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**Clinical Observations on the Atrial Fibrillation,
Paroxysmal Tachycardia and W. P. W. Syndrome
with Special Reference to the Pathogenetic Mechanism
of the Lone Arrhythmia**

By

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The authors carried out clinical and statistical studies about 161 patients with atrial fibrillation, paroxysmal tachycardia and W. P. W. syndrome. All patients have been in-patient in Maekawa Clinic of Kyoto University Hospital from 1949 to 1958. The exact mechanism of production of these arrhythmias is not clear; theories have ascribed the main factor responsible for the arrhythmia of non-cardiac origin to the unstable nervous system, to "vegetative Dystonie", or to autonomic disturbance, but there is little definite evidence against or for these theories. Especially, the authors studied clinically these lone or idiopathic arrhythmias from the point of view of the spinal sympathetic nervous regulation of the heart. And it may be concluded that pathogenetic origin of the lone arrhythmia is disturbance of spinal sympathetic control of the heart beat.

AURICULAR FIBRILLATION occurs usually in patients with mitral valvular disease, coronary artery disease, hyperthyroidism, or hypertensive-arteriosclerotic heart disease. The occurrence of auricular fibrillation in the patients without any signs of cardiac disease is frequently reported in recent years. The incidence and nature of the auricular fibrillation without evidence of organic heart disease was studied clinically and statistically by Orgain, Wolff and White with a review of the literatures. Ever since then this condition has been described under various terms by different authors: "uncomplicated" (Orgain, Wolff and White¹⁾), "functional" (Master and Eichert²⁾; Friedlander and Levine³⁾), "benign" (Friedlander & Levine³⁾), "lone" (Evans and Swann⁴⁾) and "idiopathic" (Maurice, Rulliere, Acor and Lenegre⁵⁾).

Among the paroxysmal tachycardias, the paroxysmal ventricular tachycardia is frequently found in the presence of organic heart disease of serious type, but it is well known that the supraventricular tachycardia may occur in persons who show no evidence of organic heart disease. In a few instances ventricular paroxysmal tachycardia occurs in the absence of evidences of organic heart disease⁶⁾⁷⁾⁸⁾⁹⁾¹⁰⁾.

The exact mechanism of production of these arrhythmias is not clear; theories have ascribed it to the unstable nervous system, to "vegetative Dystonie"¹¹⁾, or to autonomic disturbance, but there is little definite evidence against or for these theories.

The authors studied clinically these non-cardiac arrhythmias from the point of view of the spinal sympathetic nervous regulation of the heart.

MATERIAL AND METHODS

The hospital records of a total of 161 patients with atrial fibrillation, paroxysmal tachycardia and W. P. W. syndrome, associated or unassociated organic heart diseases, have been analysed for the salient points relative to the spinal sympathetic control of the heart beat. All patients have been in-patients in Maekawa Clinic of Kyoto University Hospital from 1949 to 1958, in whom the diagnosis was verified electrocardiographically except for some cases with paroxysmal tachycardia.

In our clinic, the myelography was performed in addition to routine examinations in the patients with these functional arrhythmias, neuroses, bronchial asthma and the internal diseases of unknown cause. After Moljodol was injected into the sub-arachnoid space, the subject was examined for the localization of such areas where irregular obstruction to the caudal and cephalic flow of the contrast medium, filling defect or the dividing of the contrast medium stream into branches and irregularly scattered distribution of the medium were observed. And these signs were interpreted as the evidence

of arachnoid adhesions.

RESULTS

1. *Underlying heart disease.*

Persistent atrial fibrillation:

There were 126 individuals with persistent atrial fibrillation. Table I shows the relative incidence of various underlying heart diseases as presented to our cases. The majority of these cases (78.5%) had rheumatic heart disease, 9.5% had hypertensive heart disease, and 3.2% did not show any evidence of organic heart disease; the last named is so-called lone or idiopathic atrial fibrillation.

Paroxysmal atrial fibrillation: (Table I)

Five out of 17 patients with paroxysmal atrial fibrillation had no organic heart disease and 3 cases had rheumatic heart disease. In other words, individuals without organic heart disease occupied a very large proportion of the cases with paroxysmal type of atrial fibrillation; next in frequency was the group of hypertensive heart disease.

Paroxysmal tachycardia: (Table II)

There were 15 cases with paroxysmal tachy-

TABLE I CLASSIFICATION OF BASIC DISEASES OF ATRIAL FIBRILLATION

Basic Diseases	Persistent			Paroxysmal		
	Male	Female	Sum	Male	Female	Sum
Rheumatic H. D.	54	45	99	2	1	3
Mitral Stenoinuffici.	29	23	52	0	1	1
Mitral Stenosis	17	15	32	1	0	1
Mitral Insuffici.	2	0	2	0	0	0
Aortic V. D.	0	1	1	1	0	1
Combined V. D.	6	6	12	0	0	0
Coronary H. D.	5	0	5	0	1	1
Hypertensive H. D.	7	5	12	3	1	4
Syphilitic H. D.	0	1	1	1	0	1
Congenital H. D.	1	1	2	2	0	2
Thyrotoxicosis	0	0	0	0	1	1
Idiopathic	2	2	4	3	2	5
Miscellaneous	5	1	6	0	0	0
	73	53	126	11	6	17

TABLE II CLASSIFICATION OF BASIC DISEASES OF PAROXYSMAL TACHYCARDIA

Basic Diseases	Male	Female	Sum
Rheumatic H. D.	0	2	2 (1)
Mitral Stenoinuffici.	0	1	1
Mitral Stenosis	0	1 (1)*	1 (1)*
Mitral Insuffici.	0	0	0
Aortic V. D.	0	0	0
Combined V. D.	0	0	0
Coronary H. D.	2	0	2
Hypertensive H. D.	0	1 (1)*	1 (1)*
Syphilitic H. D.	0	0	0
Congenital H. D.	0	1	1
Thyrotoxicosis	0	0	0
Idiopathic	6 (4)**	3	9
Miscellaneous	0	0	0
	8	6	14

Note: * Hypertension combined with Mitral Stenosis.

** Paroxysmal Tachycardia combined with W. P. W. syndrome.

cardia. 9 cases had no evidence of the organic heart disease, 2 cases had coronary heart disease and another 2 cases had rheumatic heart disease. It means that the largest part of this group is represented by the individuals with no organic heart disease. 14 out of 15 cases with paroxysmal tachycardia were the cases of supraventricular type. The cases with episode of the ventricular rhythm at the terminal stage of the myocardial infarction were excluded.

W. P. W. syndrome: (Table III)

As Table III shows, 5 out of 7 cases with W. P. W. syndrome had no organic heart disease. 1 out of 7 had rheumatic heart disease and another one, miscellaneous disease.

TABLE IV AGE DISTRIBUTION OF THE PATIENTS WITH ARRHYTHMIA PERPETUA, PAROXYSMAL TACHYCARDIA AND W. P. W. SYNDROME.

Age	Cardiac	Idiopathic	Sum
0-9	1	0	1
10-19	3	1	4
20-29	19	3	22
30-39	29	4	33
40-49	35	6	41
50-59	33	3	36
60-69	16	3	19
70-	5	0	5
	142	19	161

2. Age.

Table IV shows the age distribution among patients with the above-mentioned arrhythmias

TABLE III CLASSIFICATION OF BASIC DISEASES OF W. P. W. SYNDROME

Basic Diseases	Male	Female	Sum
Rheumatic H. D.	1	0	1
Mitral Stenoin suffi.	0	0	0
Mitral Stenosis	0	0	0
Mitral Insuffi.	0	0	0
Aortic V. D.	1	0	1
Combined V. D.	0	0	0
Coronary H. D.	0	0	0
Hypertensive H. D.	0	0	0
Syphilitic H. D.	0	0	0
Congenital H. D.	0	0	0
Thyrotoxicosis	0	0	0
Idiopathic	4 (4)*	1	5 (4)*
Miscellaneous	1	0	1
	6	1	7

Note: * accompanied with Paroxysmal Tachycardia

TABLE V DATA ON PATIENTS WITH PAROXYSMAL ATRIAL FIBRILLATION-FLUTTER.

No.	Name	Sex	Age	Basic Disease	Since Onset	Frequency	Duration	Precipitated	Ceased
1.	O. M.	F.	34	Mitral V. D.	?	few	?	exertion	
2.	N. K.	M.	42	Mitral V. D.	during adm.	once	1 day	exertion bending	spontaneously
3.	M. S.	M.	55	Aortic Insuf., N. C. A. Subcl. Ar. Sp.	during adm.	once	2 days	digitalis	spontaneously
4.	N. K.	M.	65	Aortic Insuf. Reticulosarcoma	during adm.	once	1 day	overeating	spontaneously
5.	M. R.	M.	59	Ess. Hypertension Subcl. Ar. Sp., Ess.	2 days	once	2 days	?	spontaneously
6.	W. Y.	M.	54	Hypertension, N. C. A.	during adm.	once	3 hrs.	fatigue	spontaneously
7.	Y. H.	M.	58	Ess. Hypertension	during adm.	once	?	?	spontaneously
8.	H. J.	F.	68	Ess. Hypertension	5 m.	2/5 m.	1 day	?	spontaneously
9.	S. T.	F.	66	Coronary Insuf. Adr. Cort. Insuf.	3 m.	1/1 w.	5 hrs.	defecation exertion	quinidine 0.2 p. o.
10.	M. I.	F.	61	Myxoedema	during adm.	once	50 days	thyroidine 0.03	
11.	I. T.	M.	20	Pulm. Conus Stenosis	during adm.	once	2 hrs.	?	spontaneously
12.	I. K.	M.	24	P. D. A.	during adm.	once	hrs.	digitalis	spontaneously
13.	H. S.	F.	66	Idiopathic, Subcl. Ar. Sp.	0.5 yr.	many	1 hr.-1 w.	defecation	spontaneously
14.	I. T.	M.	54	Idiopathic, Subcl. Ar. Sp.	?	many	hrs.-days	?	spontaneously
15.	O. M.	M.	43	Idiopathic, Subcl. Ar. Sp., N. C. A.	7 yrs.	1/m.	1 hr.	adrenaline tobacco exertion	spontaneously procaine amide
16.	F. O.	M.	58	Idiopathic, Subcl. Ar. Sp.	2 yrs.	1/yr.-3/m.	hrs.	tobacco exertion	spontaneously
17.	M. F.	F.	37	Idiopathic, Subcl. Ar. Sp., N. C. A.	3 m.	innumerable	hr.	?	spontaneously luminal

with and without organic heart disease. The peak of the incidence for the arrhythmia of cardiac origin is in forties and fifties, whereas for the idiopathic arrhythmias it is in thirties and forties, but this difference is not significant statistically.

3. Precipitating factors and course of paroxysmal atrial fibrillation and paroxysmal tachycardia.

Paroxysmal atrial fibrillation: (Table V)

Paroxysmal atrial fibrillation of cardiac origin occurred in many patients while in hospital, accordingly the authors closely observed the picture of its initial attack. The precipitating factors responsible for the paroxysmal atrial fibrillation of cardiac origin were: therapeutic dose of digitalis and thyroid powder, exertion, defecation, excessive eating, etc.

The attack of the paroxysmal atrial fibrillation ceased spontaneously or with medication.

In the idiopathic paroxysmal atrial fibrillation, course was longer than in organic group and attacks tended to recur on and off over several months to several years, thus the patients were hospitalized with arrhythmia as the dominant complaint. This type of fibrillation occurred with frequency of about once a month or innumerable in some cases.

An attack lasted but for a few minutes or a few hours, and only rarely for more than a few days; in this it simulated the atrial fibrillation of

cardiac origin.

Precipitating factors were: exercise, defecation, smoking and, frequently, adrenaline test performed for the evaluation of autonomic function. The attack of this type usually stopped spontaneously as in the paroxysmal atrial fibrillation of cardiac origin.

Paroxysmal tachycardia: (Table VI)

Usually the person affected was conscious of the initial attack except in 2 cases with mitral valvular disease. The interval from the initial attack to the admission was longer in cases with non-organic type tachycardia than in cases with paroxysmal tachycardia with organic heart disease. Paroxysmal tachycardia was a transient disturbance, lasting for a few minutes to a day, usually lasting for a few hours. As Table VI shows the precipitating factors were: psychic and somatic strain, drinking, excessive eating, smoking, change in position or mode of respiration, etc., and this held for both groups, with and without organic heart disease. Spontaneous reversion to the normal rhythm usually occurred. In several cases Aschner's test or Valsalva maneuver was effective in stopping the paroxysm. 3 patients reverted to the normal rhythm following the administration of Quinidine. In cases with paroxysmal atrial fibrillation these procedures were usually unnecessary.

Precipitating factors, drugs and maneuver caus-

TABLE VI DATA ON PATIENTS WITH PAROXYSMAL TACHYCARDIA

No.	Name	Sex	Age	Basic Disease	Since onset	Frequency	Duration	Precipitated	Ceased
1.	T. A.	F.	18	Mitral V. D.	?	2-3/yr.	mins.	exertion	quinidine
2.	N. I.	F.	56	Mitral V. D. Ess. Hypertension	?	7-8/yr.	mins. hrs.	exertion emotion	Aschner luminal
3.	T. S.	M.	38	Myocardial Infarction	5 m.	11/1- 2 mons.	hrs. day	alcohol, over- eating, bath	Aschner, Valsalva quinidine
4.	K. G.	M.	73	Coronary Sclerosis	2 yrs.	3/yr.	2 hrs.	alcohol overeating	quinidine
5.	S. S.	M.	46	Myocardial Infarction	6 m.	1/mon., in- numerable	30 mins.	emotion	Aschner quinidine
6.	M. K.	F.	21	Pulm. Valv. Stenosis	5 yrs.	innumerable, 1/mon.	10-20 mins.	breath hold- ing, bending	Aschner, forced respiration
7.	G. T.	F.	48	Idiopathic, Subcl. Ar. Sp., N. C. A.	13 yrs.	1-2/yr.	4-5 hrs.	exertion emotion	forced respiration
8.	A. H.	M.	41	Idiopathic, N. C. A., Subcl. Ar. Sp.	7 yrs.	1/2 mons.	mins. 2 hrs.	tobacco emotion	spontaneous.
9.	N. S.	M.	32	Idiopathic, Subcl. Ar. Sp.	15 yrs.	many	1-2 hrs.	emotion, bend- ing forward	breath hold. pressure at epigast. ar.
10.	N. K.	F.	11	Idiopathic,	1 day	once	1 day	exertion	spontan.
11.	N. C.	F.	37	Idiopathic, Subcl. Ar. Sp., N. C. A.	yrs.	1/w.-mon.	mins.-hrs.	emotion	spontan., Aschner luminal
12.	S. H.	M.	27	W. P. W. Syndr., N. C. A., Subcl. Ar. Sp.	14 yrs.	2-3/yr.	mins.	fatigue	spontan.
13.	N. T.	M.	29	W. P. W. Syndr., N. C. A., Subcl. Ar. Sp.	1 yr.	many	20 mins.	alcohol fatigue infection	spontan.
14.	T. Y.	M.	21	W. P. W. Syndr., Subcl. Ar. Sp.	5 yrs.	1/mon.	mins.	exertion	spontan.
15.	U. N.	M.	69	W. P. W. Syndr., Subcl. Ar. Sp., N. C. A.	?	1/6 mons.	mins.	?	forced respiration drink water

TABLE VII PRECIPITATING FACTORS OF PAROXYSMAL TACHYCARDIA (14 CASES) AND PAROXYSMAL ATRIAL FIBRILLATION (12 CASES).

Factors	Paroxysmal Fibrillation	Paroxysmal Tachycardia	Sum
Physical Exertion	4	5	9
Emotional Stress	0	6	6
Overeating	1	2	3
Defecation	2	0	2
Bending Forewards	1	2	3
Holding Breath	0	1	1
Fatigue	1	2	3
Infection	0	1	1
Alcohol	0	3	3
Tobacco	2	1	3
Adrenaline	2	1	3
Digitalis	3	0	3
Thyreoidine	1	0	1

TABLE VIII DRUGS AND MANEUVERS CAUSING THE CESSATION OF THE ATTACK OF PAROXYSMAL TACHYCARDIA (16 CASES) AND PAROXYSMAL ATRIAL FIBRILLATION (17 CASES)

Conditions	Paroxysmal Fibrillation	Paroxysmal Tachycardia
Quinidine	1	3
Procaine Amide	1	
Phenobarbital	1	2
Aschner's Test	1	5
Czermak's Test		1
Valsalva Maneuver		1
Holding Breath		1
Forced Respiration		3
Others		2
Spontaneous	13	6

ing the cessation of the attack are summarized as Table VII and VIII.

Case Reports

Case 1: H. I. Lone atrial fibrillation.

A 44-year-old woman. She was admitted to our clinic with chief complaints of fatigability, precordial oppression, paresthesia in the palms and the attacks of nocturnal dyspnea. Her family history was that her father had arrhythmia and her mother and sister died of apoplexy. Her past history included abortion at the age of 29 with negative Wassermann reaction, extirpation of uterine myoma at the same age and lumbago at the age of 40.

Present illness: She was told that her pulse was irregular when she had lumbago 4 years before admission. 50 days before admission she felt fatigued and a month before she suffered from fatigue in the lower extremities after exertion, substernal oppression and insomnia. 20 days before admission she noticed edema on the legs, since then substernal oppression increased and she had to take bed rest. In addition to these symptoms she noticed slightly edematous and flushed face and paresthesia on the tongue tip and the palms.

Findings at the time of admission (Oct. 20, 1958): Height 159.5cm. Weight 69.0kg. The pulse rate was 59 per minute and irregularity was found. Aschner's test negative. Czermak's

Fig. 1. Inoue H. Age 44. Female
Diagnosis: Lone Arrhythmia Perpetua (Atrial Fibrillation)
Subcl. Arachnitis Adh. Spinalis Th.

Fig. 2.

test negative. There was normal cardiac dullness without murmur (Fig. 1). Blood pressure was 92/50, 50 mmHg in the right and 104/64, 64 mmHg in the left. Venous pressure was 83 mmH₂O. The electrocardiogram on admission revealed auricular fibrillation with the depressed ST segment (Fig. 2). There was augmented ST depression in V₃, V₄, V₅ & V₆ with Master's two step test. Schellong's test normal. The capillaries of finger nail beds were tortuous and the blood flow was granular and slow. Erythrocytes 3.91 m., Hemoglobin (Sahli) 83%. Leucocytes 7800 (neutrophils 71.5%, eosinophils 6.5%, monocytes 1.5%, lymphocytes 20.5%). MBSR. 2.5. Serum Na 141.5 mEq/L, K 4.53 mEq/L. Serum protein fractions: total protein 7.9 g/dl, albumin 4.03 g/dl, alpha-globulin 0.52 g/dl, beta-globulin 0.91 g/dl, gamma-globulin 1.56 g/dl. Serum cholesterol: total 142 mg/dl, ester 92 mg/dl. Meulengracht index 10, CoR 6, CdR 6. BSP. 10-15% at the end of 30 minutes. BMR. -6.2%. Urine was normal. PSP. 24% at the end of 15 minutes. Fishberg's concentration and dilution test normal. The ophthalmological examination revealed no abnormalities. Atropine test negative. Epinephrine test and norepinephrine test abnormalities of (++) degree. Pilocarpine test positive. 4 hrs. epinephrine test -30%. There was sudden localized fall of skin temperature within the dermatome of Th₂₋₃ ventrally. There was no sensory disturbance, with reduced abdominal reflex and Mayer's reflex. In the myelograms there were signs of arachnoid adhesion at the vertebral levels of "CII-IV, DII, IV, VII, VIII" dorsally and "CV, DIII-XII" ventrally (Fig. 3). Cerebrospinal fluid was normal.

She was a patient with auricular fibrillation

Fig. 3. Myelogram of Case 1. Dorsal aspect at the upper thoracic level of the subarachnoid space: there are spotted obstructions at the caudal flow of Moljodol, and especially in DII, IV Moljodol didn't fall at the vertical position. Tilting of the table: horizontal.

Fig. 4. Electrocardiogram of Case 1 improved to normal range after Quinidine therapy.

without pulse deficit and who had attacks of dyspnea and precordial oppression. Her symptoms regressed gradually during her staying in the hospital. 4 weeks after admission her liver function improved to Meulengracht index 5, CoR 5 and CdR 6. Only once she experienced nausea, dizziness, girdle sensation on the upper chest and sighing respiration. She was given Quinidine orally and it was increased gradually to 1.6g a day. Her ECG. was improved to normal range (Fig. 4) when the arrhythmia was converted to a normal sinus rhythm 20 days after, but her precordial oppression did not improve, which regressed with the warmth

application.

Case 2: M.O. Lone paroxysmal atrial fibrillation.

A 43-year-old man. He was admitted to our clinic (Septem. 15, 1956) with chief complaints of intermittent precordial oppression and dull pain and tachycardia with arrhythmia. His family history was not contributory. His past history included the amebic dysentery at the age of 32 and pulmonary tuberculosis at the age of 35. At the age 35 he had gained in weight from 53 kg. to 64 kg. Next year (7 years before admission) he sometimes felt slight precordial oppression and cold sensation with

Fig. 5. Oka M. Age 46. Male
Diagnosis: Lone Transient Atrial Fibrillation
Subcl. Arachnitis Adh. Spinalis Th.

arrhythmia. These attacks occurred at intervals of several months and duration was obscure. It used to stop spontaneously with the bed rest and the patient gradually became attack-free. Attacks recurred again 2 years before admission. 1 year later he gave up smoking, although he used to smoke 60 to 80 cigarettes a day before this. His weight increased to 72 kg. Since 2 months before admission he felt precordial dull pain with or without arrhythmia or tachycardia. Attacks of tachycardia were noted to be precipitated most often by the physical exertion.

Findings at the time of admission: Height 156.8 cm., Weight 68.6 kg. Examination revealed a man with a blood pressure of 120 mmHg systolic and 70–60 mmHg diastolic. The pulse rate was 74 per minute and the irregularity of rhythm was not observed. Aschner's test negative. The cardiac dullness was nearly normal and no murmur was audible (Fig. 5). Roentgenologically the chest was normal. The electrocardiograms on admission were normal excepting slight ST depression in CR₅

Fig. 6. Myelogram of Case 2.
Dorsal aspect of subarachnoid space at the cervicothoracic region: there are irregular small spotted droplets of Moljodol caused by disturbances of the caudal flow.
Tilting of the table: 15 degrees.

by Master's single and double two step test. The lungs and abdomen were normal. There were no abnormal findings in urine. The blood picture was nearly normal except for shift to the left in hemograms. MBSR 22. Meulengracht index 5.5. CoR 4, CdR 8. BSP. 7% at the end of 30 minutes. Serum protein fractions: increased total protein, alpha, beta and gamma globulin and decreased albumin. Serum Na 134.5 mEq/L, K 4.2 mEq/L. 4 hours epinephrine test —43.4%. Glucose tolerance test normal. PSP. normal. Serum cholesterol: total 189.2 mg/dl, ester 121.6 mg/dl. The ocular fundi showed no abnormal findings. Pulsation of peripheral arteries was normal. BMR. 8.5%. There was pain in intercostal spaces on compression. There were no sensory disturbances and no pathological reflex. In the myelogram there were signs of arachnoid adhesions at the vertebral levels of "CIII, IV, VI, VII, DI, III, IV, VI–IX" dorsally and "CIV–DI, III–VII" ventrally (Fig. 6).

During his admission attack occurred twice. During attack the blood pressure was 140/80 and 118/70, respectively, with the pulse rate of 120–90. The electrocardiogram during the attack revealed atrial fibrillation with ventricular premature beats. Before admission, similar attacks used to occur at intervals from 1 to 2 months and it was precipitated by the physical and emotional strain, overeating or exhaustion. It didn't occur after taking a walk, cycling or Master's two step test. While under the author's observation the first attack occurred after going out with his family, but the rhythm returned spontaneously to normal 1 hour later. On another occasion, after he caught cold and had emotional strain in the afternoon and drank a large quantity of water in the evening, the attack occurred immediately following a change of position and he was given procaine amide intravenously, which established a normal sinus rhythm (Fig. 7). His symptoms of precordial oppression and dull pain were independent of electrocardiograms of existence or arrhythmia.

After leaving hospital, he still felt slight precordial oppression. Since 6 months before the second admission, he experienced dizziness, and since 3 months before the admission, there occurred arrhythmia.

Findings at the time of second admission (Septem. 23, 1959): The blood pressure was 112/75, 68 mmHg. The pulse rate was 68 per minute, and irregularity was not observed. The electrocardiograms on admission were normal including Master's two step test. On the 2nd day of admission he experienced cold sensation and arrhythmia. On this occasion the electrocardiogram revealed the auricular fibrillation with ventricular

Fig. 7. Oka M. Age 46. Male Diagnosis: Lone Transient Atrial Fibrillation
Subcl. Arachnitis Adh. Spinalis Th.

premature beats. Valsalva maneuver and phenobarbital were ineffective and the attack subsided spontaneously 5 hours later. Serum Na, K, Ca, Cl, creatinine, glycerophosphatic transaminase, glycerooxaltic transaminase, C-reactive protein, anti-streptolysin-O titre, alkaline phosphatase, serum total protein, serum cholesterol, 17 ketosteroid in urine were all normal. BSP. 10-15% at the end of 30 minutes. Serum protein fractions: slightly decreased albumin, increased alpha, beta and gamma globulin. Catechol amines in urine: increased adrenaline excretion. 17 OHCS: total 1.97 mg/dl, free 0.13 mg/dl. BMR. -12.0%. Renal clearance test and PSP. normal. MBSR. and Schellong's test normal. Streptococci were demonstrated in the throat culture. Cerebrospinal fluid pressure was 225 mmH₂O and it fell to 170 mmH₂O with removal of 7.5 cc of fluid. The precordial pain was relieved by lowering the spinal fluid pressure. Spinal fluid nearly normal. With 700 gamma adrenaline, the pulse rate increased to 90 per minute and electrocardiogram revealed ST depression in V₄, V₅ & V₆. With noradrenaline the pulse rate and the electrocardiogram were not changed, but he felt that the attack of arrhythmia may occur at any moment and he kept absolute bed rest that night.

Case 3: H. N. Lone supraventricular paroxysmal tachycardia.

A 32-year-old man. He was admitted to our clinic (March 6, 1954) with chief complaints of attack of tachycardia and palpitation and precordial oppression. His family history was not particular except that his grandfather and aunt died of apoplexy. His past history included tonsillectomy at the age of 8 and paratyphoid fever A at the age of 23. He smokes 10 cigarettes a day.

Present illness: At the age of 16, when he caught cold, he had sudden attack of tachycardia with cough and bloody sputum. Thereafter he sometimes developed attacks of tachycardia with palpitation. 9 months before admission an attack occurred after playing baseball and since then attacks became frequent. These attacks, of abrupt onset and abrupt end, were usually of two hour's duration, and it was precipitated most often by the emotional tension, dropping his head or lying on the left, and preceded by the precordial discomfort. They usually stopped after the patient assumed the bed rest, or attempted an maneuver of stopping respiration and pressing the epigastrium. During an attack he became nervous and pale, and at the terminal stage of attacks the urination occurred several times. At the age of 22 he served in the army, but he has been in good health. He could not swim because of dyspnea.

Findings at the time of admission: Height 169.5 cm, Weight 52.4 kg. The pulse rate was 65

per minute without irregularity of rhythm. There was nearly normal cardiac dullness with no cardiac murmur. Blood pressure 128/75, 69. The electrocardiogram on admission including Master's test was normal except for PQ prolongation (0.22"). The normal cardiac rhythm turned to the nodal rhythm with eye ball compression. Cardiac catheterization revealed no abnormal findings. The chest and abdomen were normal. There was pain on oppression and percussion in the fourth and fifth dorsal spinous processes. There was no edema. Urine no abnormal findings. Erythrocytes 5.39 m., Hemoglobin (Sahli) 112%, leucocytes 6400 (neutrophils 54.5%, eosinophils 9.5%, monocytes 5.5%, lymphocytes 30%). MBSR 4. Wassermann reaction negative. Serum Na 136.5 mEq/L, K 4.5 mEq/L. Serum protein fractions: slightly increased albumin and alpha globulin. Kepler-Power's water test disturbed. 4 hours epinephrine test --35.5%. Pilocarpine test three positive. With adrenaline, the patient became nervous like before an attack. Cerebrospinal fluid pressure was 250 mmH₂O and

Fig. 8. Myelogram of Case 3.
Ventral aspect of subarachnoid space: there are many droplets of Molybdenum scattered by disturbance of caudal flow at the upper thoracic level.
Tilting of the table: 30 degrees.

Fig. 9. Nakashoji H. Age 32. Male
Diagnosis: Supraventricular Paroxysmal
Tachycardia, Subcl. Arachnitis
Adh. Spinalis Th.

it fell to 150 mmH₂O with removal of 13 cc of spinal fluid. Cerebrospinal fluid normal. There were reduced Mayer's reflex and abdominal reflex and raised knee jerk without sensory disturbances. The radial and ulnar nerves were tender on pressure. In the myelogram there were signs of arachnoid adhesion at the vertebral levels of "CIII-DIV" dorsally and "CIV, V, VII, DI-V, XII, LIII, IV" ventrally (Fig. 8). The pneumoencephalogram revealed signs of diffuse superficial atrophic changes in the left parietal lobe and cerebellum.

The attacks occurred repeatedly while in the hospital. The electrocardiograms on attacks revealed supraventricular paroxysmal tachycardia (Fig. 9). On one occasion the ventricular rate was 180 per minute and serum Na was 149.1 mEq/L and K was 3.6 mEq/L. Serum K level during an attack was lower than in the attack-free period. The attacks subsided with the administration of Quinidine or with Valsalva maneuver and Aschner's test.

He was given Hydrocortisone to prevent the attack for a month. After this medication and "Luftmesser" treatment for the arachnoid adhesions, myelographical signs of the adhesion were reduced. After discharge, although he sometimes felt that an attack was about to occur and became nervous, actual attacks never occurred.

4. Clinical findings of the patients with atrial fibrillation and paroxysmal tachycardia.

Table IX, X summarize the findings of myelogram in addition to routine cardiac and laboratory data in cases with atrial fibrillation and par-

oxysmal tachycardia. As Table IX, X show, cardiac tests were entirely negative in patients with idiopathic arrhythmia, but in all cases there was the disturbance of passage of Moljodol through the subarachnoid space myelographically. In other

TABLE IX CLINICAL FINDINGS OF THE PATIENTS WITH ATRIAL FIBRILLATION

No.	Blood Pressure	Pulse Rate	BMR (%)	Serum Cholesterol	ECG. free from attack	Vertebral seg. with abnormal Myelographic find.	
						Ventral	Dorsal
1.	100/62, 55	72	—	—	R. a. d.	—	—
2.	90/70, 64	72	—	289.2 179.1	CR1-3 ST dep.	—	—
3.	140/36	72	—	—	CR1-4 T flat	DV, VIII, X	CVI, VII, DI, II
4.	135/40, 0	105	—	201.2 103.4	CR4-6 ST dep.	—	—
5.	158/110	64	-23	—	CR4, 5, 6, ST dep.	DI-III, X-XII LI-V DX-LV	—
6.	162/96	66	-10.8	172.2 116.5	L. a. d.	CI-DI, DIV-LIV	CI-IV DII-V, VII
7.	242/104	80	—	221.0	I, II, III, CR4 T flat CR5, 6 ST dep.	—	—
8.	192/90	100	25	155.2 89.2	PQ prolong. V5, 6 ST slight dep.	—	—
9.	150/90, 70	98	—	—	V3-6 T flat P: 0.12	—	—
10.	104/70	46	-33	272.4 131.0	Low voltage T flat	CV, DI, V-VIII, XII, LI-V	LII-V
11.	104/60	95	—	—	Normal	—	—
12.	170/10	140	—	—	CR6 T inversion	—	—
13.	126/74	64	—	219.0 89.4	Normal	CII-VI, DI, II	—
14.	87/53-48	52	—	—	L. a. d.	—	—
15.	120/76, 70	74	8.5	189.2 121.6	Normal	CIII, IV, VI, VII, DI, III, IV, VI, IX	CIV-DI, III-VII
16.	120/70	52	-10.4	209.4 104.4	Normal	CII, III, DII, III, VI-VIII	DIII-VIII
17.	92/60, 58	80	—	—	Normal	CII, III, VI, VII, DII, III, V, X-XII, LII-IV	not abnorm.

TABLE X CLINICAL FINDINGS OF THE PATIENTS WITH PAROXYSMAL TACHYCARDIA

No.	Blood Pressure	Pulse Rate	ECG. free from attack	Vertebral seg. with abnormal Myelographic find.	
				Ventral	Dorsal
1.	100/72, 60	70	R. Vent. hypertr., Aur. dilatation	—	—
2.	200/96, 92	52	L. vent. hypertr. with strain Aur. dilatation	—	—
3.	116/82	62	I, II, III, CR4, 5 T inversion CR6 ST dep.	—	—
4.	65/40, 25	75	Low voltage, B. B. B.	—	—
5.	85/?	145	I, II, III T flat, CR2 T sharp, CR5, 6 ST elevation, CR5, 6 T flat	—	—
6.	140/68, 59	96	R. a. d., II, V3-6 ST depression	—	—
7.	130/92, 90	82	R. a. d., Aur. dilat., I, II T inversion CR4, 5, 6, ST dep.	—	DV, VIII, IX
8.	108/70, 65	85	T sharp	DVI-VII	DV, VI
9.	146/91, 81	95	PQ prolongat. (0.22")	not abnorm.	CIV, VII DI-V, XII, LIII, IV
10.	108/65, 60	123	L. a. d., CR3 ST dep. CR4, 5, 6 ST dep. & T inversion	not abnorm.	DII, III
11.	122/76, 70	72	L. a. d. CR3 ST dep.	CIII-V	DII, III, VI, VII, IX, XII
12.	133/80, 76	72	W. P. W.	—	CIII-VI, DVIII, LI
13.	112/76, 68	68	W. P. W.	DIII-VI	CIV-VI, DII-IV, VII-X
14.	120/80, 70	66	W. P. W.	—	—
15.	175/100, 90	85	W. P. W.	LIII, IV	CII, III, DVI

words it was found that there were subclinical adhesive arachnoiditis at levels of the spinal cord corresponding to the spinal origin of the cardiac nerves.

The authors studied the myelographic findings statistically in 16 cases with paroxysmal tachycardia without organic heart disease or with idiopathic atrial fibrillation. Fig. 10 and 11 show the frequency of abnormal findings at respective vertebral levels, dorsally and ventrally. Dotted line shows the curve of frequency of arachnoid adhesions in the random control group. There was a higher incidence of arachnoid adhesions at the upper thoracic levels both dorsally and ventrally in cases with idiopathic arrhythmia than in the random control group. There was a statistically significant difference between the two groups as to the frequency of incidence of the arachnoid adhesion dorsally at the upper thoracic level.

DISCUSSION

The authors observed that atrial fibrillations were associated with rheumatic heart disease in the greatest member of cases (81%), then with hypertensive heart disease in 10%, with coronary heart disease in 4% and with no cardiac damage in 3%.

It will be noted that the greatest number of cases had rheumatic heart disease and next in frequency was the hypertensive-arterio-sclerotic heart disease. In Japan, Ando¹²⁾ reported that, of 772 cases of arrhythmia perpetua, the condition was complicated with valvular diseases in 53%, by myocardial damage in 23%, by hypertension in 20%, by Morbus Basedowi in 1% and uncomplicated in 3%. On the contrary most of cases

Fig. 10. Distribution of Frequency of Myelographic Findings with Reference to Vertebral Levels in 16 Cases with Non-cardiac Arrhythmia (Ventral).

Fig. 11. Distribution of Frequency of Myelographic Findings with Reference to Vertebral Levels in 16 Cases with Non-cardiac Arrhythmia (Dorsal).

reviewed in the European and American literatures¹³⁾ had the coronary heart disease and next in frequency was rheumatic heart disease. This fact coincides with the result of the statistical survey conducted on some types of heart diseases in Japan by the Japanese Circulation Society¹³⁾. According to that statistics, 2.4% of all in-patients or 24.7% of cardiac in-patients were afflicted with rheumatic heart disease, and 0.5% of all in-patients or 4.4% of cardiac in-patients were afflicted with myocardial infarction. Such results may suggest that in Japan coronary sclerosis as the basic disease of atrial fibrillation are encountered lesser degree than in Europe and America.

However, concerning the incidence of so-called lone atrial fibrillation, author's result coincides with others. Parkinson and Campbell¹⁴⁾, Mohler and Lintgen¹⁵⁾, Fowler and Baldrige¹⁶⁾, Friedlander and Levine³⁾, Orgain, Wolff and White¹⁾ have agreed that approximately 5-6% of all atrial fibrillations (including both persistent and paroxysmal fibrillation) occurs in normal hearts.

Among 17 cases of paroxysmal atrial fibrillation there were 5 cases (39%) in whom no other signs of cardiac disease were demonstrable. The ratio of lone persistent atrial fibrillation to lone paroxysmal one is 4:5 in our statistics and said to be 1:2 by the above mentioned authors.

In 1948 Herrmann and Hejtmancik⁸⁾ analysed 20 cases of paroxysmal ventricular tachycardia with reference to type of underlying heart disease and concluded that acute myocardial infarction was present in 8 of the patients, 6 having coronary sclerosis without infarction and 2 cases being patients with apparently normal heart. In other words the paroxysmal ventricular tachycardia developed most often in patients with coronary artery disease. The results of these investigators are at variance from our present result. According to Herrmann, Park and Hejtmancik¹⁰⁾ (1959), underlying arteriosclerotic heart disease was found in 42 out of 59 patients (71%), rheumatic heart disease in 12% and no organic heart disease in 10%. Another

analysis was reported of 36 cases of paroxysmal tachycardia by Williams and Ellis⁷⁾ in 1943. In 32 out of 36 cases arteriosclerotic and hypertensive heart disease was present, and underlying rheumatic heart disease was present in 1 case. And in 1 case no organic heart disease was revealed. 43 out of 47 cases with paroxysmal tachycardia observed by Jones²⁰⁾ had no history or physical findings of organic heart disease. The precipitating factors in Jones' cases were similar to those of our cases. Armbrust and Levine⁹⁾ studied 107 patients with paroxysmal ventricular tachycardia in 1950. In 79 out of 107 cases (74%) the underlying heart disease was the coronary artery disease, 9 patients (8%) had rheumatic heart disease, 13 cases (12%) no heart disease, and 5 (5%) W. P. W. syndrome. Among our patients with paroxysmal tachycardia the incidence of underlying coronary heart disease was low, possibly because there was a low incidence of the coronary artery disease, especially the myocardial infarction in our country. According to Hayward²¹⁾, two thirds of patients with supraventricular tachycardia had no organic heart disease and 80% of patients with ventricular tachycardia had serious organic heart disease.

Grishman et al²²⁾ reported 7 cases with W. P. W. syndrome. 5 out of 7 cases had no evidence of organic heart disease, 4 out of these 5 cases associated with paroxysmal tachycardia. These results agree with the data reported by Wolff, Parkinson and White in 1930. Most patients reviewed in literatures had W. P. W. syndrome with paroxysmal tachycardia. This fact is important not only clinically, but for understanding the etiology or mechanism of W. P. W. syndrome. Since Wolff, Parkinson and White²³⁾ insisted that the cause of W. P. W. syndrome might be the congenital one, there have been many literatures on W. P. W. syndrome in new born and in infant²⁴⁾²⁵⁾²⁶⁾²⁷⁾. But according to Levine²⁸⁾ and Grishman²²⁾ it occurs in every age.

As above mentioned, although incidence of underlying heart disease of atrial fibril-

lation, paroxysmal tachycardia and W. P. W. syndrome may be different in some respects with authors in different nations and clinics. There is agreement between these previous authors and the present authors on the point that some numbers of patients with persistent atrial fibrillation, paroxysmal atrial fibrillation, paroxysmal tachycardia and W. P. W. syndrome had no evidence of organic heart disease. And in the same ascending order one may find the increase in their occurrence and well-defined precipitating factors, the effectiveness of drugs or maneuvers in stopping the attacks.

The precipitating factors were, as shown in Table VII, physical and emotional strain, tabacco, change of position or respiration, drug poisoning, etc. In patients with paroxysmal tachycardia eye ball compression (Aschner's test) or Valsava maneuver was effective in stopping the attack (Table VIII). These facts may support the concept that the underlying mechanism of the idiopathic arrhythmia is concerned with the extrinsic nerve factor or neurogenic factor. As far the influence of the extrinsic nerves to the cardiac arrhythmia, there are numerous experiments in addition to the classic experiments by Herring²⁹⁾, Rothberger & Winterberg³⁰⁾, Kure³¹⁾ and Kure & Hata³²⁾ with regard to the vagal and accelerator nerve.

Concerning the central autonomic nervous system, Beattie, Brow & Long³³⁾ reported arrhythmia which occurred in the decerebrate animal. Walker³⁴⁾ reported electrocardiographic pattern, resembling W. P. W. type, obtained experimentally by stimulating the sympathetic nervous system with an injection of potassium phosphate solution into cisterna magna.

About the spinal regulation of cardiac function, the authors³⁵⁾ and Matsuda³⁶⁾ could reproduce all sorts of the clinically observed arrhythmia in dogs by electrical stimulation of their spinal sympathetic nerve; Ono³⁷⁾ obtained supraventricular paroxysmal tachycardia in rabbits with injection of alumina cream into subarachnoid space at the upper thoracic level.

Considering such experimental results and results of the myelographical study which showed a statistically significant difference in the incidence of arachnoid adhesions between the patients with idiopathic arrhythmia and random control group at the upper thoracic segments, such subclinical adhesive arachnoiditis may be responsible for the occurring idiopathic arrhythmia. It may be that some changes occur in the spinal center of the cardiac nerve and its disturbed control evoke the arrhythmia. Morimoto³⁸⁾ found degenerative changes of lateral horn cells in the spinal cord of patients with subclinical arachnoiditis adhesiva. In other words, the idiopathic arrhythmia may be dealt with as a clinical entity under the concept of subclinical arachnoiditis spinalis thoracalis.

It was already reported that the subclinical arachnoiditis may play an important part in the pathogenetic mechanism of neuroroses³⁹⁾⁴⁰⁾⁴¹⁾, essential hypertension⁴²⁾⁴³⁾, bronchial asthma⁴⁴⁾ and other diseases of yet undetermined cause as well as idiopathic arrhythmia.

Concerning the arrhythmia the vagal nerve has a important significance. Classic clinical or experimental studies about the arrhythmia were performed about the vagal nerve. Now the authors are performing the study on the role of the vagal nerve in pathogenesis of the lone arrhythmia.

SUMMARY

The authors carried out clinical and statistical studies about 161 patients with atrial fibrillation, paroxysmal tachycardia and W. P. W. syndrome. All patients have been in-patient in Maekawa Clinic of Kyoto University Hospital from 1949 to 1958. Especially, the authors studied clinically these lone or idiopathic arrhythmias from the point of view of the spinal sympathetic nervous regulation of the heart.

1. 78.5% of 126 cases with persistent atrial fibrillation had rheumatic heart disease as underlying disease, 9.5% had hypertensive-arteriosclerotic heart disease and 3.2% did not show any evidence of organic heart

disease.

2. 5 out of 17 patients with paroxysmal atrial fibrillation had no organic heart disease, 3 cases had rheumatic heart disease and 5 cases had hypertensive-arteriosclerotic heart disease.

3. 9 out of 15 patients with paroxysmal tachycardia had no evidence of organic heart disease, 2 cases had coronary heart disease and another 2 cases had rheumatic heart disease.

4. 3% of patients with persistent atrial fibrillation, 30% of paroxysmal atrial fibrillation, 65% of paroxysmal tachycardia and 70% of W. P. W. syndrome are lone.

5. In the same ascending order, one may find well-defined precipitating factors, the effectiveness of drugs or maneuvers in stopping the attacks.

6. The authors studied the myelographic findings statistically in patients with lone arrhythmias. There was a higher incidence of arachnoid adhesions at the upper thoracic levels and lower incidence of adhesions at the lower thoracic levels both dorsally and ventrally in cases with idiopathic arrhythmia than in the random control group. There was a statistically significant difference between the two groups as to the frequency of incidence of the arachnoid adhesion dorsally at the upper thoracic level.

It may be concluded that pathogenetic origin of the lone arrhythmia is disturbance of spinal sympathetic control of the heart beat.

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