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## Relationship between abnormal vagus nerve tension and basal ganglia cerebral infarction induced paroxysmal atrial fibrillation

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## ABSTRACT

**Objective:** To investigate the relationship between basal ganglia cerebral infarction and paroxysmal atrial fibrillation (PAF) caused by abnormal vagus nerve tension.**Methods:** A total of 1 483 cases of elder patients with cerebral infarction who received head CT or MRI examination during the period were enrolled, including 830 male and 613 female, with the average age as 78 years. These cases were divided into basal infarction ganglia group ( $n = 1\ 045$ ) and non-basal ganglia infarction group ( $n = 438$ ) according to the anatomic site of cerebral infarction. The differences of the incidence of PAF, left atrial diameter and heart rate variability were compared between the two groups.**Results:** In basal ganglia infarction group, the incidence rate of PAF was significantly higher than that of non-basal ganglia infarction group ( $P < 0.05$ ). The incidence trend of cerebral infarction in basal ganglia was age-related, in the  $>79$  years basal ganglia cerebral infarction group, the incidence of PAF was significantly higher than that of non-basal ganglia infarction group ( $P < 0.05$ ). There was no significant difference in the left atrial diameter between the basal ganglia infarction group and non-basal ganglia infarction group. Basal ganglia cerebral infarction patients with high PAF had higher heart rate variability than non-basal ganglia infarction group.**Conclusion:** Elderly patients with basal ganglia infarction have high incidence of PAF. Sympathetic nerve damage in cerebral basal ganglia, increased vagal tension and cardiac vagal tension are the direct causes of PAF. The results indicates that the increased central vagal nerve tension mediated PAF probably is an indication of supplying sympathetic neurotransmitter or cardiac vagal denervation treatment.

## 1. Introduction

Atrial fibrillation (AF) is one of the most common arrhythmia, which could cause clinical thrombosis, heart failure, higher hospitalization rates and high mortality. With the development of drugs, intervention and implantation devices, AF treatment efficacy has been greatly improved. The research of autonomic nerve imbalance has been a hot topic in studying the

mechanisms of AF. Basic and clinical studies have shown that vagus nerve tension is highly related with paroxysmal atrial fibrillation (PAF). Although there are many predictions about the causes of vagus hypertension, no exact mechanisms have been clearly described. The mechanisms, clinical diagnosis and treatment of cerebral embolism in atrial fibrillation have made significant progressed [1]. In recent years, our clinical study found that vascular Parkinson's disease (VP) with basal ganglia cerebral infarction in elderly patients have severe PAF or sinus bradycardia without levodopa treatment, and this syndrome with be effectively controlled after drug treatment recovery. This phenomenon remind us that cerebral infarction caused by central sympathetic nerve injury is the major reason of the higher central vagus nerve tension triggered PAF. Understanding the causes of PAF and the target of the central sympathetic neurotransmitter might provide new treatment

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method for PAF patients caused by the increased central vagal nerve tension.

## 2. Materials and methods

### 2.1. Patients

In this study, 1 483 patients with cerebral infarction who received head CT or MRI diagnosis in the hospital from January 2013 to December 2014 were selected, which included 838 males and 645 females, 38–92 years, with mean age of 78 years. A total of 1 045 (70.4%) had basal ganglia cerebral infarction, and 438 (29.6%) had non-basal ganglia cerebral infarction. The detail of cerebral infarction patients were as described in Table 1.

Patients were divided into two groups, basal ganglia cerebral infarction group and non-basal ganglia cerebral infarction group, by comparing the incidence of PAF, left atrial diameter and heart rate variability, the correlation between basal ganglia cerebral infarction and abnormal vagus nerve tension caused PAF were explored.

Patients with the following diseases were excluded from the study: (1) Persistent atrial fibrillation; (2) Cardiovascular disease: heart valve disease, congenital heart disease, congestive heart failure; (3) Liver and kidney disease: active liver disease, liver and kidney dysfunction; (4) Malignant tumor; (5) Functional failure; (6) History of dementia and mental illness; (7) Inflammation and rheumatism; (8) Acute cerebral infarction.

### 2.2. Methods

The general clinical data in these cases were collected according to the diagnostic criteria of patients with PAF, the number of patients with PAF was counted, and the dynamic electrocardiogram and echocardiography (ECG) results were collected from patients with PAF and basal ganglia.

PAF patients' criteria were as follows: patient's clinical symptoms and signs were consistent with the definition of PAF by ECG or 24-h ambulatory ECG confirmation. Atrial fibrillation usually will be self-terminated within 48 h, and the longest termination time was less than 7 d.

### 2.3. Heart rate variability analysis

Patient data were collected and analyzed by the United States PI company three-channel dynamic ECG analysis system. Man-

machine dialogue method was used to remove false and non-sinus beat. Following standards were used to analyze the heart rate variability (HRV): the standard deviation of normal RR interval, the average RR interval every 5 min, the root mean square of the difference between the adjacent RRs (RMSSD), the ratio of the number of RR intervals to the proportion of the largest RR interval (HRV triangular index). Frequency was analyzed by fast Fourier transform method, then components and ratios of high frequency (HF 0.15–0.40 Hz) and low frequency (LF 0.04–0.15 Hz) were analyzed by fast Fourier transform method.

### 2.4. Echocardiography

PAF patients' ECG results were collected, the left atrial diameter and ejection fraction were counted.

### 2.5. Dynamic electrocardiogram examination method

Dynamic electrocardiogram was collected by the United States PI company dynamic ECG system with three channels of 24 h continuous recording. The cardiac active drug was discontinued for 5 d before the examination, and no medication during the examination. The diary and related symptoms were recorded by the patients. Results were recorded and the parameters were analyzed by computer, manually corrected and edited.

### 2.6. Statistical analysis

Data expressed as mean  $\pm$  standard deviation were analyzed by the SPSS13.0 software. The *t* test was used for measured data, and  $\chi^2$  test for counted data.

## 3. Results

### 3.1. Incidence of PAF in different parts of brain and relevant clinical data

The incidence of PAF in different parts of the brain had no correlation with gender or complications such as coronary heart disease, hypertension, diabetes (Table 2).

The incidence of PAF in the basal ganglia cerebral infarction was 18.37% (192/1 045), and 13.92% (61/438) of that in the

**Table 1**

Characterization of cerebral infarction patients.

Variable	Basal-gangalia	Non-basal ganglia	Total
Gender			
Men	581	257	838
Women	464	181	645
Complications			
Coronary heart diseases	285	94	479
Hypertension	618	201	924
Diabetes	178	64	319
Age (Years)			
<59	69	49	118
60–69	147	98	245
70–79	428	151	579
>79	401	140	541

**Table 2**

Information of cerebral infarction patients with PAF.

Groups	Basal-gangalia	Non-basal ganglia	Total
Gender			
Men	101	32	133
Women	91	29	120
Complications			
Coronary heart diseases	129	38	167
Hypertension	141	43	184
Diabetes	39	13	52
Age (Years)			
<59	6	4	10
60–69	19	11	30
70–79	58	21	79
>79	109*	25	134

\**P* < 0.05, compared with non-basal ganglia, difference was statistically significant.

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