of the heart would amount to 18 of 28 cases (64%). LENÈGRE has stressed that although permanent complete A-V block is always associated with organic lesions of the conduction system, the amount of involvement of the conduction system is not necessarily correlated with the amount of block. ROSSI found lesions not only in the conduction system but also in the nerves, and JAMES is of the opinion that restrictive lesions in vessels supplying the A-V node and bundle in the absence of histologic changes in the latter may be sufficient to produce block.

According to HARRIS and associates, a QRS complex of a RBBB pattern was observed in 58, that of a LBBB pattern in 12 and QRS complexes of varying right and left bundle branch block patterns in 2 of 81 cases of complete A-V block. BOYADIAN and VAN DOOREN defined BBBB as the combination of the signs of both right and left intraventricular blocks. It was characterized

(Received for Publication, February 12, 1970) Second Department of Internal Medicine, Yamaguchi Red Cross Hospital, Yamaguchi

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by the simultaneous delay of intrinsicoid deflections in $V_1$ to $V_2$ on one side and in $V_5$ to $V_6$ on the other. According to Rosenbaum and Lepeschkin, the form of the ventricular complex in BBBB is determined by the branch with the greater degree of block because the stimulus reaches the ventricles through the less affected branch, and the conduction delay in the branch with less degree of block determines the lengthening of the PR interval. It is also conceivable that varying ventricular complexes in cases of complete A-V block can be a result of changes in the location of the A-V nodal or ventricular pacemaker. Stein and associates emphasized the hazard of complete A-V block during cardiac catheterization of patients with pre-existent bundle branch block. They indicated that a catheter-induced bundle branch block with a pre-existent contralateral bundle branch block would produce complete heart block in the form of BBBB. The same phenomenon can be observed when, as a result of neurovegetative, inflammatory, medical, metabolic or toxic effect on the heart, first or second degree A-V block occurs in one bundle branch of the heart with pre-existent contralateral bundle branch block.

The electrocardiographic criteria of incomplete BBBB by Sodi-Pallares and associates include absence of Q waves in leads facing the left ventricle, absence of R waves in $V_1$, and a QRS complex of duration of 0.08 to 0.11 seconds. According to Schamroth and Bradlow, however, it is necessary for the diagnosis of incomplete bundle branch block to demonstrate tracings with normal intraventricular conduction, subsequent tracings that show various degrees of
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incomplete bundle branch block and tracings that eventually show complete bundle branch block in the same patient during a short time interval.

In Japan, 2 cases of BBBB were first reported in 1965 by Hashida, Maekawa and Shirai. One of the 2 was associated with transient and the other with permanent A-V block. It appears that BBBB is not uncommon as has been considered because in a period of 5 months (from November, 1968 to March, 1969) 3 cases of BBBB have been observed in our clinic, 2 associated with A-V block, and 1 with A-V dissociation. In this paper, 2 cases of BBBB with A-V block are presented. One was associated with prompt transitions, and the other with gradual transitions between the 2. The latter case showed a QRS complex of normal conduction, accelerated conduction, incomplete LBBB and incomplete RBBB during transitions. The case of BBBB associated with A-V dissociation will be reported separately because it was combined with congenital heart disease.

![Fig.2. Rate and location of the idioventricular pacemaker](image)

When the ventricular rate was between 28 to 32 per minute with LBBB, the location of the pacemaker may be higher than when it was between 18 to 25 per minute with RBBB. For example, when the pacemaker was located at A in the former, it was located at B in the latter or at A' in the former and at B' in the latter or at A in the former and at B' in the latter.

![Fig.3. Left bundle branch block with 3 : 1 A-V block](image)

The atrial rate was ca. 105 per minute, and the ventricular rate ca. 35 per minute. While the PR interval varied from 0.44 (V₆) to 0.56 (V₅) second, the RR interval was always about three-fold of the PP interval. This indicates the presence of 3 : 1 second degree A-V block, regardless of the variation in the PR interval.
Case Reports

Case 1, Z. K., a 29-year-old unmarried police official.

On the evening of November 27, 1968, he first visited the Mitoh Co-operative Hospital with chief complaints of abdominal discomfort, general malaise and light-headedness. Outstanding features on that day were pale face and marked bradycardia. The heart rate was ca. 30 per minute, and the blood pressure 110/60 mmHg. He was a police sergeant and a chief of a policebox in Mitoh area. He was preparing himself for a pass examination. He studied far into the night, and his short sleep was frequently interrupted by a traffic accident in the district of his jurisdiction. His electrocardiogram recorded on March 23, 1966 showed stabilized anteroseptal infarction (a QS wave in V3). However, he could not recall any episode of intolerable precordial discomfort. At that time, he had been subjected to special physical training.

The white blood cell count was 7,700 with 55 per cent neutrophiles, the red blood cell count $428 \times 10^4$ with 88 per cent hemoglobin, and erythrocyte sedimentation rate 1.5 mm in 1 hour. Serum proteins were 6.3 gm per 100 ml with an A/G ratio of 2.15, serum cholesterol 196 mg per 100 ml, GPT 14, GOT 11 Sigma-Frankel units, and cobalt reaction Co R4(5). Both C-reactive protein and RA-test were negative, and ASLO 125 Todd units. Prednisolone was given 40 mg a day, Orciprenaline (Alotec) 80 to 100 mg, Chlordiazepoxide 40 mg and Diprydamole (persantin) 100 to 150 mg. Extractum scopoliae 0.08 to 0.10 gm a day was added later, which seemed to be most effective.

The electrocardiogram on November 27, 1968 (Fig. 1) showed complete A-V block with QRS complexes of both LBBB and RBBB patterns. In this case, transitions between the 2 were prompt, and there occurred no incomplete bundle branch block patterns during transitions. A normal QRS complex rarely appeared without any regularity. The atrial rhythm was regular, and its rate ca. 105 per minute. The PR interval varied from 0.08
to 0.50 second. The ventricular rhythm was irregular, and its rate ranged between 25 to 30 per minute. Although the P wave and the QRS complex were so arranged as if there were 6 : 1, 5 : 1, 4 : 1 and 3 : 1 A-V blocks, some of the PR intervals (1.88, 2.04, 2.12, 2.16, 2.18, 2.40, 2.44, 2.68, 3.04 and 3.40 seconds) were not multiples of the PP interval of ca. 0.56 to 0.60 second. The RR interval was 2.44 to 3.40 seconds (18 to 25 beats per minute) with a RBBB pattern and 1.88 to 2.04 seconds (28 to 32 beats per minute) with a LBBB pattern. This indicates, as shown in Fig. 2, that the QRS complex of a LBBB pattern originates in the upper region, and that of a RBBB pattern in the lower region of the conduction system because the rate of the idioventricular pacemaker offers a clue to the location of this pacemaker. It was reported by Lenègre⁹ that the ventricular rate was below 37 in 12 of 15 cases of complete A-V block due to complete destruction of both branches and above 46 in the remaining 2 cases. The origin of the pacemaker may change from one bundle branch or region of the ventricle to the other as a result of bilateral irritation. In such cases, the QRS configuration should not necessarily be typical of bundle branch block, and there should be considerable changes in rate during the transition.¹⁰ On November 28 (Figs. 3 and 4), all the QRS complexes showed a LBBB pattern with 3 : 1 A-V block and a PR interval of 0.36 to 0.48 second. This increase in the PR interval may be the result of conduction delay not only in the A-V node and common bundle but also in the right bundle branch. The atrial rate was ca. 105 per minute, and the ventricular rate ca. 35 per minute. On November 29 (Fig. 5), all the QRS complexes showed a RBBB pattern with
Subcutaneous injection of 0.5 mg of Orciprenaline increased the heart rate from 45 to 52 per minute by increasing the sinus rate from 90 to 104 per minute. The PR interval was shortened from 0.18 to 0.16 second, but 2:1 A-V block remained unaltered. This indicates that although the A-V conduction was slightly improved by Orciprenaline, the refractory period of the A-V conduction system was still longer than the shortened PP interval.

first degree A-V block with a PR interval of ca. 0.24 second. There was no second degree A-V block. On November 30 (Fig. 5), a QRS complex showed a RBBB pattern with first degree A-V block with a PR interval of 0.26 to 0.32 second. It was associated with intermittent 2:1 second degree A-V block. From then on, complete RBBB became permanently established. Intermittent 2:1 A-V block occurred spontaneously or could be experimentally induced (Figs. 7, 8 and 10).

Conduction Disturbances Experimentally Produced

Subcutaneous injection of 0.5 mg of Orciprenaline increased the sinus rate from 89 to 104 per minute, but 2:1 second degree A-V block remained unaltered (Fig. 6). An interval from the P wave conducted to the P wave blocked was always slightly less than that from the P wave blocked to the P wave conducted.

Hyperventilation induced transient 2:1 second degree A-V block (Fig. 7). The atrial rate was increased from 74 to 89 per minute. This increase in the atrial rate may be one of the causes of A-V block. MCHENRY and KNOEBEL reported a case in which acceleration of the sinoatrial rate gave rise to complete heart block and ventricular asystole. The changes in the contour of the P wave may be due either to changes in the intrathoracic pressure, wandering of the pacemaker or to changes of the heart position by hyperventilation.

The Valsalva maneuver also induced transient 2:1 second degree A-V block (Fig. 8). The atrial rate was increased from 70 to 90 per minute. After the maneuver, the atrial rate was decreased to 61 per minute, and 1:1 A-V conduction resumed. Also in this occasion, an increase in the atrial rate was one of the causes of 2:1 A-V block. The effects of hyperventilation and the Valsalva maneuver on S-A and A-V conductions were less on March 18 than on February 5. Second degree A-V block was not induced on March 18 by the Valsalva maneuver and hyperventila-
Fig. 7. Effect of hyperventilation
Hyperventilation induced 2:1 second degree A-V block. This indicates an abnormally elevated susceptibility of the A-V conduction system due to changes in the balance of the autonomic nervous system. Arrows show the beginning and end of hyperventilation. The atrial rate during hyperventilation was ca. 90 per minute and ca. 75 per minute before and after hyperventilation. Namely, a PP interval which was shorter than the refractory period of the A-V conduction system caused second degree A-V block.

Fig. 8. Effect of the Valsalva maneuver
The Valsalva maneuver also induced 2:1 A-V block. The atrial rate during the maneuver was ca. 89 per minute and 72 per minute before and after the maneuver. Also in this occasion, a shortened PP interval which was shorter than the refractory period of the A-V conduction system caused A-V block.

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Fig. 9. Effect of pressure on the eyeballs
Following compression of the eyeballs, the PP interval was prolonged as long as 2.32 seconds. The heart rate decreased from ca. 63 to ca. 40 per minute. This exaggerated oculocardiac reflex indicates a markedly increased irritability of the cardiac vagus. The PR interval remained nearly unaltered, and there occurred no second degree A-V block. This indicates that the pressure on the eyeballs mainly suppressed impulse formation in and impulse propagation from the sinus node (sinus arrest or various degrees of S-A block).

Fig. 10. Spontaneous occurrence of 2 : 1 A-V block
Spontaneous increase in the atrial rate from 74 to 84 per minute induced 2 : 1 second degree A-V block. The atrial rate was 74 per minute during 1 : 1 A-V conduction and 84 per minute during 2 : 1 A-V conduction. Spontaneous development of 2 : 1 A-V block indicates that the PP interval has temporarily become shorter than the refractory period of the A-V conduction system.
Fig. 11-a. Bilateral bundle branch block with A-V block

Following compression of the eyeballs, the PP interval was prolonged as long as 2.23 seconds (Fig. 9). The heart rate was decreased from 63 to ca. 40 per minute. This exaggerated oculocardiac reflex indicates a markedly increased irritability of the cardiac vagus. As shown in Fig. 9, the Aschner’s phenomenon was not accompanied by A-V block.

Spontaneous increase in the atrial rate from 74 to 84 per minute induced 2:1 second degree A-V block. The atrial rate was 74 per minute during 1:1 A-V conduction and 84 per minute during 2:1 A-V conduction (Fig. 10).

It must be mentioned that the patient did not present clinical symptoms of myocardial infarction, inflammation, metabolic diseases and drug intoxications preceding or during the period when these electrocardiograms were recorded, and laboratory studies were of little diagnostic help except for the electrocardiograms.

Case 2, T. N., a 68-year-old widow.

She had been visiting the out-patient clinic of Yamaguchi Red Cross Hospital for the treatment of hypertension and anxiety neurosis. Her electrocardiogram had shown complete RBBB with regular sinus rhythm. She lived with her daughter, her adopted son (daughter’s husband) and a grandson. Her daughter was a business woman, and the patient had to take care of her grandson daily. The patient and her daughter’s husband were always on bad terms and avoided each other. The first episode of complete A-V block occurred on September 2, 1968. She was hospitalized from September 11, 1968. The attendant physician of the first admission interpreted the electrocardiogram as complete A-V block with LBBB and ventricular extrasystoles. The electrocardiogram actually showed complete A-V block with BBBB. She was hospitalized again from February 13 to March 9, 1969. A few days before the second admission, she was shocked when she saw her daughter’s husband drunken throw her grandson down on the ground. She complained of nausea, dizziness and tightness in the throat. The pulse rate was ca. 40 per minute with a regular rhythm.

The white blood cell count was 4,300, and the red blood cell count $430 \times 10^4$ with 73 per cent.
hemoglobin. The erythrocyte sedimentation rate was 6 mm in 1 hour. GPT was 12, GOT 18 Sigma-Frankel units, LDH 270 units (normal range: 50 to 400 units), and HBD 130 units (normal range: 38 to 166 units). Both C-reactive protein and RA-test were negative, and ASLO 12 Todd units. Prednisolone was given 30 mg a day, Orciprenaline (Alotec) 80 mg, Chlordiazepoxide 30 mg, and Dipyriramole (Persantin) 150 mg. Extractum scopoleia 0.06 to 0.08 gm a day was added later, which seemed to be most effective.

The electrocardiogram on February 13, 1969 showed complete A-V block with LBBB and 2:1 A-V block with RBBB (Figs. 11-a and 11-b). Transitions from LBBB to RBBB and vice versa were gradual, and the QRS complex during transitions showed an intermediate form or a fusion beat such as the QRS complexes of normal conduction, accelerated conduction, incomplete LBBB and incomplete RBBB. The atrial rate was ca. 72 per minute regardless of the pattern of the QRS complex. The ventricular rate was ca. 32 per minute when the QRS complex showed a LBBB pattern and ca. 36 per minute when the QRS complex showed a RBBB pattern. When the QRS complex showed a LBBB pattern, A-V block was complete, and when it showed a RBBB pattern, A-V block was incomplete with 2:1 A-V conduction. This indicates that the atrionodal junction or the beginning of the left bundle branch was also blocked in LBBB, and the A-V nodal pacemaker was made to escape. In transitions from complete RBBB to complete LBBB or the reverse, there was an enhancement of conduction in that bundle branch in which no conduction had occurred (Figs. 12 and 13). On February 14 (Fig. 14), the QRS complex showed only a LBBB pattern. The atrial rate was ca. 83
Two cases of bilateral bundle branch block

When 2:1 A-V block was present with complete RBBB, the atrial rate was ca. 75 per minute, and the ventricular rate ca. 37.5 per minute. When complete A-V block was present with atypical LBBB, the atrial rate was ca. 74 per minute, and the ventricular rate ca. 37.5 per minute. However, the atrial rate was ca. 72 per minute at the beginning and ca. 80 per minute at the end of complete A-V block with atypical LBBB. Nevertheless, the ventricular rate remained unaltered at ca. 37.5 per minute regardless of the presence of 2:1 A-V block with RBBB or complete A-V block with atypical LBBB. The fluctuation in the atrial rate caused complete A-V block to appear in the form of nodal escape rhythm. During the transition, an impulse spread through both right and left bundle branches and thus produced the QRS complexes of normal conduction, accelerated conduction, incomplete LBBB and incomplete RBBB.

Fig. 12. Impulse propagation in bilateral bundle branch block

per minute in 2:1 A-V block and ca. 77.5 per minute in complete A-V block. This slight difference in the atrial rate might have been responsible for the transient 2:1 A-V block observed during esophageal leads. The ventricular rate was ca. 42 per minute in both complete and incomplete A-V block. A-V block was complete when the atrial rate was ca. 94 per minute, and the ventricular rate ca. 37 per minute. Namely, 1 RR interval (1.44 seconds) was slightly less than 2 PP interval (1.48 seconds). As mentioned above, McHenry and Knoebel12 reported a case in which acceleration of the S-A rate was documented as an unusual mechanism giving rise to complete heart block and ventricular asystole. They explained this phenomenon on the basis of an abnormal prolongation of the refractory period of the A-V junctional tissues. Namely, when the maximal rate at which the A-V junctional tissues could transmit impulses was exceeded, complete A-V block ensued. Complete A-V block with a QRS complex of a LBBB pattern and an atrial rate of more than 90 per minute continued from February 14 to February 19 for 6 days (Fig. 15). From February 24, combinations of 2:1 A-V block with complete RBBB and complete A-V block with complete LBBB with gradual transition between the 2 continued until March 3, 1969. On March 7 (Fig. 16), the electrocardiogram showed 2:1 A-V block with a complete RBBB pattern only. On March 8 (Fig. 16), it showed first degree A-V block with a PR interval of ca. 0.40 second associated with a QRS complex of a complete RBBB pattern. The PR interval was normal (0.16 second) in 2:1 A-V conduction and prolonged (0.40 second) in 1:1 A-V conduction. The atrial rate was 82 per minute in 2:1 A-V conduction and 71 per minute in 1:1 A-V conduction. This indicates that the PP interval was shorter than the refractory period of the A-V conduction system in 2:1 A-V conduction.13 Also in this case, S-A
Fig. 13. Locations of block, pacemaker and impulse pathways
If the pre-existent complete RBBB is permanent, the block must be located above the A-V nodal pacemaker because an impulse from the A-V nodal pacemaker must reach the left ventricle by way of the right bundle branch for LBBB to occur. If the block is present below the A-V nodal pacemaker, an impulse from it can not spread through the right bundle branch to the left ventricle.

Fig. 14. A-V Block dependent on the atrial rate
On February 14, the ventricular rate was 42 per minute with regular rhythm. When the atrial rate was ca. 83 to 84 per minute, A-V block was incomplete with 2 : 1 A-V conduction (1st strip). When it was 78 to 80 per minute, A-V block was complete (2nd, 3rd and 4th strips). However, the 2 : 1 A-V block in E35 (1st strip) may be merely in appearance as a result of 2 : 1 arrangement of the P wave and the QRS complex simulating 2 : 1 A-V block.

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Fig. 15. A faster atrial rate with a slower ventricular rate
On February 19, the atrial rate was ca. 94 per minute, and the ventricular rate ca. 37 per minute. Namely, the faster the atrial rate was, the slower was the ventricular rate. The same phenomenon was observed also in case 1 on November 27 and 28. These observations indicate that sinus acceleration is one of the causes of A-V block.

Fig. 16. Normal PR interval in 2:1 A-V block and prolonged PR interval in 1:1 A-V conduction
An interval between the P wave conducted and the succeeding R wave was normal (0.16 second) in 2:1 A-V block. It was prolonged as long as 0.40 second in 1:1 A-V conduction. The atrial rate was ca. 82 per minute, and the ventricular rate ca. 41 per minute in the former. Both the atrial and the ventricular rates were ca. 71 per minute in the latter. This indicates that the PP interval is shorter than the refractory period of the A-V conduction system in the former and there were conduction delays in the A-V node, common bundle and left bundle branch in the latter.
Fig. 17. Progressive increase and progressive decrease in the PR interval in 1:1 A-V conduction

In the upper strip, the PR interval was progressively prolonged from 0.14 to 0.46 second. The atrial and the ventricular rates were ca. 77 per minute. In the middle strip, the PR interval remained prolonged between 0.40 to 0.44 second. Both the atrial and the ventricular rates were ca. 70 per minute. In the lower strip, the PR interval was progressively shortened from 0.40 to 0.16 second. The atrial and ventricular rates were ca. 74 per minute. This indicates the presence of first degree A-V block not only with the Wenckebach phenomenon but also with the counter-Wenckebach phenomenon.

acceleration was one of the causes of A-V block. Before the normalization of the PR interval, it showed a progressive decrease and then a progressive increase or the reverse without second degree A-V block, the longest PR interval being 0.42 and the shortest 0.14 second (Fig. 17).

As illustrated in Figs. 12 and 13, when the atrial impulse reaches the atrionodal junction outside the refractory period, it propagates through the left bundle branch. Accordingly, the QRS complex shows a pattern of RBBB. When the atrial impulse reaches the atrionodal junction or the beginning of the left bundle branch within the refractory period, the atrial impulse is blocked, and the A-V nodal pacemaker is made to escape through the right bundle branch. Therefore, the QRS complex shows a pattern of LBBB. During transitions between the 2, the impulse propagates more through one bundle branch than the other. When one bundle branch becomes exhausted, the impulse propagates more through the other bundle branch. The fact that transitional patterns such as QRS complexes of normal conduction, accelerated conduction, incomplete LBBB and incomplete RBBB can appear even when complete A-V block is present indicates that the origin of the QRS complex of a LBBB pattern in complete A-V block is located above the bifurcation of the bundle of His. From above the bifurcation, the impulse can propagate through either right or left bundle branch or both. During the period when the QRS complex shows a LBBB pattern with complete A-V block, 2 PP intervals are slightly greater than 1 RR interval (Figs. 12 and 13).

SUMMARY

Two cases of bilateral bundle branch block (BBBB) with atrioventricular (A-V) block were reported, and their mechanisms were discussed.

Case 1, a 29-year-old unmarried police sergeant.

On the day of admission, the electrocardiogram showed BBBB with 6:1 to 3:1 second degree A-V block. Transitions between LBBB and RBBB and the reverse were mostly prompt, and a QRS complex of an intermediate configuration rarely appeared. In general, the faster the atrial rate was, the severer was A-V block. The sinus rhythm was resumed on the 3rd hospital day with RBBB. The stabilized anterenoscal infarction pattern which had been present for 3 years before admission was disappeared as a result of this permanent RBBB. The Valsalva maneuver, hyperventilation and pressure on the eyeballs disclosed hypersensitivity of the autonomic nervous system, especially of the cardiac vagus. It should be emphasized that there were neither clinical sympt-
toms nor laboratory findings of myocardial infarction, inflammation, metabolic disorders and drug intoxications. Outstanding features were that BBBB occurred after long-standing mental tension and bodily exhaustion due to daily preparation for a pass examination far into the night and frequent interruption of his short sleep by traffic accidents in the district of his jurisdiction.

Case 2, a 68-year-old widow.
She had been visiting the out-patient clinic, and her electrocardiogram had shown complete RBBB with regular sinus rhythm. The electrocardiogram on admission showed complete A-V block with complete LBBB and 2:1 A-V block with complete RBBB. The transitions between the 2 were gradual. During the transitions, there occurred a QRS complex of normal conduction, accelerated conduction, incomplete LBBB and incomplete RBBB. Also in this case, the faster the atrial rate was, the severer was A-V block. Before the normalization of A-V conduction, the PR interval was once prolonged as long as 0.46 second in 1:1 A-V conduction and shortened to 0.16 second in 2:1 A-V conduction. Occasionally, the PR interval was progressively prolonged and then progressively shortened and vice versa. RBBB with complete A-V block disappeared on the 21st hospital day, but RBBB was left unaltered with regular sinus rhythm. Also in this case, there were neither clinical symptoms nor laboratory findings of myocardial infarction, inflammation, metabolic disorders and drug intoxications. It should be stressed that the patient was shocked when she saw her daughter's husband drunken throw her grandson down on the ground before the onset of BBBB with A-V block. Her daughter was a business woman, and she had to take care of her grandmother. Moreover, she and her daughter's husband were always on bad terms, and her sleep was frequently interrupted by violence of her daughter's husband returning home drunken late at night.

REFERENCES