Electrocardiographic Masking of a Myocardial Infarction by a Fasciculoventricular Mahaim Fiber, Atrioventricular Reentry with Anterograde Conduction over a Fasciculoventricular Mahaim Fiber and Retrograde Conduction over a Concealed Kent Bundle

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SUMMARY
An intracardiac electrophysiologic study was performed in a patient with paroxysmal tachycardia. The patient had two previous episodes of myocardial infarction, but abnormal Q waves were absent in previously obtained electrocardiograms. Electrocardiographic diagnosis of inferior and anterior infarction was made because the electrocardiogram taken on admission temporarily showed abnormal Q waves. Atrioventricular reentry with anterograde conduction over a fasciculoventricular Mahaim fiber and retrograde conduction over a concealed Kent bundle was observed during the tachycardia. It was also demonstrated that electrocardiographic masking of the Q waves was caused by conduction over a fasciculoventricular Mahaim fiber.

Additional Indexing Words:
Fasciculoventricular Mahaim fiber  Concealed Kent bundle
Electrocardiographic masking of myocardial infarction

It is widely recognized that one rare feature of the classic type of Wolff-Parkinson-White (WPW) syndrome is masking of abnormal Q waves of a myocardial infarction by its delta waves, thus making it difficult to use electrocardiographic criteria to confirm the infarction.

On the other hand, a nodoventricular Mahaim fiber with insertion into the right ventricle has been recently reported to be one of the causes of "wide" QRS tachycardia, either as an active participant into the "wide" QRS tachycardia or a passive bystander to atrioventricular nodal reentry.3,4
However, there have been no reports indicating that a fasciculoventricular Mahaim fiber plays a direct role in reentrant supraventricular tachycardia.\textsuperscript{3,4} In this report, we describe a patient in whom the electrocardiographic features of a myocardial infarction were masked by ventricular preexcitation via a fasciculoventricular Mahaim fiber and also in whom atrioventricular reentrant tachycardia reflected both anterograde conduction over a fasciculoventricular Mahaim fiber and retrograde conduction over a concealed Kent bundle.

**Case Report**

A 59 year old man was admitted to our hospital with a 4 year history of recurring episodes of palpitations. Initially, episodes of short duration

![Fig. 1. 12-lead electrocardiogram during tachycardia.](image)
occurred only rarely but the episodes had increased recently in both frequency and duration. The patient had a history of tuberculous pleuritis at the age of 21 and two episodes of myocardial infarction at the age of 40 and 54, respectively. Since the electrocardiograms during the course of myocardial infarctions were not available, the infarct sites were unknown. There was no family history of episodic palpitations, syncope, heart disease or sudden death. The patient was a well-nourished, well-developed man with a blood pressure of 110/70 mmHg and a regular heart rate of 70 beats/min. The cardiac examination and the remainder of the physical examination showed no abnormalities except diminished breath sounds over the right lower lung field. Numerous 12-lead electrocardiographic records taken during episodes of palpitations showed a regular "narrow" QRS tachycardia at a rate of 150/min, and retrograde P waves seemed to be present on the descending limb of each T wave in lead V1. No definite Q waves were seen in any lead (Fig. 1). Previously recorded 12-lead electrocardiograms during sinus rhythm showed neither abnormal Q waves nor definite delta waves, with a normal PR interval of 0.20 sec. The poor R wave progression in leads V1-V3 might imply the presence of localized anterior infarction (Fig. 2). The electrocardiogram taken on admission (Fig. 3) temporarily showed a slightly different pattern from those recorded previously; small Q waves were seen in leads II and aVF and abnormal Q waves appeared in leads III, V2 and V3. These elec-

Fig. 2. 12-lead electrocardiogram before admission. Although a very slowly rising wave is barely visible before the QRS wave in leads V4 and V5, it is almost impossible to accurately distinguish between baseline drift and a slight ventricular preexcitation from the surface electrocardiogram.
Fig. 3. 12-lead electrocardiogram on admission.

trocardiographic findings were consistent with inferior and anterior infarctions. A thallium-201 myocardial scintigraphic study at rest showed a perfusion defect in the infero-apical segment and a decreased uptake in anterolateral and inferior segments. Coronary arteriography revealed a 75% right coronary artery and a 90% proximal left anterior descending artery stenosis and left ventriculography showed an infero-basal akinetic and an anterior hypokinetic zone with a decreased ejection fraction of 0.39. These scintigraphic and angiographic findings confirmed the electrocardiographic diagnosis of inferior and anterior myocardial infarctions.

**Electrophysiologic study:**

The patient was studied in a lightly-sedated, postabsorptive state after informed written consent was obtained. The His bundle and other intracardiac electrograms were recorded by conventional catheter techniques. The posterior left atrial electrogram was obtained from a bipolar electrode catheter positioned within the esophagus. The intracardiac and esophageal electrograms were displayed simultaneously with surface electrocardiographic leads I, II and V1 on a multichannel oscilloscope (Fukuda Denshi MCM-8000, bandpass settings: 50–500 Hz) and recorded on magnetic tape (SONY UFR-6730). The recordings were subsequently reproduced on a Mingograph 804 ink-jet recorder (Siemens-Elema) and on a thermal recorder (Fukuda Denshi RF-80) at a paper speed of 100 mm/sec. Pacing stimuli were provided by a programmable digital stimulator (San-jei Sokki 3F-51) at an intensity of
approximately twice diastolic threshold and a duration of 2 msec. Left atrial pacing was performed with the esophageal electrode at a current intensity of 15 mA and a pulse duration of 15 msec.\(^8\) During sinus rhythm, the sinus cycle length, PR interval, atrioventricular conduction time (AH), infranodal conduction time (HV) and QRS duration were 880, 200, 95, 20 and 100 msec, respectively. Although these conduction intervals suggested some form of ventricular preexcitation, no definite delta wave was seen on the 12-lead electrocardiogram (see Figs. 1, 2 and 3). Atrial extrastimulus testing at a basic cycle length of 600 msec from the high right atrium showed an increase of the AH interval from 105 to 140 msec when the coupling interval of the extrastimulus was decreased from 600 to 320 msec. However, HV interval (20 msec) was not altered (Fig. 4A). This was thought to represent ventricular preexcitation via anterograde conduction over a fasciculoventricular Mahaim fiber.\(^9\) When the coupling interval was further decreased to 300 msec, the HV interval was suddenly prolonged to 50 msec, with a concomitant appearance of abnormal Q waves in lead II (Fig. 4B, arrow). This indicated that the effective refractory period of the fasciculoventricular Mahaim fiber was 300 msec, such that a small amount of ventricular preexcitation via conduction over the fasciculoventricular Mahaim fiber masked the Q wave in lead II at coupling intervals longer than 300 msec. In the absence of these electrophysiologic findings, it would be almost impossible to correctly

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**Fig. 4.** Atrial extrastimulus testing at a basic cycle length of 600 msec. In this and subsequent figures the high right atrial (HRA), His bundle (HBE) and esophageal (ESO) electrograms, electrocardiographic leads V\(_1\), I and II are recorded simultaneously and electrocardiographic lead II is recorded with the amplitude sensitivity about 1.5 times as high as standard so as to clarify the Q wave (arrow). Panel A shows recordings at a coupling interval of 320 msec and panel B at a coupling interval of 300 msec, respectively. See text for explanations.
Fig. 5. Atrial extrastimulus testing at a coupling interval of 260 msec (basic cycle length = 600 msec). The measured H₂V₂ interval is 65 msec and the deflection recorded between the His bundle (H₂) and the ventricular (V₂) electrograms may be the right bundle potential. See text for explanations.

Fig. 6. Alternating cycle length recorded during induced tachycardia. A 30 msec decrease in the HV interval (from 50 msec to 20 msec) causes a reduction in the tachycardia cycle length from 370 msec to 350 msec, indicating that tachycardia utilizes conduction over a fasciculoventricular Mahaim fiber (active participant). See text for explanations.
recognize ventricular preexcitation in the initial portion of the QRS wave in lead II from the surface electrocardiogram. Sustained supraventricular tachycardia was induced at coupling intervals ranging from 280 to 250 msec. Supraventricular tachycardia at a coupling interval of 260 msec is shown in Fig. 5. During the induced tachycardia, the cycle length was 360–365 msec and the His bundle deflections (H) constantly preceded the ventricular wave (V) by 50 msec. The earliest retrograde atrial activation was recorded in the esophageal electrogram (Figs. 5 and 6). Premature ventricular stimulations given during tachycardia were able to preexcite the atrium and reset the tachycardia when the His bundle was refractory (not shown). This finding indicated that the retrograde limb of this reentrant tachycardia is a left-sided Kent bundle. During the episode of tachycardia shown in Fig. 5, a small Q wave in lead II (arrow) was associated with the HV interval of 50 msec. However, the Q wave in lead II disappeared when the tachycardia cycle length lengthened slightly. The tachycardia in Fig. 6 showed alternating cycle length. When the cycle length was 370 msec, the HV interval was 50 msec and a Q wave was present. On the other hand, when the cycle length was 350 msec, the HV interval was shortened to 20 msec with disappearance of the Q wave. Accordingly, the anterograde limb of the reentry circuit was thought to involve the atrioventricular node and the His bundle when the HV interval was 50 msec, while the atrioventricular node, the His bundle and a fasciculoventricular Mahaim fiber were thought to form the anterograde limb when the HV interval was 20 msec. Incremental high right atrial pacing produced neither a lengthening of the HV interval (20 msec)

Fig. 7. Constant high right atrial pacing at the rate of 170 beats/min. See text for explanations.
nor an appearance of a Q wave in lead II up to the paced rate of 160 beats/min. However, there was a progressive prolongation of the AH interval. At an atrial pacing rate of 170 beats/min (Fig. 7), the AH interval lengthened progressively from 105 msec (1st paced beat) to 145 msec (3rd paced beat) and the HV interval suddenly increased to 50 msec for the 4th paced beat, with the appearance of a small Q wave (arrow) in lead II. The latter finding was thought to represent a conduction block of the 4th paced impulse over a fasciculoventricular Mahaim fiber because of the refractoriness of this accessory pathway. Transesophageal atrial pacing produced no morphological alterations in the QRS complex in surface electrocardiograms, indicating that the left-sided Kent bundle in this patient was a concealed bypass tract functioning in the retrograde direction only.8) With incremental ventricular pacing from the right ventricular apex, 1:1 ventriculoatrial conduction was maintained and the ventriculoatrial conduction time remained constant up to a paced rate of 200 beats/min, with the same retrograde atrial activation sequence during the tachycardia (earliest in the recording site of the esophageal electrogram). This finding indicated that the pacing impulses were conducted retrogradely over the concealed left-sided Kent bundle.

DISCUSSION

The diagnosis of inferior and anterior myocardial infarctions was confirmed in this patient by coronary arteriography. In addition, a manifest fasciculoventricular Mahaim fiber and a concealed Kent bundle were demonstrated to be involved in a circuit producing atrioventricular reentrant tachycardia. However, the process leading to these diagnoses was relatively complicated. Previous 12-lead electrocardiograms recorded had shown no abnormal Q waves in leads II, III and aVF (Fig. 2) and an electrocardiographic diagnosis of inferior myocardial infarction was difficult. The electrocardiographic diagnosis of inferior myocardial infarction was made incidentally because the electrocardiogram taken on admission temporarily demonstrated Q waves in leads II, III and aVF (Fig. 3). The diagnosis of anterior myocardial infarction was easy even in the previous 12-lead electrocardiogram (Fig. 2) because it displayed poor R wave progression5) in leads V1–V3, already suggesting anterior myocardial infarction. However, these findings raised the question about the nature of the conduction abnormality that produced electrocardiographic changes in the QRS wave form, namely, masking of the Q wave. In the present case, a detailed electrophysiologic study was performed in order to investigate the mechanism of the supraventricular tachycardia, and it was demonstrated that the mechanism
was atrioventricular reentry. This study also demonstrated that a slight ventricular preexcitation by conduction over a fasciculoventricular Mahaim fiber masked the Q wave and caused the electrocardiographic changes. There have been a few reports in the literature that ventricular preexcitation by a Kent bundle conduction in the WPW syndrome can mask the Q wave in cases of myocardial infarction but, to our knowledge, no case of the masking of the Q wave after infarction by a fasciculoventricular Mahaim fiber has been reported. This may reflect the fact that, in the typical WPW syndrome, it is easy to make a diagnosis of ventricular preexcitation by Kent bundle conduction from the surface electrocardiogram. However, in case of slight ventricular preexcitation by conduction over a fasciculoventricular Mahaim fiber, it is almost impossible to make a differential diagnosis from the relatively small ventricular preexcitation by Kent bundle conduction or from mere slurring of the initial portion of the normal QRS wave. In addition, while an invasive electrophysiologic is necessary to confirm a fasciculoventricular Mahaim fiber, it would be performed rarely only for diagnosing this accessory pathway. The typical electrocardiographic feature of a slight ventricular preexcitation, produced by conduction over a fasciculoventricular Mahaim fiber, is a normal PR interval either with a small delta wave or with a slightly widened QRS wave. Atypical electrocardiographic features have been reported in only a limited number of studies and include abnormal Q waves in right precordial leads and vertical or left axis deviation in the frontal plane QRS axis. Our patient also indicates that the Q wave in the myocardial infarction can be masked by a fasciculoventricular Mahaim fiber conduction.

Since the first report by Wellens et al in 1971, there have been several reported cases in the literature about reentrant supraventricular tachycardia associated with a Mahaim fiber. Almost all of the reported tachycardias were associated with a nodoventricular Mahaim fiber with insertion into the right ventricle. However, Fu et al demonstrated a fasciculoventricular Mahaim fiber with insertion into the left ventricle, but the fasciculoventricular Mahaim fiber was not involved in the reentry circuit (passive bystander) because the tachycardia mechanism was intraatrial reentry. Electrophysiologic investigations in the present case demonstrated that the fasciculoventricular Mahaim fiber was part of an anterograde limb of the atrioventricular reentrant tachycardia. This accessory pathway was not an essential component of the reentrant circuit, because atrioventricular reentrant tachycardia with anterograde conduction over the atrioventricular node was present in its absence. A similar phenomenon associated with a nodoventricular Mahaim fiber was mentioned by Tonkin et al and Ward et al, but
there have been no previous cases in the literature involving a fasciculoventricular Mahaim fiber.

REFERENCES
