

THE EFFECT OF QUINIDINE UPON SINUS TACHYCARDIA,
INCLUDING THE PRODUCTION OF TRANSIENT BUNDLE
BRANCH BLOCK

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THE observations to be described in this paper were made in connection with a study of auricular paroxysmal tachycardia. It is well known that quinidine frequently slows the cardiac rate in auricular paroxysmal tachycardia, and sometimes stops the paroxysm. The same effects may be produced by quinine, and the actions of these two drugs are, no doubt, similar. Singer and Winterberg¹ have shown that in sinus tachycardia quinine either does not alter the heart rate or increases it somewhat. It seemed desirable to investigate the effect of quinidine in sinus tachycardia.

A single dose of 0.6 Gm. of quinidine sulfate was given by mouth to each of five patients with sinus tachycardia. Four of the patients had exophthalmic goiter, and the fifth had chronic ulcerative colitis with fever and anemia. In all five cases the heart was organically sound, and none of the patients was receiving any drug which might affect the heart. The heart rate was counted at intervals before, and for at least two hours after, giving the quinidine, and electrocardiograms were likewise taken before giving the quinidine and two hours later.

The effect of quinidine upon the heart rate is shown in Table I. The rate was not affected or was slightly increased. These observations are similar to those of Singer and Winterberg,¹ who used quinine. They show that quinidine does not slow the rate of impulse formation in the sinoauricular node.

The electrocardiograms showed, in some instances, slight changes in the T waves after the administration of quinidine. With one exception, however, there were no significant alterations in the P-R or QRS intervals.

The one exception was Case 5, in which there was transient right bundle branch block two hours after the administration of the quinidine. Electrocardiograms taken a few minutes before the quinidine was given, and again on the following day, were essentially normal (Fig. 1). Multiple precordial leads showed relatively late activation of the right ventricle at the time when the bundle branch block was present. Two weeks later, 0.6 Gm. of quinidine sulfate was again given by mouth,

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TABLE I
THE EFFECT OF QUINIDINE BY MOUTH UPON THE HEART RATE IN SINUS TACHYCARDIA

CASE 1			CASE 2			CASE 3			CASE 4			CASE 5		
TIME	RATE		TIME	RATE		TIME	RATE		TIME	RATE		TIME	RATE	
12/15/41			2/ 7/41			3/ 6/41			11/12/41			2/ 7/41		
11:00 a.m.	156		1:00 p.m.	108		4:00 p.m.	120		1:45 p.m.	150		1:00 p.m.	90	
11:15	147		1:15	110	Quinidine	4:01	Quinidine		2:20	142		1:15	110	
2:30 p.m.	Quinidine		1:35	124	0.2 Gm.		0.2 Gm.		2:23	136		1:35	108	
	0.2 Gm.		1:45	112	(test dose)		(test dose)		2:25	Quinidine		1:45	110	Quinidine
	(test dose)		2:15	115		5:00	108			0.6 Gm.		1:55	107	0.6 Gm.
12/16/41			2:20	Quinidine		3/ 7/41			3:00	140		2:45	130	
9:20 a.m.	143			0.6 Gm.		8:55 a.m.	108		3:25	143		2:00	Quinidine	
9:25	150			110		9:45	100		3:55	150		3:45	130	
9:30	Quinidine			104		9:55	107		4:25	141		3:50	136	
	0.6 Gm.			108		10:00	Quinidine		11/13/41			2:40	106	
10:45	150			108			0.6 Gm.		11:15 a.m.	120		3:00	106	
11:30	144			115		11:00	128					3:30	113	
				124		12:00	107					4:00	118	
						1:00 p.m.	136					4:05	109	
												5:15	100	
												2/ 8/41		
												12:05 p.m.	88	

and two hours later the electrocardiogram again showed transient right bundle branch block (Fig. 2). Multiple precordial leads again showed relatively late activation of the right ventricle at the time when the bundle branch block was present. This observation is of interest in connection with the interpretation of the aberrant ventricular deflections which are sometimes observed after the administration of quinidine to patients with auricular fibrillation.

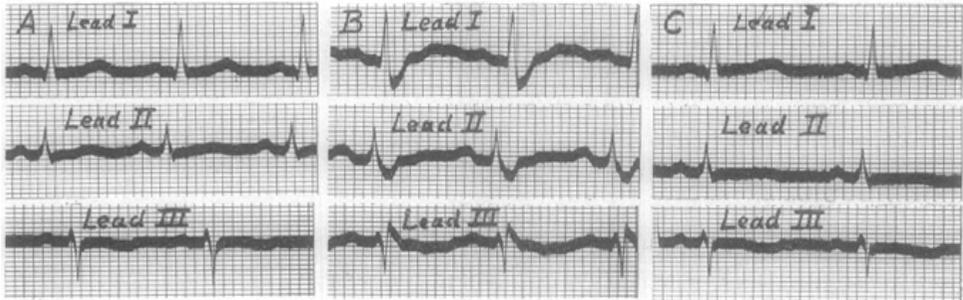


Fig. 1.—Quinidine sulfate, 0.6 Gm., by mouth at 2:00 P.M. A, Control curve, 1:55 P.M. B, Right bundle branch block, 4:05 P.M. C, The following day, 12:05 P.M.

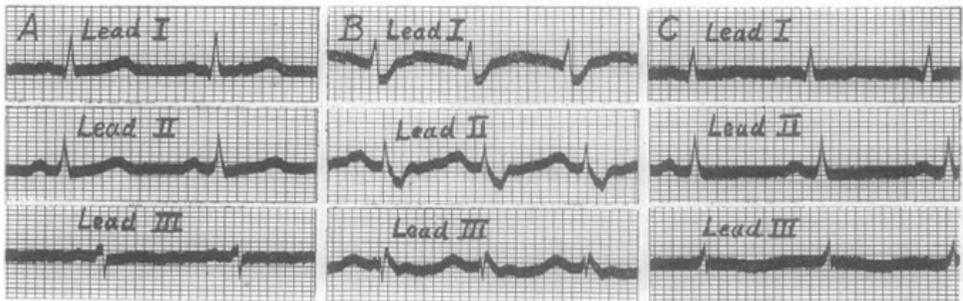


Fig. 2.—Two weeks after Fig. 1. Quinidine sulfate, 0.6 Gm., by mouth at 1:45 P.M. A, Control curve, 1:35 P.M. B, Right bundle branch block, 3:50 P.M. C, The following day, 10:45 A.M.

When quinidine is administered in the treatment of auricular fibrillation, and its effects followed closely by means of frequent electrocardiograms, approximately one-third of the patients show abnormal QRS deflections.^{2, 3} These resemble the abnormal complexes of ectopic ventricular beats or of defective intraventricular conduction. They occur at the height of the quinidine effect, and disappear shortly after the quinidine has been stopped.

It has been abundantly demonstrated that quinidine depresses intraventricular conduction, as shown by prolongation of the QRS interval,³⁻⁹ and the abnormal QRS complexes which follow quinidine have been attributed by some observers to defective intraventricular conduction.^{3, 5, 7, 9} Furthermore, it is well known that quinidine may abolish ectopic ventricular beats, and that it is especially effective in stopping

ventricular paroxysmal tachycardia.^{10, 11, 12} Nevertheless, the abnormal QRS deflections which follow quinidine have been attributed by some observers to ectopic ventricular beats.^{2, 6, 10, 13}

In auricular fibrillation, quinidine almost invariably causes an increase in ventricular rate. This makes it specially difficult to distinguish between abnormal deflections caused by ectopic premature ventricular beats, occurring singly or in runs, and those caused by defective intraventricular conduction, which would be especially likely to appear at higher rates or after the shorter diastoles. Lewis and his associates² have pointed out that there should be a close relation between prematurity and abnormality of ventricular deflections, if the abnormality were the result of impaired conductivity. They recognized that, in a general way, such a relationship existed, but did not consider it sufficiently close to indicate impaired intraventricular conductivity. In published electrocardiograms showing abnormal ventricular deflections which are attributed to ectopic ventricular beats caused by quinidine, alternative interpretations are possible, namely, that the ectopic beats occurred spontaneously or for some other reason, and were not caused by quinidine, or that the deflections were aberrant because of impaired intraventricular conductivity.^{6, 14}

White, Marvin, and Burwell⁵ mentioned a case in which quinidine produced marked intraventricular block without abolishing the auricular fibrillation. Korns⁷ published curves from a patient who showed bundle branch block after 6 grams of quinidine, which disappeared shortly after withdrawal of the drug; auricular fibrillation was present and the ventricular rate was 90 per minute, both in the presence of the bundle branch block and in its absence. Wilson, Wishart, Clark, and Herrmann³ described a case of auricular fibrillation in which all of the ventricular deflections became abnormal after 1.6 Gm. of quinidine. Normal rhythm returned two hours later, and all of the ventricular deflections were still abnormal; the normal type returned, however, as soon as the quinidine effect passed off. The abnormal deflections were attributed to impaired intraventricular conductivity.

In the present case, the aberrant ventricular deflections were clearly the result of impaired intraventricular conductivity caused by quinidine. It is our opinion that when quinidine causes abnormal QRS deflections, it does so by depressing conduction within the ventricles.

SUMMARY

A single dose of 0.6 Gm. of quinidine sulfate was given by mouth to each of five patients with sinus tachycardia. The cardiac rate was either not affected or was slightly increased.

In one patient, transient right bundle branch block occurred two hours after the administration of quinidine, upon two separate occasions.

When quinidine causes abnormal QRS deflections, it does so by depressing conduction within the ventricles.

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