PAROXYSMAL ATRIAL FIBRILLATION WITH ASYSTOLE AND SYNCOPE:
REPORT OF A CASE OF SINUS NODE DYSFUNCTION
WITH HYPOKALEMIA AND HYPERTENSION

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THE various clinical and electrocardiographic manifestations of disorders of sinus node function include sinoatrial block, sinus pauses or sinus arrest, sinus bradycardia, and the brady-tachy arrhythmia syndrome. These constitute the sinus node dysfunction. It has gained clinical importance due to several reports ascribing Stokes-Adams attacks to this syndrome. It is the purpose of this report to present a patient with paroxysmal atrial fibrillation or supraventricular tachycardia followed by asystole with syncope and S-A block whose episodes seemed to be correlated with hypokalemia and disappeared with normalization of the serum potassium level.

CASE REPORT

A 49 year old man was admitted to Kyoto University Hospital on August 6, 1971, with complaints of palpitation and syncopal attacks. The patient had hypertension for 10 years which had been treated until July, 1971, with reserpine (Sesasil®) and hydralazine (Aresoline®) and thiazide (Flutran®) with no favourable effect. For 5 years he had daily attacks of palpitation, tachycardia and dyspnea which occurred at rest or on exertion and lasted 2-8 hours. Polyuria usually followed the onset of the attacks. During the year before admission he tended to develop faintness at the end of the episode of tachycardia. On August 2, 1971, while at work, he lost consciousness and fell down for a few seconds when the tachycardia stopped. Then he was admitted to Kyoto University Hospital on August 6, 1971. There was no history of chest pain, myocardial infarction, heart failure, rheumatic fever or syphilis.

Physical examination revealed a well-nourished, rather small man. The pulse rate was 60 per minute and regular. Blood pressure was 200/100. No puffiness of the eyelids was noted. The cardiac dullness was within normal limits. Heart sounds were clear. No abnormal findings were present in the lungs. The liver was not palpable. No pitting edema was observed in the legs.

Laboratory findings on admission were as follows. Chest X-ray revealed infiltration in the right upper lung field, but no cardiomegaly. Tomography of chest X-ray showed many bullae in the right upper lung field, but no active findings of tuberculosis. Thyroid function was normal. Urinary 17-KS and 17-OHCS were nor-

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S. T. 49-y. o. male Feb. 17, 71

Fig. 3.

nal. CRP and latex RA were negative. Anti-
streptolysin O titer was 13 Todd unit. Fundi were
KW-1. The UCG showed no thrombus echo in
the atrium but showed a pattern of the mitral
movement with low amplitude. The cardiac in-
dex was 3.46 L/min/m² by radiocardiogram.
Venous pressure was 55 mmH₂O. The plasma
renin activity was 0.17 ng/ml/hr (normal,
1.02 ± 0.21). The plasma aldosterone level was
49 ng/100ml (normal, 10-20). After a low salt
diet (2g/day) and a low potassium, diet (60 mEq/
day), the plasma renin activity was
0.14 ng/ml/hr. After three hours in the upright
posture it was found low (0.16 ng/ml/hr), and
the plasma aldosterone level was 174 ng/100ml.
The plasma renin activity during administration
of spironolactone 75 mg (Aldactone-A® 3 tab)
daily was 4.43, 0.14, and 2.83 ng/ml/hr.

An electrocardiogram revealed normal sinus
rhythm at a rate of 60 with P-R interval of 0.12
seconds, QRS axis of 5°, and U waves in leads
V₂, V₃ and V₄, and slight S-T depression in leads
II, aVF, V₅ and V₆ (Fig. 1). Master's single two
step test of electrocardiogram was negative.
Episodes of tachycardia were caused by atrial
fibrillation (Fig. 2) and supraventricular tachy-
cardia (Fig. 3). During the recording of an epi-
sode of tachycardia, sinus arrest for 14 seconds
was noted, followed by an escape beat and sinus
bradycardia (Fig. 4). An electrocardiogram
showed intermittent 2:1 S-Ablock (Fig. 5).
HOSPITAL COURSE (Fig. 6)
Quinidine sulfate 200 mg orally every 8 hours was started on September 2. Hypertension was successfully treated with reserpine 0.3 mg (Serpasil®) and spironolactone 75 mg (aldactone-A® 3 tab) per day. When spironolactone was discontinued on October 5, 1971, the duration of the attack of tachycardia increased and serum potassium level fell down to 3.4 mEq/L on October 18, 1971. Since December 5, 1971, spironolactone 75 mg daily was administered again, the serum potassium level became normal and the attacks of tachycardia disappeared. The pa-
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Clinical Course

![Clinical Course Diagram]

Reserpine – Serpasil®
Propranolol – Inderal® (10mg)
Spironolactone – Aldactone-A® (25mg)
Aji maline – Gilurymal® (50mg)

Fig. 6.

The patient was discharged after approximately 35 weeks admission in this hospital and returned to his daily activity under doctor’s supervision.

DISCUSSION

The mechanism of asystole in this patient is best explained by postulating that during episodes of tachycardia due to atrial fibrillation intrinsic cardiac pacemakers were suppressed, and when atrial fibrillation stopped the suppressed pacemakers did not function and asystole resulted. West3–5 reported that repetitive electrical stimulation of the SA node is followed by a brief asystole accompanied by membrane hyperpolarization of pacemaker fibers and then by a period of accelerated firing. The initial negative chronotropic effect is thought to be due to stimulus released acetylcholine and the late acceleration to a release of norepinephrine. This explanation of consequences of imposed drive is supported by the observation of Hutter and Trautwein, Furchgott et al., Vincenzi and West. It has been amply demonstrated that acetylcholine depresses the pacemaker potential and that catecholamines accelerate its rise.

The term “sick sinus syndrome1,2” was first coined by Lown6 (1967) to denote the group of unstable and chaotic supraventricular rhythm after cardioversion for chronic atrial fibrillation in patients on digitals. While Ferrer8 (1968) has reviewed extensively the pathophysiological mechanism and clinical manifestations of sick sinus syndrome. Ferrer7 stated that “the basic disorders responsible for the events characterizing the sick sinus syndrome are sinus arrest and/or sinoatrial exit block. While it is well known that vagotonia may produce these two effects, vagotonia as well as the effects of excessive potassium, quinidine, nicotine, beta blocking agents, and aerosol propellants with fluorinated hydrocarbons is a transitory influence and not consider part of the sick sinus syndrome”.

In this case, S-A block was found when quinidine sulfate were administered under normalized serum potassium level. And atrial fibrillation with sinus arrest for 2-14 seconds occurred when quinidine was not administered under serum potassium level 3.7-4.7 mEq/L. When the serum potassium level was 4.0-3.4 mEq/L, tachycardia attack happened easily (Fig. 6, Oct.13-Nov.5). When quinidine sulfate was reduced from 0.6g to 0.3g per day
and ajmaline 150 mg per day were added, the frequency of the tachycardia attacks were reduced. Moreover when the serum potassium level was normalized by spironolactone administration, the tachycardia attack disappeared. These findings are considered to indicate that S-A block is correlated quinidine sulfate and atrial fibrillation with hypopotassemia. As a whole, this case is considered to show sinus node dysfunction. The duration of the episode of tachycardia seemed to be correlated with the serum potassium level (Fig. 6). When the serum potassium level was about 3.5 mEq/L, episode of tachycardia happened easily. When the potassium level became over 4.1 mEq/L, the tachycardia attack disappeared. Therefore hypopotassemia probably triggered the episodes of tachycardia. After the administration of thiazide, the serum potassium level fell to 2.3 mEq/L on July 12, 1971, and tachycardia with syncope easily happened.

In this case the plasma renin activity was low and after a low salt, low potassium diet plasma renin activity did not increase. Plasma renin activity after three hours in the upright position was low. Hypertension was effectively treated by spironolactone (Aldactone-A®). Plasma aldosterone level was high. The urinary 17-KS and 17-OHCS were normal. Therefore this patient was suspected to have idiopathic hyperaldosteronism. He did not have adrenal gland surgery.

**SUMMARY**

A pattient is presented who developed syncope due to asystole following paroxysmal atrial fibrillation. Suppression of intrinsic cardiac pacemakers during tachycardia is postulated as the mechanism for asystole. This patient might have sinus node dysfunction. The episodes of tachycardia occurred more often when the serum potassium level was low.

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**REFERENCES**
