VENTRICULAR fibrillation is the most serious arrhythmia encountered in the human heart and it has been estimated that ventricular fibrillation is the terminal arrhythmia in 40 to 50 percent of all patients who die, while in the remainder it is ventricular asystole.¹

Wigger and Wegria, in 1940, demonstrated in the dog the presence of a "vulnerable period" in which a repetitive ventricular firing resulted from a single localized electrical shock delivered to the ventricular muscle.² Subsequently, Smirk and Palmer have noted in man the relation between ventricular fibrillation and T wave interruption by the QRS complex, and termed it "R on T" phenomenon.³

Although the mode of onset and mechanism of ventricular fibrillation have been investigated since then, there are no published reports of such arrhythmia induced by sinus or supraventricular beat in man except in patients with pre-excitation.⁴ This paper presents a case of ventricular fibrillation induced by sinus or supraventricular beat conduct-

d during incomplete atrioventricular (A-V) dissociation following cardiac surgery.

Case Report

A 35-year-old housewife was admitted to Tokyo Medical and Dental University Hospital on December 3, 1971, complaining of shortness of breath on exertion, easy fatigability, and palpitation. She had had acute rheumatic fever at age ten years. Four years later she was told that she had an atrial septal defect. She had signs and symptoms of cardiac failure at the age of 30 and two years later became conscious of arrhythmia and palpitation.

On admission, physical examination showed a slightly accentuated first heart sound and a widely split and fixed second heart sound. An ejection type systolic murmur (4/6) was heard along the upper left sternal border, and a pansystolic murmur (3/6) and a diastolic rumbling murmur in the apical region. Chest roentgenogram revealed a markedly enlarged right atrium and right ventricle, a slightly enlarged left atrium and a normal left ventricle with bulging pulmonary arteries and signs of pulmonary hypervascularity. Cardiac catheterization data demonstrated evidences consistent with atrial septal defect with a mild degree of mitral and tricuspid insufficiency. Other laboratory examinations yielded results within normal limits. An electrocardiogram recorded prior to operation showed impure flutter and incomplete right bundle branch block, where the Q-T interval was 0.32 sec (fig. 1).

On December 8, 1971, mitral annuloplasty via the atrial septal defect and closure of the defect were accomplished during total extracorporeal bypass for 67 min, when anoxic arrest continued for 40 min.
Immediately after the operation, however, persistent mitral regurgitation was observed and reoperation was performed on the same day. The mitral valve was replaced with a Starr-Edwards prosthesis. Two hours after the operation, under assisted respiration by Engström respirator, arterial blood analysis revealed PO$_2$ 195 mm Hg, PCO$_2$ 40 mm Hg, pH 7.45, bicarbonate concentration 27 mEquiv/liter and serum potassium concentration 4.4 mEquiv/liter.

Within two hours following surgery, an electrocardiogram showed an A-V junctional rhythm with a ventricular rate of 62 beats per minute which suddenly changed to short episodes of paroxysmal atrial tachycardia with Wenckebach period (usually showing 3:2 A-V ratio) for 10 sec followed by return to the former rhythm (fig. 2A). Seven hours after the operation (3:05 a.m., December 9, 1971), a monitor showed incomplete A-V dissociation, i.e., one ventricular capture beat with an aberrant ventricular conduction following six or seven A-V junctional beats (fig. 2B). Fifteen minutes later, auscultation showed disappearance of the clicks of the prosthesis, and paroxysmal ventricular fibrillation appeared on the monitor. The paroxysmal ventricular fibrillation became repetitive at nine hours after operation (fig. 3). Lidocaine was given intravenously twice as a single

\[ \text{An electrocardiogram recorded before the cardiac operation.} \]

\[ \text{A. Lead II, continuous strip. A-V junctional rhythm changed temporarily to paroxysmal atrial tachycardia.} \]

\[ \text{B. Lead II, continuous strip. Incomplete atrioventricular dissociation.} \]
bolus of 50 mg at 5:43 a.m. and 6:43 a.m., resulting in no improvement.

At the onset of rapid repetitive ventricular discharge, pulmonary systolic pressure dropped, left atrial pressure was increased gradually and digital plethysmogram showed a rapid fall to an imperceptible level (fig. 4). Arterial blood analysis at noon of December 9, showed PCO₂ 33 mm Hg, PO₂ 148 mm Hg, pH 7.54, and bicarbonate concentration 27 mEq/liter.

Although incomplete A-V dissociation continued, ventricular fibrillation disappeared at 4:00 p.m., December 9, 1971. On December 14, 1971, the rhythm reverted to atrial fibrillation which has persisted. The patient was discharged 20 days after operation, and is well now and has returned to full activity.

Discussion

The P waves in figure 2B cannot be identified with certainty as of sinus origin because only atrial fibrillation was present before the operation. The arrhythmia in this figure may be interpreted as reciprocating rhythm induced by retrograde Wenckebach periods because of the later appearance of the P waves preceding the apparent capture (arrows). The upright P waves in lead II would not necessarily preclude retrograde conduction, but it appears more likely that we are dealing with incomplete A-V dissociation (A-V interference dissociation). Regardless of the site of the pacemaker (sinus or atrium) controlling the atria, we conclude that the supraventricular beat precipitated the ventricular fibrillation.

All QRS complexes in the conducted premature beats show aberrancy due to slow interventricular conduction during the relative refractory period. In addition, the QRS complex in these premature beats is dissimilar to the QRS complex of the first beat of the fibrillation. These findings suggest that the mechanism of the fibrillation may be due to reentry, possibly involving dissociation between the bundle branches, rather than to repetitive discharge.

It should be noted that the R-R intervals prior to the first beat of the fibrillation are consistently longer. This reason is unclear, but it may be attributable to the delayed rate of rise of action potential, or reentry with slow velocity of the first beat of the fibrillation.

The configuration of the T wave, which had been positive (fig. 2A), changed distinctly to a biphasic five hours later (fig. 2B). However, the negative portion of the apparent T wave may consist mainly of U wave. At any event, the Q-T interval is greatly prolonged and the entire premature QRS complex is inscribed during the T or T+U wave, showing "R on T" phenomenon.

Figure 3 shows two types of beats preceded by P wave and conducted to the ventricles; one type followed by ventricular fibrillation, and the other not followed by ventricular fibrillation. The Q-Q' interval, i.e., the interval between the onset of the dissociated beats preceding ventricular capture and the conducted beats, is 0.43 ± 0.014 sec in the ventricular fibrillation group (N = 96) and 0.45 ± 0.028 sec in the nonventricular fibrillation group (N' = 111). The difference between these two groups is statistically significant (P < 0.001). If the termination of the T wave is assumed to be at the
nadir of the biphasic T + U wave, the Q-Q'/Q-T (or Q-Q'/Q-TU) ratio is 67.9 ± 3.06 percent in the former, whereas it is 70.5 ± 3.50 percent in the latter. The difference is also statistically significant. These findings show that the tendency to precipitate ventricular fibrillation is greater when the ventricular excitation interrupts the T wave earlier.

Experimentally, ventricular fibrillation could be induced by atrial premature beats in infant goats and pigs, since the refractory period of the A-V transmission system is shorter than that of the ventricle in these animals. Moreover, it was shown that atrial premature beats could induce ventricular fibrillation, if properly timed in the setting of lowered ventricular fibrillatory threshold. Our case seems to be the first clinical report of this kind because all previously published reports of "R on T" phenomenon have been examples of ventricular activity induced by artificial pacemakers, or by spontaneous ventricular premature beats.

On the other hand, the phenomenon described has many of the characteristics of "torsades de pointe": a self limited bout of ventricular fibrillation occurring when a ventricular premature beat begins during a long Q-T interval. In this case, it is difficult to define the end of the T wave, because of its biphasic configuration and prolonged Q-T interval. The early appearance of the conducted beats

Hemodynamic polygraph. LA = left atrial pressure; PA = pulmonary arterial pressure; PTG = digital plethysmogram.

Figure 4

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precipitating ventricular fibrillation can be interpreted as an evidence of occurrence during the vulnerable period of the ventricle. The range of the vulnerable period in this case was 0.4 sec to 0.46 sec following the onset of the ventricular excitation.

The mode of termination of ventricular fibrillation also deserves comment. There are two types: the termination by a QRS complex of the same configuration as the A-V junctional rhythm (the last of the fibrillation in the middle and lower strips in fig. 3) and the termination without this junctional type of QRS complex (in the upper strip in fig. 3). The former type would suggest that the A-V junction participates in the reentry process during the ventricular fibrillation, and that fibrillation stops because the impulse is blocked below the A-V junction.

Our patient showed very bizarre repolarization. Its etiology is unknown because the patient had no hypopotassemia, hypocalcemia, or congenital Q-T prolongation. It was transient and could be attributed to some unknown metabolic processes associated with alkalosis, hypotension, and hypoxia.

In conclusion, this case suggests that appropriately timed supraventricular beats may induce ventricular fibrillation in man even in the absence of preexcitation. This has significance both in regard to naturally occurring arrhythmias and to potential complications of fixed rate atrial pacing.

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Ventricular Fibrillation Induced by Conducted Sinus or Supraventricular Beat

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