Prognostic Significance of Premature Ventricular Contractions without Obvious Heart Diseases Determined by Standard 12-Lead Electrocardiography Considering their Morphology

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Background: Although ventricular premature contraction (VPC) commonly arises in subjects with and without heart diseases, the prognosis of VPC has remained controversial and the effect of their morphology on mortality has not been fully determined in subjects without obvious heart diseases. The objective of this study was to assess the morphologic effect of VPC on mortality.

Methods: Japanese atomic bomb survivors ($n = 6685$) underwent baseline health examinations and standard 12-lead electrocardiogram (ECG) between January 1990 and December 1991. Of these, we extracted data from 5,685 (67.1% women) subjects who had neither heart diseases nor electrocardiographic abnormalities at baseline. Among them, we identified 131 VPC cases using standard 12-lead ECG and classified them into left bundle branch block (LBBB) type ($n = 74$), right bundle branch block (RBBB) type ($n = 21$), and undetermined type ($n = 36$) according to their morphology. These subjects were followed up until December 2008; we compared all-cause, cardiac and coronary heart disease (CHD) mortality rates using multivariate Cox regression analysis between those with and without VPC.

Results: No VPCs were associated with all-cause and cardiac mortality, but the LBBB type was significantly associated with CHD mortality (hazard ratio, 2.73; 95% confidence interval, 1.11–6.73) after controlling for age, sex, smoking status, alcohol consumption, and underlying diseases.

Conclusions: Among Japanese atomic bomb survivors without obvious heart diseases, LBBB-type VPC was associated with increased CHD mortality. Larger studies are needed to confirm the effect of morphology as it might help to predict the risk.

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arrhythmia; epidemiology; mortality

Ventricular premature contraction (VPC) is a common arrhythmia in individuals with and without structural heart diseases. The notion that VPC suppression would prevent sudden death

had been accepted as a dogma until the late 1980s.¹ Since the Cardiac Arrhythmia Suppression Trial (CAST) showed that suppressing VPC with encainide and flecainide decreased survival rate

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after the incidence of myocardial infarction,^{2,3} VPC suppression was regarded during the 1990s as being of no clinical significance. On the other hand, more recent studies have found that VPC is a significant predictor of cardiovascular mortality, not only in patients with established heart diseases, but also in apparently healthy general populations.⁴⁻⁷ However, the studies in healthy general populations might have included individuals with occult structural heart diseases that could lead to adverse outcomes due to insufficient exclusion. Moreover, the effects of VPC morphology on prognosis have not been fully investigated. Therefore, we examined the prognostic significance of VPC detected by standard 12-lead electrocardiography (ECG) considering morphological differences, such as left bundle branch block (LBBB) and right (RBBB) bundle branch block (RBBB) types of VPC among Hiroshima and Nagasaki atomic bomb survivors who had no evident heart disease or electrocardiographic abnormalities.

METHODS

General Procedures

Since July 1, 1958, 23,418 atomic bomb survivors (59.2% women) in Hiroshima and Nagasaki, Japan, have undergone biennial health examinations as a part of a follow-up program conducted by the Radiation Effects Research Foundation (RERF). Detailed descriptions of the program have been published elsewhere.^{8,9} Assessments comprise a physical check-up, blood pressure measurements, standard 12-lead ECG, chest x-rays, and blood tests. Nurses also collect medical and lifestyle information. Clinical and electrocardiographic diagnoses are stored in a database using the International Classification of Disease (ICD) 7th, 8th, 9th, and 10th editions and the modified Minnesota codes and Kagan-Yano code¹⁰ adopted by the RERF.

During the baseline period between January 1990 and December 1991, a total of 6685 subjects (65.2% women) underwent health examinations. Those excluded from the study had coronary heart disease (CHD), cardiomyopathy, valvular heart diseases or congestive heart failure, and electrocardiographic abnormalities, such as atrial fibrillation or flutter, complete right or left bundle branch block, or left ventricular hypertrophy defined by the Sokolow-Lyon criterion of ≥ 3.5 mV.

Underlying heart diseases and electrocardiographic abnormalities were reviewed according to the ICD code (Table 1) and an ECG database, respectively. One thousand subjects with either heart diseases or other electrocardiographic abnormalities were also excluded (see below). Finally, data from 5685 (67.1% women) subjects followed up until December 2008 were analyzed (Fig. 1). We used the current DS02 dosimetry system to estimate individual colon atomic bomb radiation dose as a surrogate for heart dose.¹¹ The Research Protocol Review and Human Investigation Committees of RERF approved the protocol (RP-A13-08).

Confirmation and Definition of VPC and Its Morphology

Baseline standard 12-lead ECGs were recorded for 20 seconds at a paper speed of 25 mm/s and at a gain of 10 mm/mV from the limb leads to the chest leads. We extracted subjects who had VPC diagnosis at baseline from the ECG database. One author (D.H.) confirmed the diagnosis without reference to basic characteristics, comorbidities and subsequent vital status, and according to the morphology of VPC in the precordial lead, further classified the VPC as LBBB type indicating right ventricle origin, RBBB type indicating left ventricle origin, and undetermined type. There existed no cases with frequent or multifocal VPC among 5685 subjects without obvious heart disease. Those VPCs with no distinct morphology of LBBB or RBBB were defined as undetermined type.

Adjustment Factors and Underlying Diseases at Baseline

The information on hypertension (HT), diabetes mellitus (DM), hyperlipidemia (HL), obesity, smoking status, alcohol consumption, CHD, cardiomyopathy, valvular heart disease, congestive heart failure, stroke, and chronic obstructive pulmonary disease (COPD) were collected from all included individuals at baseline.

HT was defined as SBP ≥ 140 mmHg and/or DBP ≥ 90 mmHg, or medication with antihypertensive agents. Height (m) and body weight (kg) were measured and body mass index (BMI; kg/m²) was calculated as body weight divided by the square of height. Obesity was defined as a BMI of ≥ 25.0 kg/m² according to the definition of the Japan Society for the Study of Obesity.¹²

Table 1. International Classification of Diseases Codes for Cardiac and Coronary Heart Disease Mortality and Underlying Diseases

	ICD 9 to 1998	ICD 10 from 1998
Mortality		
Cardiac disease	390–429	I01–09, I11, I13, I20–52
Coronary heart disease (CHD)	410–414	I20–25
Underlying diseases		
Cardiomyopathy	422, 425	
Congestive heart failure	428	
Valvular heart disease	394–397	
Coronary heart disease	410–414	
Chronic obstructive pulmonary disease (COPD)	492	
Stroke	430–434

Figure 1. Selection process of study participants.

Among 6685 subjects assessed examined at baseline, we excluded 1000 with either heart diseases or electrocardiographic abnormalities. The final analysis included data from 5685 individuals.

LBBB = left bundle branch block; LVH = left ventricular hypertrophy; RBBB = right bundle branch block; VPC = ventricular premature contraction.

HL was defined as total cholesterol ≥ 220 (mg/dL) or serum triglyceride ≥ 150 (mg/dL), or medication with lipid-lowering agents. DM was defined as apparent diabetic history or medication with hypoglycemic agents. Current and previous smokers or drinkers are described as smokers or drinkers. Angina pectoris at exertion and at rest, and myocardial infarction were defined as CHD. The diagnosis of angina pectoris and myocardial infarction were made based on specific ECG changes, findings of coronary angiography, and existence of medical or invasive treatments, such as percutaneous coronary intervention or coronary

artery bypass grafting. Cardiomyopathy included dilated and hypertrophic types. Mitral, aortic, tricuspid, and pulmonary valve diseases were defined as valvular heart diseases. Cerebral infarction, hemorrhage, and subarachnoid hemorrhage were defined as stroke. Chronic pulmonary emphysema was defined as COPD. The above information was extracted from the database.

Confirmation of End Points for Mortality

The RERF followed the vital status of all participants using the Japanese family registration

system. Death certificates were collected for all deceased participants and the primary cause of death was recorded according to the coding rules of the ICD 9th and 10th editions. Table 1 shows the list of ICD codes used to identify cardiac and CHD death.

Statistical Analysis

We compared baseline characteristics of blood pressure, body weight, height, total cholesterol, triglyceride, fasting blood glucose, cardiothoracic ratio, ECG parameters, atomic bomb radiation dose, and underlying diseases (HT, HL, DM, COPD, stroke, obesity), between subjects with and without VPC and between LBBB and RBBB type VPC using a linear model adjusted for age and sex for continuous variables, or the chi-square or Fisher's exact test, as appropriate, for categorical variables.

The effects of smoking status, alcohol consumption, and other underlying diseases and conditions on the odds of VPC were assessed using logistic regression adjusted for age and sex. Prognostic significance of VPC and its morphology (LBBB, RBBB, and undetermined type) for all-cause, cardiac, and CHD mortality were assessed using Cox proportional hazards analyses adjusted for age, sex, smoking status, alcohol consumption, and underlying diseases with non-VPC group as reference group. Survival was taken as the amount of time from baseline until death or December 31, 2008, whichever came first. The proportional hazards assumption in Cox model for each variable was assessed by the diagnostic log-log plot. All data were statistically analyzed using SAS version 9.1 for UNIX (SAS Institute, Cary, NC, USA). Estimates are reported with appropriate measures of precision. $P < 0.05$ was considered statistically significant.

RESULTS

We identified a total of 131 (68.7% women; 56.5% LBBB, 16.0% RBBB, 27.5% undetermined) VPC cases among 5685 subjects (67.1% women) at baseline. The prevalence of total VPC among subjects without the obvious heart diseases was 2.3% (2.2% and 2.4% for men and women, respectively) and did not significantly differ between the sexes ($P = 0.97$, by the chi-square test).

Among the follow-up study subjects, sex, other characteristics, and radiation dose did not significantly differ between the groups with or without VPC (LBBB, RBBB, and undetermined type). Those factors were also not different between the cases with LBBB and RBBB types of VPC (Table 2). Age and ventricular rate were significantly higher among subjects with, than without VPC. Underlying diseases, smoking status, and alcohol consumption had no statistical effect on the odds of VPC or its morphologies (data not shown). These findings indicate that the VPC identified herein is likely idiopathic.

Table 3 shows causes of death and Table 4 compares each VPC group (total, LBBB type, RBBB type, and undetermined type) to those without VPC on the hazard of each cause of death considered. None of those with RBBB type VPC died due to CHD. Total VPC (LBBB type, RBBB type, and undetermined type) was not associated with all-cause, cardiac, and CHD mortalities according to analyses adjusted for age, sex, and multiple factors. RBBB type and undetermined type VPC were not associated with all-cause, cardiac, and CHD mortality, whereas LBBB type was associated with CHD (HR, 2.73; 95%CI, 1.11–6.73; $P = 0.03$), but not with either all-cause (HR, 0.76; 95%CI, 0.51–1.13; $P = 0.17$) or cardiac mortality (HR, 2.00; 95%CI, 0.55–2.49; $P = 0.68$). Current analyses did not find the significant relationship between radiation dose and the prevalence of any VPC and its morphology (P for total = 0.86, P for LBBB = 0.56, P for RBBB = 0.36, and P for undetermined = 0.15, respectively) or with any cause of mortality (data not shown).

DISCUSSION

The prevalence of VPC (LBBB, RBBB, and undetermined type) among Japanese atomic bomb survivors without either heart disease or electrocardiographic abnormalities was 2.3%. Cox regression analysis showed that total VPC, RBBB type VPC, and undetermined type VPC in standard 12-lead ECG did not associate with all-cause, cardiac, and CHD mortality, whereas LBBB type VPC related with CHD mortality after adjusting the effects of age, sex, smoking status, alcohol consumption, and underlying diseases. We did not find any association between radiation dose and prevalent VPC ($P = 0.86$) and any cause of mortality (data

Table 2. Baseline Characteristics of Participants

	With VPC					P ^a	P ^b
	Without VPC	Total VPC	LBBB type	RBBB type	Undetermined type		
Number	5554	131	74	21	36		
Sex (male/female)	1831/3723	41/90	25/49	7/14	9/27	1.00	0.76
Age (y)	63.5 ± 10.9	66.4 ± 10.4	64.7 ± 10.2	68.5 ± 9.0	68.9 ± 11.0	0.13	0.002
Smoker (%)	38.7	40.5	43.2	47.6	30.6	0.91	0.75
Alcohol consumption (%)	64.4	64.1	68.9	57.1	58.3	0.46	1.00
SBP (mmHg)	127.9 ± 21.2	130.4 ± 22.8	129.9 ± 22.2	129.7 ± 27.3	131.8 ± 21.8	0.66	0.65
DBP (mmHg)	78.5 ± 11.5	78.5 ± 12.4	78.3 ± 11.4	79.4 ± 15.3	78.3 ± 13.1	0.63	0.91
BMI (kg/m ²)	22.8 ± 3.4	22.7 ± 3.8	22.5 ± 3.2	22.8 ± 4.3	23.2 ± 4.7	0.78	0.87
Hemoglobin (g/dL)	13.4 ± 1.5	13.3 ± 1.4	13.5 ± 1.4	13.1 ± 1.0	13.1 ± 1.4	0.34	0.57
T-cholesterol (mg/dL)	212.3 ± 39.2	209.0 ± 38.1	208.9 ± 37.1	213.7 ± 35.3	206.4 ± 42.4	0.63	0.33
TG (mg/dL) (n = 2597)	136.3 ± 89.8	123.0 ± 73.6	123.3 ± 59.2	140.1 ± 89.7	142.3 ± 106.9	0.40	0.66
HbA1c (%) (n = 757)	6.3 ± 1.6	5.7 ± 0.8	5.5 ± 0.6	6.3 ± 1.6	5.7 ± 0.7	0.30	0.11
Fasting glucose (mg/dL) (n = 2591)	101.5 ± 22.4	104.0 ± 26.9	99.8 ± 16.6	116.0 ± 43.1	108.0 ± 36.0	0.08	0.46
Uric acid (mg/dL)	5.0 ± 1.4	5.1 ± 1.4	5.1 ± 1.4	5.0 ± 1.3	5.0 ± 1.6	0.63	0.80
Potassium (mEq/L) (n = 918)	4.1 ± 0.4	4.2 ± 0.4	4.1 ± 0.3	4.5 ± 0.5	4.2 ± 0.2	0.06	0.54
CTR (%)	48.0 ± 5.0	49.0 ± 4.9	48.4 ± 4.7	50.7 ± 4.8	49.2 ± 4.8	0.32	0.29
Vent-rate (beat/min)	66.2 ± 10.0	68.7 ± 11.3	67.3 ± 10.3	75.8 ± 15.7	67.3 ± 9.0	0.01	0.01
QTc (s ^{1/2})	0.42 ± 0.02	0.42 ± 0.03	0.42 ± 0.03	0.44 ± 0.04	0.42 ± 0.02	0.09	0.43
QRS (s)	0.08 ± 0.02	0.08 ± 0.02	0.08 ± 0.02	0.08 ± 0.03	0.08 ± 0.02	0.45	0.14
PR interval (s)	0.16 ± 0.03	0.16 ± 0.03	0.16 ± 0.02	0.16 ± 0.02	0.16 ± 0.03	0.55	0.54
HT (%)	41.3	42.8	37.8	52.4	47.2	0.35	0.80
DM (%)	10.2	9.9	8.1	9.5	13.9	1.00	1.00
HL (%)	45.7	43.5	40.5	61.9	38.9	0.14	0.68
COPD (%)	2.6	1.5	1.4	0	2.8	1.00	0.78
Stroke (%)	3.9	2.3	1.4	4.8	2.8	0.40	0.47
Obesity (%)	23.4	21.6	18.1	19.1	31.3	1.00	0.71
Radiation dose (Gy) (n = 4491)	0.38 ± 0.58	0.32 ± 0.54	0.33 ± 0.53	0.47 ± 0.74	0.22 ± 0.41	0.43	0.43

Unless otherwise indicated, values are shown as means ± SD.

^aLBBB vs. RBBB type VPC; ^bWith vs. without VPC. Continuous variables except for age were tested with adjustment for age and sex.

BMI = body mass index; COPD = chronic obstructive pulmonary disease; CTR = cardiothoracic ratio; DBP = diastolic blood pressure; DM = diabetes mellitus; HL = hyperlipidemia; HT = hypertension; LBBB = left bundle branch block; RBBB = right bundle branch block; SBP = systolic blood pressure; VPC = ventricular premature contraction.

Table 3. Causes of Death According to VPC Morphology Compared with Subjects without VPC

	With VPC	LBBB Type	RBBB Type	Undetermined Type	Without VPC
n	131	74	21	36	5554
Causes of death					
All-cause	55 (42.0%)	26 (35.1%)	11 (52.4%)	18 (50.0%)	2055 (37.0%)
Cardiac	15 (11.5%)	8 (10.8%)	2 (9.5%)	5 (13.9%)	396 (7.1%)
CHD	8 (6.1%)	5 (6.8%)	0 (0%)	3 (8.3%)	139 (2.5%)

VPC = ventricular premature contraction; LBBB = left bundle branch block; RBBB = right bundle branch block; CHD = coronary heart disease.

not shown). Thus, our results may be generalized to nonatomic bomb survivors.

Two-minute ECG has identified VPC in about 6% of middle-aged African American and Caucasian men, including those with heart disease.¹³ The prevalence of VPC determined by a meta-analysis of short-term (10 seconds or 2 minutes) or long-term (24 hours) ECG recordings in general populations ranges from 1.2% to 10.7%.¹⁴ The prevalence detected by routine 12-lead ECG is 1.4% and 1.1% for healthy Japanese men and women, respectively, without a history of myocardial infarction and stroke.¹⁵ Our findings of 20-second ECG data from individuals without overt heart diseases and electrocardiographic abnormalities essentially agreed with these results.

The prognostic significance of VPC in apparently healthy individuals is inconsistent between studies (Table 5). Kennedy et al. found no association of frequent or multifocal VPC (n = 73) in apparently healthy individuals with mortality due to all-cause, myocardial infarction, or CHD over a 6.5-year follow-up period.¹⁶ Fleg et al. also found no statistical evidence that VPCs detected by 24-hour ambulatory ECGs among healthy participants aged ≥60 years predict the development of coronary events at 10 years of follow-up.¹⁷

In contrast, several studies have associated VPC among healthy individuals with increased all-cause, cardiac, and CHD mortality and men without CHD who had frequent and multifocal VPC in the Framingham Heart Study were at increased risk for all-cause mortality (relative risk, 2.30; 95%CI, 1.65–3.20) and CHD mortality (relative risk, 2.12; 95%CI, 1.33–3.38).¹⁸ The pooled adjusted relative risks for sudden and total cardiac death were 2.64 (95%CI, 1.93–3.63) and 2.07 (95%CI, 1.71–2.50), respectively, in a recent meta-analysis comprising a general population of 106,195 individuals.¹⁴ The relative risk of CHD events associated with VPCs adjusted for age, sex, race, and CVD risk

factors on 2-minute ECG was 1.30 (95% CI, 1.06–1.58) among subjects without CHD.¹⁹ Our overall results were consistent with the earlier mentioned studies^{16,17} on mortalities of total VPC. In these previous studies, the exclusion of underlying heart diseases was limited and VPC morphology was not taken into consideration. Moreover, some studies have either a smaller sample size or a shorter follow-up period than the present study. Here, we analyzed single and monomorphic VPC detected by short-term standard 12-lead ECG and used clinical and electrocardiographic assessments to exclude as many subjects as possible who were likely to have any heart diseases. We divided VPC cases according to its morphology, and then individually assessed the impact of LBBB, RBBB, and undetermined type VPC morphology. Total, RBBB, LBBB, and undetermined type VPC were not associated with increased all-cause and cardiac mortality, but LBBB type VPC was a significant predictor of CHD mortality. Very few subjects had RBBB type VPC and none of them died of CHD. Although our results should be interpreted with care, the inconsistency of CHD mortality of total VPC between studies might be due to the difference in the proportion of LBBB type to total.

In this study, LBBB type VPC predicted CHD mortality, so LBBB type VPC implying right ventricle origin may be likely to reflect the latent coronary dysfunction. Although we are not able to deny the effects of small sample size and mechanistic difference, the present study suggests that the prognostic value of VPC for CHD mortality is different between its morphology. To draw a more definitive conclusion about the CHD mortality of RBBB type VPC and its difference between morphologies, further analysis of larger sample size and of the mechanistic difference is required.

Short-term 12-lead ECG is the current standard, noninvasive approach to the early detection of

Table 4. Risk of Death According to VPC Morphology Compared with Subjects without VPC

	Total Mortality HR (95%CI)		Cardiac Mortality HR (95%CI)		CHD Mortality HR (95%CI)	
	Age and sex adjusted	Multiple adjusted	Age and sex adjusted	Multiple adjusted	Age and sex adjusted	Multiple adjusted
Total VPC	0.82 (0.63–1.08) P = 0.16	0.88 (0.66–1.16) P = 0.35	1.14 (0.68–1.90) P = 0.63	1.22 (0.70–2.13) P = 0.48	1.74 (0.85–3.55) P = 0.13	2.09 (0.97–4.50) P = 0.06
LBBB type	0.73 (0.49–1.07) P = 0.11	0.76 (0.51–1.13) P = 0.17	1.13 (0.56–2.27) P = 0.74	1.17 (0.55–2.49) P = 0.68	2.00 (0.82–4.90) P = 0.13	2.73 (1.11–6.73) P = 0.03
RBBB type	1.10 (0.61–1.98) P = 0.76	1.27 (0.70–2.31) P = 0.43	1.17 (0.29–4.70) P = 0.82	1.24 (0.31–5.02) P = 0.76	ND	ND
Undetermined type	0.86 (0.54–1.37) P = 0.52	0.90 (0.54–1.49) P = 0.67	1.14 (0.47–2.76) P = 0.77	1.29 (0.48–5.02) P = 0.62	1.99 (0.63–6.25) P = 0.24	2.15 (0.53–8.76) P = 0.29
Without VPC	1.00	1.00	1.00	1.00	1.00	1.00

Multiple Cox regression analysis adjusted for age, sex, smoking status, alcohol consumption, hypertension, diabetes mellitus, hyperlipidemia, obesity, chronic obstructive pulmonary disease, and stroke with a group without VPC as a reference. Note that 346 subjects with missing data of covariates were excluded. CHD = coronary heart disease; CI = confidence interval; LBBB = left bundle branch block; ND = not done; RBBB = right bundle branch block; VPC = ventricular premature contraction.

Table 5. Comparisons of Risk for Each Event between Previous Studies and the Present Study

First Author (Year) (Ref)	Location	Size of Population	Follow-Up (yrs)	Exposure Definition	End Points	Main Findings
Kennedy et al. (1985) ¹⁶	United States	73	6.3	Frequent or complex VPC (Holter ECG)	All-cause CHD death	No associations between VPC vs non-VPC event rates
Fleg et al. (1992) ¹⁷	United States	98	10	Any VPCs (Holter ECG)	CHD events including death	No associations between VPC vs non-VPC event rates
Bikkina et al. (1992) ¹⁸	United States	6,033	4–6	Any VPCs (Holter ECG)	All-cause CHD death	All cause, RR, 2.30; 95%CI, 1.65–3.20 CHD, RR, 2.12; 95%CI, 1.33–3.38
Massing et al. (2006) ¹⁹	United States	15,070	10	Any VPCs (Short strip ECG)	CHD events including death	CHD events, RR, 1.30; 95%CI, 1.06–1.58
Ataklte et al. (2013) ¹⁴	Meta-Analysis	106,195	5–30	Any VPCs (Short strip or Holter ECG)	SCD TCD	SCD, RR, 2.64; 95%CI, 1.93–3.63 TCD, RR, 2.07; 95%CI, 1.17–2.50
Present study	Japan	5,685	19	Single or Monofocal VPC (short strip ECG)	All-cause death cardiac death CHD death	All-cause, RR, 0.88; 95%CI, 0.66–1.16 Cardiac, RR, 1.22; 95%CI, 0.70–2.13 CHD, RR, 2.09; 95%CI, 0.97–4.50

VPC = ventricular premature contraction; CHD = coronary heart disease; ECG = electrocardiogram; SCD = sudden cardiac death. TCD = total cardiac death; RR = relative risk; CI = confidence interval.

heart diseases in the general public. However, appropriate measures and advice to subjects with VPC detected by chance at regular health checkups have not been established. Our results indicate that subjects with LBBB type VPC should be carefully followed up to detect the early development of CHD.

Limitations

This epidemiological study has several limitations. We could not completely exclude heart diseases at baseline because we did not perform echocardiography and cardiac catheterization. Since the number of undetermined type VPC cases was relatively large, we did not take the axis of VPC into consideration. Unlike previous studies based solely on a history of heart disease, we excluded subjects with heart disease based on electrocardiographic abnormalities in addition to such history. Our conclusions might be limited by the fact that few subjects had VPC and related events. Mortality due to CHD was undetectable in our small sample of subjects with RBBB type VPC. Larger studies are needed to draw definitive conclusions about the prognostic significance of this morphology. We recorded ECG for only 20 seconds and the detection of VPC depends on their frequency of occurrence and recording duration. We might thus have underestimated VPC prevalence. However, many epidemiological studies have used a short-term rhythm strip to detect VPC as a screening tool. Evenson et al. validated the ability of short-term rhythm strips compared with ambulatory 24-hour monitoring to detect VPC in epidemiological studies. The sensitivity and specificity of the strips were 76% and 94%, respectively, when the average number of VPC/h was >30 .²⁰ We used death certificates to identify cardiac and CHD deaths. There exist uncertainties in the accuracy of death certificate information. This may limit the present results.

CONCLUSION

Total VPC (LBBB, RBBB, and undetermined types) was not associated with all-cause, cardiac, and CHD mortality in Japanese atomic bomb survivors without either heart diseases or electrocardiographic abnormalities. The LBBB, but not RBBB type VPC, associated with CHD mortality. Atomic bomb radiation dose was not associated with the prevalence of VPC, its morphology,

and mortality. Therefore, our results would be generalized to nonatomic bomb survivors. The morphology of VPC in standard 12-lead ECG might be a useful tool for CHD risk assessment in regular health check-ups.

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