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The Influence of Ventricular Premature Contraction on Cardiac Structure and Function

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Abstract: ***Ventricular premature contraction (VPC) constitutes a frequently encountered cardiac arrhythmia in clinical settings. This review aims to summarize the effects of VPC burden, origin site, morphology, QRS duration, and coupling interval on cardiac structure and function.***

Keywords: Ventricular premature contractions, Cardiac structure, Cardiac function.

1. Introduction

Ventricular premature contraction (VPC) denotes the premature depolarization of the ventricular myocardium that originates from ectopic pacemakers situated below the His bundle and its branches. The electrocardiogram typically reveals the manifestation of an abnormal QRS complex that occurs earlier than anticipated, with a duration of at least 120 ms, and a corresponding T wave that is typically broader and opposite in direction to the QRS main waveform, and with no preceding P wave.

The pathogenesis of VPC is primarily attributed to abnormal autonomic function of the myocardium, trigger activity, and reentry. Frequent VPC can potentially result in VPC-induced cardiomyopathy. This might be caused by the occurrence of VPC, which leads to asynchronous contraction of the ventricles and reduces the efficiency of heart contraction, thereby increasing myocardial oxygen consumption. It could also be due to the fact that VPC triggers sympathetic nervous system excitation and the elevated level of catecholamines in plasma, ultimately leading to myocardial ischemia.

VPC constitutes one of the most common arrhythmias in clinical practice. Occasional VPC is typically observed in individuals with normal cardiac structure, while frequent VPC is often a sign of underlying cardiac substrate abnormalities. Factors associated with an adverse prognosis of PVC include a high PVC burden (>20%), VPC not originated from the outflow tract, multiple VPC morphologies, a wide QRS duration in VPC, and short coupling intervals between VPC (R-on-T) [1]. This review will further scrutinize the effects of these factors on cardiac structure and function.

2. The Effects of VPC Burden on Cardiac Structure and Function

VPC burden is defined as the ratio of VPC frequency to total heart rate within 24 hours in a patient. Studies have indicated that, due to individual variations, the total heart rate of VPC patients within 24 hours is also inconsistent. Consequently, VPC burden is frequently employed as one of the indicators for evaluating the condition of patients with VPC in clinical practice [2]. With the increase of VPC burden, the influence on cardiac structure and function will also increase. Song [3] classified the 220 VPC patients into a low-load group (<10%), a middle-load group (10%-20%), and a high-load group (>20%), and selected 68 healthy individuals without VPC as the normal control group. It was found that the cardiac structure and function indicators in the low-load group were not statistically different from those in the control group. However, with the increasing load, the levels of left atrial diameter (LAD), left ventricular outflow tract diameter (LVOT), left ventricular relaxed end-diastolic diameter (LVEDd), right ventricular relaxed end-diastolic diameter (RVEDd), left ventricular relaxed end-diastolic volume (LVEDV), stroke volume (SV), left ventricular mass index (LVMI), and NT-proBNP of VPC patients increased significantly, and the LVEF decreased significantly. Zhang [4] collected the data of LVEF, left ventricular end-systolic diameter (LVESd), LVEDd, and NT-proBNP for 75 patients with a high VPC burden. It was found that the high VPC burden negatively correlated with LVEF in a linear fashion, positively correlated with LVESd, LVEDd, and NT-proBNP. This indicates that a high VPC burden has a more pronounced impact on cardiac structure and function, and the degree of myocardial damage intensifies as the burden of ventricular premature contractions increases. Altıntaş et al. [5] discovered that VPC burden was independently related to the reduction of the patient's LVEF. When the VPC burden exceeded 5%, a more significant decline in LVEF was observed as the VPC burden increased. Furthermore, when the VPC burden surpassed 20%, the LVEF of the patients was below the normal range. Lie et al. [6] incorporated 52 patients with atrial fibrillation who underwent ablation and determined that a VPC burden >8% correlated with impaired myocardial function in patients with atrial fibrillation. Park Y et al. [7] recruited 146 VPC patients and 292 control subjects for comparison, and found that after excluding the interference of hypertension, diabetes, etc., patients with higher VPC burden had larger left atrial volume, larger LVEDd, and lower LVEF.

3. The Effects of VPC Origin Site on Cardiac Structure and Function

Research studies have demonstrated that the majority of VPCs have their origin in the right ventricle, particularly in the outflow tract [8]. Farzaneh et al. have also verified that 80% of VPCs originate from the right ventricle, with the preponderance emerging from the right ventricular outflow tract (RVOT), while the remaining 20% arise from the left ventricle (LV) [9]. However, it is not yet clear which of the

VPC origins from the left ventricular outflow tract (LVOT) or the RVOT has a greater impact on cardiac structure and function. Wang [10] et al. recruited 200 cases of patients with non-structural cardiac diseases whose VPC frequency exceeded 1,000 times within 24 hours, and discovered that the left ventricular origin group had significantly larger LAD, LVESd, and LVEDd than the right ventricular origin group ($P<0.05$). Xu et al. [11] enrolled 102 patients without coronary heart disease or structural heart disease in their study and underwent 7-day ambulatory electrocardiogram monitoring. They discovered that the NT-proBNP level was lower in the RVOT group than that in the LVOT group. Additionally, patients with VPCs originating from the tricuspid annulus had the highest burden and NT-proBNP level, while those originating from the left ventricular papillary muscle and fascicle had higher LVEF but lower LVESd than other sites. However, Munoz et al. [12] conducted a retrospective study on 70 patients who underwent VPC ablation and were categorized into two groups based on LVEF (<50% versus >50%). There were no significant discrepancies in baseline characteristics between the two groups. The study determined that a left ventricular origin VPC burden of ≥20% could lead to a decrease in LVEF, while a right ventricular origin VPC burden of ≥10% could also bring about a reduction in LVEF. It is possible that PVC of right ventricular origin delays the excitation and contraction of the left ventricle, thereby causing asynchronous contraction of the heart chambers and resulting in a decrease in LVEF.

4. The Effects of VPC Morphology on Cardiac Structure and Function

VPC can be categorized into monomorphic and polymorphic forms, and current researches have indicated that polymorphic VPC exerts a more significant influence on cardiac structure and function compared with monomorphic VPC. Munoz et al. [12] incorporated 70 patients who underwent VPC ablation and discovered that 46 of them (66%) had more than one distinct VPC morphology, suggesting that patients with reduced LVEF are more prone to possess multiple VPC morphologies in comparison with those with normal LVEF (In the group characterized by reduced LVEF, 88% of the patients presented with multiform VPC, whereas in the group featuring normal LVEF, 58% of the patients presented with multiform VPC). A retrospective cohort study by Ephrem et al. [13] involving 222 patients revealed that patients with polymorphic VPC had a fourfold higher risk of adverse outcomes such as acute coronary syndrome, stroke, or all-cause mortality within four years compared with patients with monomorphic VPC. Gallagher et al. [14] enrolled 2,332 patients who underwent ambulatory electrocardiographic monitoring and discovered that the morphological and numerical traits of ventricular ectopic beats were capable of predicting the all-cause mortality.

Furthermore, Liu [15] holds that the risk stratification method of ventricular arrhythmia frequency and QRS wave morphology put forward by Myerburg et al. is both qualitative and quantitative, and its role in predicting the prognosis of patients is more extensive than that of the Lown classification. It bears certain clinical significance for judging the prognosis of patients and the selection of treatment. The final outcome is expressed by the stratification results of frequency and morphology, with a higher score indicating a more severe ventricular arrhythmia. The morphological stratification encompasses monomorphic and polymorphic VPC, which to some extent reflect the influence of VPC morphology on the patient's cardiac structure and function.

5. The Effects of QT Interval and QRS Duration of VPC on Cardiac Structure and Function

The QT interval refers to the total duration of the depolarization and repolarization process of ventricular muscle, which is easily affected by heart rate, therefore, the corrected QT interval is usually used to evaluate the QT interval in clinical practice. Zhang et al. [16] contend that the prolonged QT interval constitutes a major risk factor for fatal ventricular arrhythmias, Through their research, they discovered that in the case of the prolonged QT interval, VPC can be generated by two mechanisms: induction by repolarization gradient and early afterdepolarization induction in phase 2. Currently, studies have shown that abnormal changes of the QT interval can reflect cardiac function to a certain extent [17]. Furthermore, prolonged QT interval is likewise a recognized risk factor for the incidence of ventricular tachycardia, encompassing torsade de pointes and ventricular fibrillation [18]. The QRS duration is usually a good indicator of the level of left ventricular function. When heart remodeling occurs, it leads to cardiac enlargement and a decrease in LVEF, meanwhile, it also interferes with the electrical conduction capacity of the ventricular muscle, giving rise to an elongation of the QRS wave duration on an ECG [19]. Hu [20] discovered through a study involving 98 VPC patients that when patients had no structural heart disease, the QRS duration of VPC was typically narrower (132 ± 10 ms). Although the QRS duration of VPC in the structural heart disease group was significantly greater than that of the group without structural heart disease, when the patients had normal cardiac function, LVEF, and left ventricular cavity size, the QRS duration of VPC did not exhibit significant widening (138 ± 15 ms), showing no statistical difference compared with the group without structural heart disease. As cardiac function was compromised, LVEF decreased, and the left ventricular cavity expanded, the QRS duration of VPC significantly broadened, particularly when cardiac function was at grade III or IV, with a more pronounced widening of the QRS duration. This indicates that the QRS duration of VPC is closely associated with LVEF, cardiac function status, and the size of the left ventricular cavity. Yokokawa et al. [21] demonstrated that the duration of the QRS wave in VPC is closely associated with VPC-induced cardiomyopathy. A QRS duration of ≥150 ms is the most effective parameter in discriminating between patients with and without VPC-induced cardiomyopathy. Park K et al. [22] enrolled 412 consecutive outpatients diagnosed with frequent VPC and no structural cardiovascular disease in their study to explore the influence of VPC QRS duration on LV function. The study indicated that a QRS duration in frequent VPC >157 ms correlated with LV dysfunction.

6. The Effects of VPC Coupling Interval on Cardiac Structure and Function

Studies have demonstrated that when VPC emerges, the short coupling interval between the sinus rhythm and VPC is prone to induce a decrease in calcium handling capacity and abnormal calcium exchange. Concurrently, the long interval subsequent to VPC will augment the perfusion of the ventricle, thereby giving rise to abnormal ventricular diastolic and contractile functions [23]. Nurşen Keleş et al. [24] incorporated 57 patients with a VPC frequency surpassing 10,000 within 24 hours as the experimental group and 54 healthy volunteers as the control group. After eliminating the influences of factors such as gender, body mass index, hypertension, and diabetes, they discovered that a significant positive correlation ($P<0.001$) existed between the coupling interval and early diastolic strain rate (SRe) in the correlation analysis. Moreover, the SRe of VPC patients was lower than that of normal individuals, suggesting that the lower the coupling interval in VPC, the more significant the impact on cardiac diastole. Furthermore, Xie [25] enrolled 102 hospitalized patients with frequent monomorphic VPC who underwent radiofrequency ablation. Among them, 22 patients with VPC-induced myocarditis were designated as the experimental group, and the remaining 80 were classified as the control group. After eliminating the disparities in age and gender, the VPC coupling interval before surgery and the LVEF and LVEDd before and after 6 months of surgery were compared between the two groups of patients. The experimental group exhibited a significantly longer VPC coupling interval (506 ± 35 ms) than the control group (466 ± 38 ms) ($P<0.01$). This indicated that PVC with longer coupling intervals might be more susceptible to developing PVC-induced myocarditis. The potential pathogenesis could be that longer coupling intervals lead to a reduction in the left ventricular filling time before the next sinus rhythm, which triggers the sympathetic nervous system and the renin-angiotensin-aldosterone system, thereby causing ventricular remodeling. A retrospective analysis involving 206 patients also revealed that a VPC coupling interval greater than 500 ms was associated with abnormal left ventricular remodeling [26].

7. Conclusions

The mechanism that how VPC influences cardiac function remains unclear at present. It is predominantly postulated that asynchronous ventricular contraction, enhanced sympathetic nerve excitability, abnormal calcium ion levels, excessive activation of the RAAS system etc. might be involved in the impairment of cardiac function caused by VPC. In clinical practice, the assessment of the impact of VPC on cardiac structure and function should be comprehensively evaluated from multiple aspects such as PVC burden, origin site, morphology, QT interval, QRS duration, and coupling interval to formulate more rational and effective treatment plans.

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