PROGNOSTIC SIGNIFICANCE OF LONG VENTRICULAR PAUSES IN ATHLETES

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The long-term prognosis of athletes with long ventricular pauses has been unknown. In this study, thirty highly-trained cross-country skiers and 24 age-matched controls underwent 24 h Holter monitoring to detect the duration of the longest ventricular pause (LVP), and we followed the athletes for more than 3 years to evaluate prognostic significance of prolonged LVP. The LVPs of the athletes averaged 2.2±0.6 sec (range 1.3–3.4), which were significantly longer than those, 1.6±0.3 sec, of the controls. In the athletes, 20 cases (66.7%) had LVPs more than 2 sec, and 5 (16.7%) showed LVPs longer than 3 sec. During 3 years follow-up, no athletes had symptoms of near syncope, syncope or death under continued heavy training. Our study suggested that athlete with prolonged ventricular pause, even of 3 sec or more, did not have a different clinical risk than those without long pauses.

Heavy physical training may cause anatomic and physiologic cardiac changes, which are sometimes difficult for a clinician to evaluate as normal or pathological. Several studies\(^1\)\(^–\)\(^3\) have reported that both bradyarrhythmias and prolonged ventricular pauses are more common in athletes than in untrained individuals. In practice, these electrocardiographic signs among athletes are considered harmless. However, earlier reports\(^4\)\(^–\)\(^6\) have suggested that excessive training produces syncope related to sinus bradycardia or atrioventricular (AV) conduction abnormalities via a highly accelerated vagal tone. Although bradyarrhythmias and prolonged ventricular pauses are common in athletes, their clinical significance in regard to symptoms and prognosis has not been fully established. And there has been no previous study which has defined long-term prognosis of athletes with prolonged ventricular pauses.

In this study, we followed athletes for more than 3 years to evaluate the prognostic significance of long ventricular pauses detected by 24 h Holter monitoring. We evaluated ventricular pauses, not sinus pauses, because we wanted to clarify the relationship between length of pauses and symptoms or prognosis.

SUBJECTS AND METHODS

Subjects

Thirty male highly trained athletes aged 23.6±4.0 (mean±SD) years and 24 male age-matched (24.2±4.0 years) untrained subjects were studied. Every athlete was a member of a cross-country ski team of the Japanese self defense force. Ten were top international skiers, 12 top Japanese skiers,
TABLE I SINUS ARREST, SINOATRIAL BLOCK AND ATRIOVENTRICULAR BLOCK OBSERVED DURING 24-HOUR HOLTER MONITORING IN 30 ATHLETES AND 24 UNTRAINED SUBJECTS

<table>
<thead>
<tr>
<th></th>
<th>Athletes</th>
<th>Untrained subjects</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>No.</td>
<td>% of total</td>
</tr>
<tr>
<td>Sinus arrest, SA block</td>
<td>4</td>
<td>13.3</td>
</tr>
<tr>
<td>AV block</td>
<td>13</td>
<td>43.3</td>
</tr>
<tr>
<td>First degree</td>
<td>3</td>
<td>10.0</td>
</tr>
<tr>
<td>Second degree (Wenckebach)</td>
<td>10</td>
<td>33.3</td>
</tr>
<tr>
<td>Second degree (Mobitz II)</td>
<td>0</td>
<td>0.0</td>
</tr>
<tr>
<td>Third degree</td>
<td>0</td>
<td>0.0</td>
</tr>
</tbody>
</table>

and the remaining 8 were well trained skiers who had won regional competitions. All of them had regularly run 20 to 30 km/day and had been in intensive training for at least one year, and the mean duration of training was 4.5 ± 3.6 years. All athletes had continued less intensive training for three or 6 years before joining this team. The athletes entered in this study were asymptomatic, normotensive, and had no past history of cardiovascular disease. The untrained subjects were outpatients who underwent Holter monitoring for atypical chest pain. They had no abnormality in physical examination, 12-lead ECG and echocardiography, and were taking no medication.

Holter recordings
The athletes underwent Holter monitorings in October and December 1986. Four athletes received both monitorings. The untrained subjects were investigated between January 1989 and March 1990. A two-channel tape recorder (Tracker I, Reynolds) was used to obtain ambulatory ECGs. The total recording time for each subject was 24 h. All subjects carried out their normal activities and the athletes performed a training conforming to their ordinary life during Holter recording.

Tapes were scanned by a commercial analyser (Pathfinder 3, Reynolds) at 60 times normal speed and the entire 24 h tape was written out beat by beat. Maximal and minimal rates were defined as the fastest or slowest rate of consecutive beats averaged for 15 sec, respectively. The longest ventricular pause was defined as the longest R-R interval identified in the entire 24 h ECG. We printed out all arrhythmias and the longest ventricular pause at a paper speed of 25 mm/s, and two investigators reviewed and analysed these ECGs.

Follow-up Methods
All athletes were followed up for more than 3 years. In all cases, the interviewer attempted to ascertain the athlete's status as

Fig. 1. Longest ventricular pauses observed during 24 h Holter monitoring in athletes and untrained subjects.
alive and well, alive with symptoms or dead with a face to face interview or telephone interview. We guaranteed there would be no disadvantage resulting from their answers.

The above questionnaires were performed from April to June 1990. The mean follow-up period after the Holter monitoring was 42.5 months (range, 40 to 44 months), and no athlete was lost during this study.

Statistical Methods
Student’s unpaired t test and $\chi^2$ test were used in statistical analysis of the results. A p value less than 0.05 was considered statistically significant.

RESULTS

Heart rate
The minimal heart rate deduced from Holter monitoring was significantly lower in athletes ($36.2 \pm 5.3$ beats per minute (bpm)) than in the untrained subjects ($45.3 \pm 7.9$ bpm) (p<0.01). All athletes had sinus bradycardia of less than 50 bpm, and 21 athletes (70%) had marked (less than 40 bpm) bradycardia. Maximal heart rate was $163.2 \pm 16.0$ bpm for athletes and $131.2 \pm 16.2$ bpm for untrained subjects. All athletes had a heart rate of more than 120 bpm. These maximal heart rates were obtained at exercise.

Bradyarrhythmias
Bradyarrhythmias observed during 24 h are summarized in Table I. Four athletes (13.3%) and one untrained subject (4.2%) had an episode of sinus arrest or SA block more than once during the sleeping period (n.s.). First-degree AV block was observed in three athletes (10.0%) and in one untrained subject (4.2%) (n.s.). Wenckebach type second-degree AV block was observed in 10 of the athletes (33.3%) and in 2 of the untrained subjects (8.3%) during 24 h recording period (p<0.05). Mobitz type 2 second-degree AV block and complete AV block were not observed in both groups.

Longest ventricular pause

![Image of ECG tracing and annotations]

Fig.2. Sinus arrest or SA block with pause of 3.44 sec. P₁: sinus rhythm P₂: atrial escape beat.

<table>
<thead>
<tr>
<th>TABLE II</th>
<th>ETIOLOGY AND DURATION OF LONGEST VENTRICULAR PAUSES OBSERVED DURING 24-HOUR HOLTER MONITORING IN 30 ATHLETES</th>
</tr>
</thead>
<tbody>
<tr>
<td>Length of pause (in seconds)</td>
<td>&lt;2.0</td>
</tr>
<tr>
<td>No. of cases</td>
<td>10</td>
</tr>
<tr>
<td>Sinus arrhythmia</td>
<td>10</td>
</tr>
<tr>
<td>Sinus arrest, SA block</td>
<td>0</td>
</tr>
<tr>
<td>Second-degree AV block (Wenckebach type)</td>
<td>0</td>
</tr>
<tr>
<td>Compensatory pause after extrasystole</td>
<td>0</td>
</tr>
</tbody>
</table>

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TABLE III  THE INCIDENCE OF CLINICAL DETERIORATION RELATED TO LONGEST VENTRICULAR PAUSES IN ATHLETES DURING FOLLOW-UP OF MORE THAN 3 YEARS

<table>
<thead>
<tr>
<th>Length of pause (in seconds)</th>
<th>&lt;2.0</th>
<th>2.0-3.0</th>
<th>&gt;3.0</th>
</tr>
</thead>
<tbody>
<tr>
<td>No. of cases</td>
<td>10</td>
<td>15</td>
<td>5</td>
</tr>
<tr>
<td>Asymptomatic</td>
<td>10</td>
<td>15</td>
<td>5</td>
</tr>
<tr>
<td>Syncope</td>
<td>0</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Death</td>
<td>0</td>
<td>0</td>
<td>0</td>
</tr>
</tbody>
</table>

The athletes had significantly more prolonged longest ventricular pauses (LVP: 2.2 ± 0.6 sec) than the untrained subjects (1.6 ± 0.3 sec) (Fig. 1). In the athletes, 20 LVPs (66.7%) were more than 2 sec, and 5 (16.7%) longer than 3 sec. Maximal LVP in the athletes was 3.4 sec (Fig. 2). No relation was found between the LVPs and age or exercise duration. In 4 athletes who underwent Holter recordings twice, LVPs detected by each study were not very different.

Table II shows the etiology and duration of LVPs in the athletes. All cases having LVPs shorter than 2 sec were noted during sinus arrhythmia. In 15 athletes with LVPs ranging from 2 to 3 sec, 7 LVPs obtained during sinus arrhythmia, 6 were noted during Wenckebach type second-degree AV block, 1 was during sinus arrest or SA block, and 1 was after ventricular extrasystole. In 5 athletes with LVPs longer than 3 sec, 3 obtained during sinus arrest or SA block, and 2 cases during Wenckebach type second-degree AV block.

Follow-up study

Table III shows the long-term prognosis of the athletes divided into three subgroups according to the length of LVP. All athletes except one continued hard training at follow-up interview. No athlete had an episode of near syncope, syncope or death during follow-up of more than 3 years. No relation was found between length of pauses and the incidence of symptoms or survival.

DISCUSSION

In relative few studies of ventricular pauses exceeding 2 sec are relatively rare in healthy untrained individuals. In the past, ambulatory electrocardiographic studies have revealed that 2 of 50 healthy medical students and 2 of 259 healthy subjects aged 40 to 79 years had ventricular pauses of more than 2 sec. In the present study, they were seen in 2 of 24 normal nonathletes. In athletes, the incidence of ventricular pauses longer than 2 sec is significantly higher than in controls. Viitasalo et al. and Talan et al. detected LVPs longer than 2 sec with Holter monitoring in 13 of 35 endurance athletes (37.1%) and 7 of 20 long-distance runners (35%), respectively. The longest pauses in both studies reached a maximum of 2.8 sec. In this study, 20 of 30 athletes (66.7%) had LVPs longer than 2 sec, and 5 (16.7%) had those longer than 3 sec which had not been shown in asymptomatic athletes in previous studies. The reason why cross-country skiers in our study exhibited longer ventricular pauses with Holter monitoring than previously documented in reports describing other endurance athletes is unclear. The type and intensity of physical training should be one of the reasons. And another reason may be that intensive training was allowed and produced more acceleration of vagal tone during Holter recording in contrast to previous.
Ventricular Pause in Athlete

In conclusion, 24 h Holter monitoring revealed that asymptomatic athletes had significantly longer ventricular pauses than controls. However, follow-up study of more than 3 years suggested that athletes with prolonged pauses did not have a different risk than those without long pauses. The prolonged ventricular pauses of athletes without symptoms, of even 3 sec or more, appeared to be of little therapeutic or prognostic significance.

Acknowledgements

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REFERENCES


Studies

There is a controversy over the clinical significance of prolonged ventricular pause. Scheimann et al.\textsuperscript{10} stressed that patients with prolonged (≥ 2 sec) pauses were more apt to respond to pacemaker intervention. Ector et al.\textsuperscript{11} proposed that ventricular pause of 3 sec or more was a definite indication for the implantation of a permanent pacemaker, even in cases without symptoms. In contrast, Mazuz and Friedman\textsuperscript{12} reported that length of sinoatrial pauses correlated poorly with symptoms and did not predict sudden death in 84 patients with sick sinus syndrome. In our follow-up study of athletes, no relationship was found between the duration of ventricular pauses and the occurrence of symptoms or survival.

An abrupt cessation of cardiac output may result in a deficit in cerebral or coronary perfusion with its attendant effect producing either syncope or death. However, a critical length of ventricular pause at which symptoms occur has not yet been defined. It might be difficult to determine this critical length of pause because cerebral blood flow depends not only on the interval between heart beats, but also on regional vascular conditions\textsuperscript{13–16} or stroke volume, which has been reported to be increased in athletes\textsuperscript{17}. We speculate that symptomatic athletes reported by previous studies\textsuperscript{4–6} might have less adaptability of these other factors to physical training than asymptomatic ones, even if their LVPs were similar.

There are some limitations in our study. First, no athletes followed up in the present study had ventricular pauses longer than 3.4 sec. It is possible that athletes with more marked prolongation of ventricular pauses may have a worse prognosis. However, in previous studies, Holter recordings in asymptomatic athletes\textsuperscript{2,3,6,7} have never shown ventricular pauses longer than 3 sec. Therefore, we suppose that few athletes without symptoms are likely to have ventricular pauses longer than 3.4 sec. The second limitation of this study is that the study population was small and young so that the observations need to be only applicable to a specific subset of asymptomatic young adult athletes. Similar studies must be performed in a large number of subjects of different ages.


