A Case of Ventricular Paroxysmal Tachycardia with Abnormal P Waves

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ALTHOUGH a number of papers on ventricular paroxysmal tachycardia have been published,¹⁾⁻¹⁵⁾ only few reports referred to abnormal P waves in such cases.¹⁶⁾ The purpose of this paper is to describe a case of ventricular paroxysmal tachycardia in which abnormal P waves were observed during the period free from attacks and to discuss the probable mechanism of this P abnormalities.

CASE REPORT

M.A., a 19-year-old man, was admitted to the Kyushu University Hospital on June 28, 1963, with a chief complaint of sudden attack of palpitation. The patient had been well until August 3, 1961, when he succumbed to the first attack of palpitation. The attacks occurred almost regularly with the frequency of about once per month. They were abrupt in onset and offset, and lasted for 7 to 10 days. They never continued over 11 days. They were accompanied by a great anxiety, moderate dyspnea, nausea and vomiting, but no syncope or edema. Twenty days prior to admission, he has had such an attack while sleeping. The efforts to cease the attack were in vain.

There was no history of rheumatic fever and other past illness to be relevant to his main condition.

Family history was noncontributory.

Physical examination revealed a poorly nourished man with height of 167.6 cm. and weight of 46.5 Kg. The blood pressure was 104/78 mm.Hg. The pulse rate was 135 per min. almost regular, and its tension changed alternately. The respiration was regular and the rate was 16 per min. Anemia and icterus were not visible. Tonsils were normal. The percussion over the lung fields revealed no abnormalities and respiratory sound was normal. The cardiac dullness was enlarged, but no murmurs were heard. The liver was palpable one finger-breadth below the right costal margin and was slightly tender. The spleen and kidney were not palpable. There was no edema. The neurological examination was normal.

The results of laboratory examinations were summarized in Table I.

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Table I. Laboratory Data on Admission

Hemogram :		Biochemical study of the serum	
R.B.C.....	401×10^4	Total protein.....	6.9 Gm./100 ml.
Hb	11.6 Gm. per 100 ml.	A/G	1.8
W.B.C.....	4,600	NPN	25.4 mg./100 ml.
Differential count		Na	142 mEq./L.
Neutrophils	65%	K	4.2 mEq./L.
Eosinophils.....	0%	Ca	5.4 mEq./L.
Basophils.....	0%	Cl.....	109 mEq./L.
Lymphocytes	29%	Total cholesterol	140 mg./100 ml.
Monocytes	6%	Icterus index	4
Urinalysis :		Thymol turbidity test ...	1.0
Albumin	slightly positive	SGO-T	27.0 u.
Urobilinogen	normal	SGP-T.....	24.0 u.
Serum Wassermann reaction.....		negative	
Blood sedimentation rate		2 mm./1 hr., 6mm./2hr.	
Stool specimen.....		normal

Fig. 1. Electrocardiogram taken on June 28, 1963.

Fig. 2. Analysis of this arrhythmia.

Fig. 3. The identification of P waves by the high gain electrocardiogram taken at a sensitivity of 1 mm. per 25 mv. and at a paper speed of 100 mm. per sec.

Fig. 4. X-ray picture of the chest taken during tachycardia, showing the enlarged heart.

Fig. 7. X-ray picture of the chest taken during the period free from attacks, showing the heart with normal configuration.

Fig. 5. Ventricular extrasystoles observed during treatment with ajmalin.

Clinical course and electrocardiographic findings: The electrocardiogram taken on admission revealed the rapid ventricular beating at a rate of 167 per min. and the prolonged duration of the QRS complexes (Fig. 1). Analysis of this arrhythmia showed the P waves at an independent and slower rate in comparison with QRS complexes (Fig. 2). The P waves were clearly identified by the high gain electrocardiogram (Fig. 3). The X-ray picture of the chest during tachycardia showed the enlarged heart (Fig. 4). Carotid sinus pressure and ocular pressure revealed no effect on paroxysm. The patient responded to treatments with procaine amide and ajmalin only transiently. During the treatment with ajmalin sinus rhythm restored and ventricular extrasystoles irregularly occurred among the abnormal rhythm (Fig. 5). From above findings ventricular tachycardia was considered to be most probable. The paroxysm ceased abruptly during auscultation on July 5, 1963 without any specific treatment. The electrocardiogram taken immediately after cessation revealed broad P wave, marked S-T deviation and inversion of T wave (Fig. 6). During the period free from attacks, the phonocardiographic findings and X-ray picture of the chest were normal (Fig. 7). No abnormal findings were found in further examination including psychoanalysis.

The form of P wave, marked S-T deviation and inversion of T wave changed

Fig. 6. Electrocardiogram taken on July 5, 1963.

gradually (Fig. 8). Thirty days after cessation of the paroxysm, on August 4, 1963, these findings returned almost normal (Fig. 9).

During the period free from attacks, the changes of P wave as shown in Fig. 8, were induced by exercise test, deep respiration and carotid sinus pressure (Fig. 10). The form of P wave which occurred most frequently in the clinical course was shown in the electrocardiogram taken simultaneously in limb lead at a standard sensitivity and at a high sensitivity (Fig. 11). Mere inspection in the conventional electrocardiogram led to suspicion of right atrial overloading because of tall, peaked shape and short duration of P wave in leads II and III, but precise analysis of the tracings in the high gain electrocardiogram allowed the correct diagnosis of left atrial overloading. Slow upstroke of right atrial activation which was hardly recognized in the conventional electrocardiogram was interrupted by sudden rise of left atrial activation 0.04 sec. after the P wave onset. The peak of P wave developed markedly high amplitude 0.075 sec. after the beginning of the atrial activation. The notch on the descending limb 0.095 sec. after the P wave onset would probably show the end of right atrial activation. The P wave was considerably widened, amounting to 0.135 sec. Thus the presence of marked left atrial overloading was concluded in the high gain electrocardiogram.

Fig. 8. Gradual changes of P wave, S-T segment and T wave after paroxysm in II lead.

The similar attacks recurred 4 times for 8 months during admission, but the electrocardiographic findings were essentially the same at every attack. The attacks often occurred during or immediately after taking a bath, but no other trigger mechanism was found in this case. The treatment consisted essentially of reassurance and sedation.

The patient had oliguria for a few days after the onset of paroxysm, then polyuria, often amounting to 3,400 ml. per day was observed.

The patient was discharged on February 28, 1964.

DISCUSSION

It will be almost confirmative from the electrocardiographic findings during the attacks that the paroxysmal tachycardia of this case is not supra-ventricular but ventricular. In confirming the diagnosis to be ventricular tachycardia, it is necessary to identify the P waves occurring independently to QRS complexes. However, it is often difficult. As regard to P wave

Fig. 9. Electrocardiogram taken on August 4, 1963.

Fig. 10. Changes of P waves under various conditions in II lead.

Fig. 11. Abnormal P waves recorded by the high gain electrocardiogram.

a) control b) abnormal P wave

identification, the high gain electrocardiogram is more useful than the conventional electrocardiogram.¹⁷⁾

The P wave abnormalities in paroxysmal tachycardia, especially in paroxysmal atrial fibrillation and supraventricular tachycardia, were reported by Davies,¹⁶⁾ while in this case we have noticed the gradual changes of P waves in ventricular paroxysmal tachycardia during the period free from attacks. Davies suggested that the abnormality of P wave lay in the left atrial component. In this case, though the form of P wave was variable, the left atrial overloading was revealed constantly in high gain electrocardiogram.

On the other hand, Wood reported polyuria during or at the termination of the paroxysm, and suggested that the polyuria was caused by an increase in the volume of blood in the left atrium.^{18),19)} In our case such polyuria was also observed. Polyuria in our case is considered to support the presence of left atrial overloading during or at the termination of paroxysm.

Of course, many factors in addition to hemodynamic effect, for instance ectopic pacemaker formation in the atria, should be considered. Anyway, the P wave abnormalities show the presence of conduction disturbance in the atria. In recent studies with rabbits, authors noticed that the aconitine-induced ventricular tachycardia was preceded by the P wave abnormali-

ties in the majority of the cases²⁰) and this fact will give the suggestion that the conduction disturbance in the atria is deeply connected with the development of ventricular tachycardia and in such circumstances ectopic pacemaker activity in the ventricle will be more easily to develop. In this case the conduction disturbance in the atria may be the precipitating factor for the ventricular tachycardia.

SUMMARY

A case of ventricular paroxysmal tachycardia with abnormal P waves is presented. As regard to the identification of P waves during paroxysm, the high gain electrocardiogram is more useful than the conventional electrocardiogram. The P wave abnormalities are considered to show left atrial overloading. The conduction disturbance in the atria may be the precipitating factor for the ventricular tachycardia.

REFERENCES

1. Adams, C.W.: *Am. J. Cardiol.* **9**: 215, 1962.
2. Armbrust, C.A. and Levine, S.A.: *Circulation* **1**: 28, 1950.
3. Barker, P.S.: *Heart* **11**: 68, 1924.
4. Campbell, M.: *Lancet* **2**: 641, 1947.
5. Campbell, M. and Elliot, G.A.: *Brit. Heart J.* **1**: 123, 1939.
6. Cooke, W.T. and White, P.D.: *Brit. Heart J.* **5**: 33, 1943.
7. Freundlich, J. and Vancouver, B.C.: *Am. Heart J.* **31**: 557, 1946.
8. Froment, R., Gallavardin, L., and Cahen, P.: *Brit. Heart J.* **15**: 172, 1953.
9. Foster, R.F. and Thayer, R.H.: *Am. Heart J.* **40**: 24, 1950.
10. Hair, T.E., Eagan, J.T., and Orgain, E.S.: *Am. J. Cardiol.* **9**: 209, 1962.
11. Hansen, J.E.: *Am. Heart J.* **61**: 296, 1961.
12. Herrmann, G.R. and Hejtmanick, M.R.: *Ann. Int. Med.* **28**: 989, 1948.
13. Irvin, C.W. and Cutts, F.B.: *J.A.M.A.* **146**: 1282, 1951.
14. Pick, A. and Langendorf, R.: *Prog. Cardiovas. Dis.* **2**: 391, 1960.
15. Ring, A. and Blankfein, J.: *Ann. Int. Med.* **42**: 680, 1955.
16. Davics, L.G. and Ross, I.P.: *Brit. Heart J.* **25**: 570, 1963.
17. Ohya, Z., Ohshita, K., Okada, T., Shibata, T., Takeshita, I., Hidaka, Y., Kawamura, H., and Mori, H.: *Naika* **13**: 555, 1964 (in Japanese).
18. Wood, P.: *Brit. Heart J.* **25**: 273, 1963.
19. Wood, P.: *Brit. Heart J.* **25**: 689, 1963.
20. Yanaga, T., Ito, M., and Mashiba, H.: Unpublished observation.
21. Katz, L.N. and Pick, A.: *Clinical electrocardiography I*, The arrhythmias, Lea & Febiger, Philadelphia, 1956.