**Paroxysmal Ventricular Tachycardia: A Study of One Hundred and Seven Cases**

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A review is presented of 107 cases of paroxysmal ventricular tachycardia. In the majority it was associated with acute coronary thrombosis or coronary sclerosis, in some with rheumatic valvular disease, and in others with no organic heart disease. It is generally critical and demands careful attention. Occasional cases are overlooked because only one heart sound is present for each cardiac cycle and the heart rate is misjudged to be one-half the actual rate. The outlook often is serious if the condition is not effectively treated. Therapy generally is successful in controlling the arrhythmia. The most valuable drug is quinidine. Other medications are magnesium sulfate, atropine, potassium salts, and possibly morphine.

Paroxysmal ventricular tachycardia is one of the less common types of paroxysmal rapid heart action. In spite of its rarity, prompt diagnosis and treatment are most important because often the patient’s life is at stake and with proper therapy the results may be excellent. The arrhythmia occurs predominantly in patients with serious organic heart disease and this, in addition to the effect of the rapid ventricular rate, makes it imperative that the attack be terminated as soon as possible if a fatal outcome is to be averted.

Lewis reported the first case of ventricular tachycardia. His patient was a seaman who had repeated premature ventricular contractions occurring singly and in runs of as many as 11 beats. Shortly after Lewis’ publication in 1909, reports of isolated instances of this arrhythmia appeared which were followed by reports of larger series and more complete reviews. Lewis produced ventricular tachycardia in dogs by ligating the coronary arteries and Robinson and Herrmann called attention to the relationship of paroxysmal ventricular tachycardia to coronary occlusion in man. Scott, in 1921, was successful in controlling this arrhythmia by the use of quinidine administered orally. There was 1:1 retrograde conduction to the auricles and no evidence of organic heart disease. The present study of 107 cases is reported with the hope of clarifying some of the problems of diagnosis, prognosis, and treatment encountered in patients with this disorder.

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**Material**

All cases of paroxysmal ventricular tachycardia occurring in the Peter Bent Brigham Hospital and the private practice of one of us (S. A. L.) from 1915 to 1948, inclusive, have been studied. Sixteen of these cases have been reported previously.

**Table 1.—Types of Underlying Heart Disease**

<table>
<thead>
<tr>
<th>Type of Disease</th>
<th>No. of Cases</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Coronary Artery Disease</td>
<td>79</td>
</tr>
<tr>
<td>2. Rheumatic Heart Disease</td>
<td>9</td>
</tr>
<tr>
<td>3. No Heart Disease</td>
<td>13</td>
</tr>
<tr>
<td>4. Miscellaneous Group</td>
<td></td>
</tr>
<tr>
<td>(A) Wolff-Parkinson-White Syndrome</td>
<td>5</td>
</tr>
<tr>
<td>(B) Congenital Heart Disease</td>
<td>1</td>
</tr>
</tbody>
</table>

The total number of episodes of ventricular tachycardia observed in this study of 107 subjects was 131. The youngest patient was 13 years of age and the oldest 83 years. The age distribution by decades is shown in figure 1. The average age was 54.8 years. Seventy-one patients were males and 36 were females. The types of underlying heart disease are shown in table 1, and it will be noted that
coronary artery disease was present in 74 per cent of the patients. The duration of the attacks varied from a few seconds to twenty-three days. Eighty-two patients had persistent attacks (hours to days) and 25 had intermittent attacks (seconds to a few minutes). The ventricular rate during the episodes varied from 135 to 292 with one exception; in one patient the rate was 115.

**Clinical Diagnosis**

The bedside findings which suggest the clinical diagnosis of paroxysmal ventricular tachycardia have been described by Levine and Strong and Levine. These consist of a changing intensity of the first heart sound at the apex, a slight irregularity of the ventricular cycle length, and the failure of vagal stimulation to produce any effect on the tachycardia.

A changing intensity of the first sound was noted in forty-three episodes. This sign will be detected only on careful auscultation and is not present in patients who have concomitant auricular fibrillation or in those in whom there is 1:1 retrograde conduction. In the rare instance in which 2:1 retrograde conduction to the auricles takes place one might expect to find alternation in the intensity of the first sound. The mechanism of production of the changing first sound is thought to be dependent on the changing relationship between auricular and ventricular contraction, similar to that which occurs in conjunction with complete heart block. Excluding the patients with auricular fibrillation, a changing intensity of the first sound was noted in approximately 50 per cent of the patients in this series.

In the common type of paroxysmal auricular tachycardia the cycle length is perfectly constant, whereas in ventricular tachycardia slight but detectable irregularities in the cycle length may be observed in an appreciable number of instances. The slight irregularities in rhythm in ventricular tachycardia can easily be overlooked unless auscultation is carried out carefully. Cooke and White on the other hand found thirteen of fifteen tracings of definite and prolonged paroxysms of ventricular tachycardia to be perfectly regular. Cooke and White also found that irregular rhythm was most likely to occur during short paroxysms and in patients with auricular fibrillation. Williams and Ellis found only 4 of 24 patients with the persistent type of tachycardia to have an obvious irregularity of the rhythm. In the 107 episodes of the persistent type of tachycardia in this series, in 32 per cent the rhythm was slightly irregular, while in 76 per cent of the intermittent type a similar type of slight irregularity was manifest. In considering the slight irregularity that is seen in paroxysmal ventricular tachycardia it must be clear that the actual heart rate can be quite constant over long periods of time. It is the length of contiguous cycles that may change. The difference may not be more than 0.03 second though occasionally it is much greater and may not occur but once in many cycles. The ear is able to appreciate these slight variations in cycle length. In contrast, the contiguous cycles in the common type of paroxysmal auricular tachycardia rarely vary more than 0.01 second in length.

Paroxysmal ventricular tachycardia does not respond to vagal stimulation. This finding is helpful in the differentiation of ventricular from paroxysmal auricular tachycardia, auricular flutter, and sinus tachycardia.

Gallarvardin described changes in the jugular pulse wave in patients with ventricular tachycardia. Further observations of the jugular pulse during the arrhythmia have been reported by Prinzmetal and Kelley. In ventricular tachycardia the auricular rate is generally slower than the ventricular rate and the pulsations of the auricles at this slower rate may be visible in the jugular vein. In addition, occasional prominent jugular waves will appear during cycles when the auricles are contracting while the ventricles are in systole. Such findings may be particularly helpful in those patients in whom P waves are not visible in the electrocardiogram. The abnormal jugular pulsations will be absent in subjects in whom there is no change in the intensity of the first heart sound, that is, in those with auricular fibrillation and 1:1 retrograde conduction.

There is one additional unusual auscultatory phenomenon that merits discussion, particularly since it has not been hitherto described. There were three instances in this series in which only one heart sound could be heard.
with each cycle. In 2 patients this resulted in the heart rate being misinterpreted by some observers as being one-half the actual rate. In one of these patients, the sounds alternated in intensity so that they closely resembled the normal sounds. It was difficult at the bedside to tell whether the rate was 115 with two sounds to a beat or 230 with only one sound. This could not be determined from the radial pulse since the patient was pulseless and the blood pressure was not obtainable. The electrocardiogram quickly revealed the fact that the ventricles were beating at a rate of 230. It is of interest that in this case two physicians who had been seeing the patient for some hours had overlooked the tachycardia and thought the heart rate was "about one hundred." Such experiences must be more frequent than has been realized as we have seen other instances of paroxysmal rapid heart action in which only one heart sound could be heard with each cardiac cycle.

In summary, one may state that there are bedside methods available which will enable the physician to suspect the diagnosis of paroxysmal ventricular tachycardia in most cases and even to make a fairly definite diagnosis in many instances without the aid of the electrocardiograph.

**Electrocardiographic Diagnosis**

The criteria for the electrocardiographic diagnosis of ventricular tachycardia were first proposed by Robinson and Herrmann.\(^9\) They emphasized three features. The first is the detection of auricular complexes (P waves) occurring independently and at a slower rate than the ventricular rate. The second is the presence of ventricular complexes that are abnormal in form and different from those observed in the same patient when the paroxysm is not present. The third is the finding of isolated ectopic ventricular complexes before or after a paroxysm that have the same form as the ventricular complex during the paroxysm.

The presence of P waves may be difficult to detect in the standard limb leads and therefore special auricular leads may be of value in their demonstration.\(^8\) If the condition of the patient is not too serious, esophageal leads taken in the auricular region may be helpful.

The abnormal ventricular complexes in themselves are not sufficient to make the diagnosis, as there may be aberration of the ventricular complexes due to the rapid rate alone.\(^10\) Also auricular tachycardia, auricular flutter, and nodal tachycardia with aberrant ventricular conduction or with permanent bundle branch block may simulate ventricular tachycardia.\(^22\) The concomitant presence of auricular fibrillation and paroxysmal ventricular tachycardia which occurred in 20 of the cases adds further difficulties in diagnosis.

Cooke and White\(^7\) noted that ectopic ventricular beats exactly similar in type to those of the paroxysm may be very difficult to find and that complexes almost alike are adequate to aid in the diagnosis. It is of interest that these authors have reported other cases in which auricular premature beats preceded and followed the paroxysms of ventricular tachycardia. They have also observed that the occurrence of paroxysms of abnormal ventricular complexes, regular and more rapid than the previous rate during auricular fibrillation, is diagnostic of ventricular tachycardia.

**Patients with Coronary Artery Disease**

In our series, in 79 cases (74 per cent) coronary artery disease was the cause of the underlying heart disease. The diagnosis of coronary artery disease was restricted to those patients who had angina pectoris or myocardial infarction, either acute or old. A number of the patients in this group had, in addition, hypertensive cardiovascular disease, but no distinction has been made here between the hypertensive and nonhypertensive. In 44 patients the attacks occurred following an acute myocardial infarction. The youngest patient was 31 years of age and the oldest 83 years. There were 55 men and 24 women. The attacks of tachycardia were of two types, persistent or intermittent. The latter were short paroxysms of tachycardia lasting seconds or minutes which ceased spontaneously or were controlled readily in most cases by therapy. There were 18 patients of the group with coronary artery disease who had intermittent paroxysms. The persistent type of
attack, of which there were seventy-six instances presents the more difficult therapeutic problem. The episodes, as can be seen in figure 2, tend to occur most frequently with the onset of acute myocardial infarction or during the first two weeks after the acute infarction. The attacks of tachycardia were regarded as occurring with the onset of an acute myocardial infarction when they were noted during the first six hours after the first evidence of coronary pain. It is not possible to determine how many subjects if any, developed the rapid heart rate directly with or a few seconds after the first evidence of heart pain. If the attack of tachycardia occurs at the same time as the acute infarction there may be some difficulty in either confirming or excluding the diagnosis of the underlying infarct. It is quite well known that paroxysmal rapid heart action of any type can result in chest pain, fever, leukocytosis, fall in blood pressure, and even subsequent electrocardiographic changes without any myocardial infarction.\textsuperscript{23,24} An added difficulty is that the shock state resulting from the tachycardia may be followed by a myocardial infarction, such as occurs in association with shock following hemorrhage or surgical operation.

Patients may have single or multiple attacks of ventricular tachycardia. The control of multiple attacks depends on the use of varying doses of quinidine prophylactically. There were 10 patients with coronary artery disease who had multiple attacks of ventricular tachycardia while under observation and 17 additional patients who gave a history of multiple episodes. The attacks in 3 of these patients were particularly interesting.

One patient had his first attack of paroxysmal ventricular tachycardia on the twenty-sixth day after an acute myocardial infarction. In the ensuing sixteen days he had twelve recurrent episodes. Following each attack reversion to normal rhythm occurred after a single dose of quinidine sulfate, either 0.6 Gm. given intravenously or 1.0 Gm. orally. This patient then had an uneventful recovery and is alive and working at the present time, two and one-half years following the last episode. The second patient, a 55 year old Negro, had an acute myocardial infarction complicated by ventricular tachycardia in 1939. From 1939 to March 1949 he had 111 episodes of paroxysmal rapid heart action. In five observed attacks the arrhythmia was identified as ventricular tachycardia and each episode was arrested by quinidine therapy. It is reasonable, therefore, to assume that all the other attacks were of similar origin. These attacks have recurred in spite of the prophylactic use of quinidine and potassium salts but the patient has continued his work as a janitor.

The third patient, a 58 year old white man, had an acute myocardial infarction in 1943 complicated by ventricular tachycardia. From December 1943 to the present time he has been treated in the hospital for eight episodes of ventricular tachycardia and has controlled many attacks at home by taking quinidine orally. He is working at the present time.

These 3 cases are exceptional but they bring out the fact that a patient with serious underlying organic heart disease may have multiple attacks of ventricular tachycardia and still carry on relatively normal activity for many years. They also indicate that repeated attacks of ventricular tachycardia may occur in a patient who once had a myocardial infarction, purely as a neurogenic upset without any additional injury to the coronary arteries or the heart muscle. In that sense the attack then behaves as paroxysmal auricular tachycardia does in an otherwise healthy individual.

The duration of the attacks varied from a few seconds to twenty-four days. There were seventeen instances in which the attack lasted one to five hours; eleven, six to twelve hours; nine, twelve to twenty-four hours; eighteen, one
to five days; and fourteen, five to twenty-four days. The longest case on record persisted for seventy-one days while the patient was under observation in the hospital and a probable six additional days prior to admission to the hospital.29 The patient expired with uncontrolled ventricular tachycardia. The duration of the attack is largely dependent on the promptness with which the diagnosis is made and effective therapy instituted.

The presence of auricular fibrillation during ventricular tachycardia is rather difficult to determine with accuracy. It was assumed to be present if found directly after the ventricular tachycardia disappeared. Accepting this assumption, auricular fibrillation was present in 14 patients of the coronary group. When the ventricular tachycardia was effectively treated with quinidine, the concomitant auricular fibrillation did not generally revert to normal sinus rhythm and even resisted reversion on large doses of quinidine.

The production of ventricular tachycardia by the use of quinidine has been previously reported4, 26, 27 and was observed in 2 patients in this group. Both had auricular fibrillation and had received large doses of digitalis in an unsuccessful effort to slow the ventricular rate. Quinidine was administered and both patients promptly developed paroxysms of ventricular tachycardia which ceased when the quinidine was discontinued.

Patients with Rheumatic Heart Disease

There were 9 patients (8.4 per cent) in our series who had rheumatic valvular disease. This was present in 15 per cent of the subjects of one reported series28 and 3 per cent of another.8 The age of our parents with rheumatic valvular disease ranged from 21 to 62 years; 8 were over 40 years of age. Seven had persistent attacks of tachycardia and 2 intermittent attacks. All the patients with rheumatic valvular disease were in severe congestive failure before the attacks of ventricular tachycardia occurred. One patient was in extremis. Five had coexistent auricular fibrillation. Digitalis had been given to 8 prior to the onset of the tachycardia. In 4 of these 8 the doses were in all likelihood too small to have produced any toxic effects but in the other 4 one might readily suspect that digitalis was in some way responsible for the tachycardia.

Subjects with Normal Hearts

Thirteen patients (12 per cent) had no evidence of organic heart disease. In other reported series, 17 per cent,3 14 per cent,6 and 10 per cent28 showed no evidence of organic heart disease. The youngest in the present series was 20 years of age and the oldest 53 years. Five had intermittent and 8 persistent attacks. The intermittent attacks were short paroxysms of tachycardia, with the patient usually complaining of palpitation and occasionally of dyspnea. Some of these episodes occurred with great frequency and were so distressing that medical advice was sought. These attacks were precipitated by exercise, emotion, and smoking. In the majority of instances the episodes were of no great significance.

In this group there were 4 patients with persistent tachycardia in whom the clinical features are worthy of comment.

A 20 year old medical student had had recurrent attacks of ventricular tachycardia beginning in his middle teens. All the attacks were related directly to exercise. During eight months of 1945 he had eleven episodes of tachycardia, all of several hours' duration. These either ceased spontaneously or reversion to normal rhythm was effected by the oral administration of quinidine.

A 23 year old dental student had recurrent attacks of palpitation and dyspnea with ventricular tachycardia beginning at the age of 13 years. This patient had from one to nine paroxysms a month, varying in duration from several minutes to thirteen hours. The longest period during which he was free from attacks was two years.

The third patient was a 34 year old woman, whose first attack of ventricular tachycardia lasted twenty days and was refractory to large oral doses of quinidine. Her condition was desperate. Reversion to normal rhythm occurred after a large oral dose of quinidine followed by 1 mg. of atropine. Following this the patient had two more short episodes while receiving prophylactic quinidine, 0.2 Gm. four times a day. She then had no more attacks until nine and ten years later, when she had two episodes. At the present time she is alive and fairly well.

The fourth patient, a 42 year old man, developed auricular fibrillation, paroxysmal ventricular tachycardia, and congestive failure. He had at least
three episodes of ventricular tachycardia during a six weeks' stay in the hospital. Following recovery, he has been working every day; he is symptomless and has no findings suggestive of heart disease at the present time, three years later.

It can be seen that patients without any evidence of organic heart disease may have episodes of ventricular tachycardia and in some cases present the same difficult therapeutic problems and dangers as those with underlying organic heart disease. However, after reversion to normal rhythm the prognosis is excellent.

**MISCELLANEOUS GROUP**

Five patients with the Wolff-Parkinson-White syndrome, with peculiar ventricular complexes, have been included in this study. On ordinary inspection the electrocardiographic pattern resembled that of paroxysmal ventricular tachycardia. Three of these cases have previously been reported. The interpretation of the exact mechanism can be disputed. It is well known that the customary disturbances in rhythm associated with the Wolff-Parkinson-White syndrome are auricular in type, usually paroxysmal auricular tachycardia or fibrillation. Therefore, it is quite likely that these 5 cases showing rapid broad ventricular complexes were instances of conduction through the anomalous bundle rather than cases of paroxysmal ventricular tachycardia. The only method of identifying a true ventricular tachycardia, in the presence of the Wolff-Parkinson-White syndrome, would be to detect auricular complexes coming regularly, independently, and more slowly than the ventricular beats. This has not as yet been demonstrated.

Despite the difficulty in interpretation, these five cases are included because they responded in general just as the more classic instances of ventricular tachycardia. None of these patients showed any other evidence of organic heart disease. As a group they were young patients, the ages being 13, 30, 36, 37, and 52 years. Two of these 5 died suddenly, the other 3 remaining quite well. One young boy presented a picture that was confused with and misdiagnosed as an acute myocardial infarction. In one case the tachycardia stopped spontaneously and in another reversion took place after a moderate amount of digitalis was given. However, the role played by digitalis in producing the reversion is doubtful. In the other 3, reversion resulted from quinidine administration. In one case there was a gradual but rapid slowing of the ventricular rate from around 300, before the normal rhythm was established. This is what one might expect from quinidine therapy if the abnormal mechanism was ventricular in origin, for if auricular fibrillation was present it is much more customary to observe an acceleration of the ventricular rate following quinidine, before regularization occurs, than a deceleration.

The important inference to be drawn from these experiences is that although the Wolff-Parkinson-White syndrome with paroxysmal arrhythmias may be unassociated with any other detectable organic heart disease, sudden death is not a very rare eventuality.

The last patient in this group was a young man, 30 years of age, who had Eisenmenger's complex. The diagnosis was confirmed by intracardiac catheterization studies. His primary complaints were palpitation and attacks of syncope. When the spells of unconsciousness became frequent, he had to discontinue his work which otherwise he would have been able to perform. It was found that his difficulty was due to ventricular extrasystoles, which occasionally developed into short runs of paroxysmal ventricular tachycardia and were followed by syncopal attacks. He seemed to be helped by a bilateral upper dorsal sympathectomy and quinidine therapy.

**PART PLAYED BY DIGITALIS**

Forty-seven patients received digitalis in varying amounts prior to the onset of ventricular tachycardia. Twenty-six were on what was considered maintenance doses or less at the time the tachycardia developed. In these patients it was doubtful if digitalis played any part in the precipitation of the episodes. Thirteen received what was considered as probably excessive doses. In these digitalis may have been important in the production of the paroxysms. In 6 there was definite evidence of digitalis intoxication, such as nausea, vomiting, premature ventricular contractions, and bigem-
inal and trigeminal rhythm. In these cases digitalis may have precipitated the arrhythmia but conclusive evidence of this is not available. Two patients with definite digitalis intoxication had bidirectional ventricular tachycardia and one with probable intoxication had alternation of the ventricular complexes.

Digitalis has long been considered as one of the more common precipitating factors in the development of ventricular tachycardia. Lundy and McLellan in their review in 1934 stated that digitalis was responsible for 100 per cent of the cases in which alternating bidirectional complexes were observed. Marvin reported 5 cases of ventricular tachycardia with alternation of the complexes and postulated that factors other than digitalis alone must play a part as 3 patients had no headache, nausea, or vomiting and only one had yellow vision. Scherf and Kisch in a study of 18 patients showing bidirectional ventricular tachycardia found that all but 4 were receiving digitalis at the time the tachycardia developed. They also noted that there was no definite relationship between the amount of digitalis and the development of ventricular tachycardia. They believed that there must be in addition to the digitalis factor an injured myocardium.

Digitalis cannot be looked upon as an important or frequent cause of ventricular tachycardia when it is appreciated that in this very clinic from which the cases are being analyzed, thousands of different individuals have received full doses of digitalis and yet only a very few have ever showed this arrhythmia. In one patient with valvular heart disease who had had previous attacks of paroxysmal ventricular tachycardia an attack appeared for the first time directly after a full dose of digitalis had been given. There was little doubt about the relationship between cause and effect in this instance. In another patient it was clearly established that the ventricular rate during the ventricular tachycardia accelerated under digitalis therapy. It is therefore very difficult to evaluate, with any degree of exactness, what part digitalis had in evoking the attacks of tachycardia, though we are of the opinion that it does play a role in a small number of cases.

Clinical Features

The clinical features that characterize the episodes of ventricular tachycardia have been studied in seventy-seven attacks in which adequate data were available. The onset of paroxysmal ventricular tachycardia may be entirely unnoticed by the patients. In such cases the diagnosis is made only when the examining physician or nurse notices that the patient has a rapid heart rate, and the physician investigates the rapid rhythm more thoroughly. There are, however, certain clinical features that make one suspect that the patient is having a bout of paroxysmal rapid heart action. The sudden onset of palpitation and slight to moderate dyspnea were the most common subjective symptoms noticed by the patients in this study. Dizziness and syncopal attacks with loss of consciousness may usher in the episodes. These symptoms may occur regardless of whether or not organic heart disease is present. Cerebral manifestations such as these were present in 13 patients and have been described previously as occurring in association with paroxysmal tachycardia. There is another type of syncope that may occur, for some patients with complete heart block may have a short paroxysm of ventricular tachycardia or fibrillation which is then followed by ventricular arrest of sufficient length to cause unconsciousness and convulsions. Such a mechanism producing Adams-Stokes episodes in persons with complete heart block is well known. There were 5 patients in this series who exhibited these findings. One patient, a 46 year old woman, had had complete heart block with Adam-Stokes attacks for a period of three years, the latter always being preceded by paroxysms of ventricular tachycardia or ventricular fibrillation. The patient failed to respond to any type of therapy and finally expired suddenly during an episode. At postmortem examination the heart weighed 360 grams, there was no evidence of coronary atherosclerosis, the interventricular septum showed no abnormalities, nor were there
any other structural abnormalities of the heart. Therefore, judged by postmortem examination, this patient had a normal heart.

Eighteen patients noticed substernal and precordial pain with the onset of the attack of tachycardia. The pain may be identical with that of angina pectoris or of an acute myocardial infarction. In patients who have had previous episodes of angina pectoris or a previous myocardial infarction the pain was usually not as severe as that due to the angina or infarction. The majority of patients described the pain as a dull ache, a heavy feeling, or a choking sensation.

In any patient, especially one in with an acute myocardial infarction, in whom sudden collapse occurs, ventricular tachycardia should be suspected. With the onset of the rapid rate there may be a marked fall in blood pressure; the skin becomes cold and clammy and profuse sweating occurs. The condition changes in a matter of minutes from satisfactory to critical. There were 44 patients in shock during the episode of ventricular tachycardia. The shock varied from mild, in which the systolic blood pressure was slightly less than 100 mm., to profound in which the blood pressure was unobtainable. In sixty-two instances congestive heart failure was present. The congestive failure was manifested by moist râles at the lung bases or throughout the chest and varying degrees of right-sided failure. These variations depended on the type of underlying heart disease and the duration of the rapid rate.

It may be said in summary that a patient with ventricular tachycardia may be asymptomatic during the attack or may have syncope, episodes, dizziness, or sudden collapse with the onset or during the paroxysm. Chest pain similar in all respects to that of angina pectoris may occur.

Prognosis

Strauss reported that in 50 patients with ventricular tachycardia with underlying organic heart disease, 40 were dead within three hours to six months of the onset, with an average duration of life of twenty-four days. Cooke and White noted that 17 of 21 patients with coronary artery disease expired in from a few hours to eighteen months. Follow-up information was available on 84 patients in this group of 107. In 42 patients in whom ventricular tachycardia complicated an acute myocardial infarction, 28, or 64 per cent, were dead within one month after the attack of tachycardia. Ten lived two years or longer; the longest period of survival was eleven years. Of 24 patients with coronary sclerosis and attacks of ventricular tachycardia, 10 were dead within six months after the first attack. The remainder lived for periods varying from one to ten years. The outlook, as can be seen, is particularly grave in those in whom the tachycardia complicates an acute myocardial infarction. Patients, however, may live for many years if there is recovery from the acute attack, and in addition these years may be useful and productive ones.

There was follow-up information available on 6 patients with rheumatic valvular disease and ventricular tachycardia. Three were dead within one month after the episode. Two died within eighteen months and one is alive at the end of five years.

The patients with no underlying heart disease have a very favorable prognosis as has been previously mentioned. There were 13 such patients in this group. In one patient, death was due to Adams-Stokes attacks which had recurred for three years; in another it was due to a neoplasm. Eight were alive and well for periods of from nine to twenty-one years after the attacks. In some of the patients the paroxysms ceased spontaneously. The prognosis in these patients was not affected by their having multiple attacks.

In general it may be said that although paroxysmal ventricular tachycardia is a serious complication of coronary artery disease, there are enough instances in which a satisfactory and useful recovery takes place to make one hopeful. It appears to be an especially ominous complication when occurring in association with rheumatic valvular heart disease but the outlook may be very favorable when the arrhythmia is unassociated with any structural disease of the heart.
TREATMENT

Several drugs have been used in the treatment of this arrhythmia. In 1921, Scott31 successfully used quinidine orally to control recurrent episodes of ventricular tachycardia. In 1927, Levine and Stevens32 tried intravenous quinidine therapy in an effort to effect reversion of ventricular tachycardia complicating an acute myocardial infarction. This attempt was unsuccessful with the small intravenous dose used but the arrhythmia was reverted to normal sinus rhythm with much larger oral doses of quinidine. In 1937, Hepburn and Rykert37 successfully treated 8 patients having paroxysmal ventricular tachycardia with quinidine administered intravenously. These authors advocated the use of quinidine intravenously only in patients who were moribund or in shock.

Zwillinger36 first reported the value of magnesium sulfate in patients with ventricular tachycardia. Boyd and Scherl38 treated 2 patients with this drug; the treatment was effective in one. In 1932, Sampson and Anderson38 used potassium salts in an effort to control auricular and ventricular ectopic beats and auricular and ventricular tachycardia. One patient with ventricular tachycardia was treated successfully but in another a paradoxical effect occurred and ventricular tachycardia was produced. Stempien and Katz39 used potassium chloride with success in a case that was refractory to all other forms of therapy. Salley40 described the use of atropine with quinidine after the ventricular rate has been slowed by the latter. Sabathie40 noted that morphine given intravenously was of value in 9 of 10 patients.

In some instances, both the persistent form (of hours' or days' duration) of the tachycardia and the intermittent type ceased spontaneously without any form of therapy. There were 6 patients with persistent tachycardia in whom reversion was effected without treatment and 12 with intermittent episodes who required no therapy. There were 4 patients who expired before any type of treatment could be given.

Fifty-seven episodes of ventricular tachycardia of the persistent type were treated with oral quinidine. Forty-six responded favorably (fig. 4). The dosage varied from 0.2 Gm. to 2.5 Gm. in a single dose and the total dosage at the time of reversion was from 0.2 Gm. to 45.8 grams. There were eleven failures. Of these eleven, in five episodes reversion to normal rhythm was brought about by intravenous quinidine therapy, in three the reversion was spontaneous, in one response was obtained following rectal administration of the drug, and in two reversion was not effected.

Table 2.—Reversion with Oral Quinidine in Persistent Paroxysmal Ventricular Tachycardia

<table>
<thead>
<tr>
<th>Maximum Single Successful Dose in Grams</th>
<th>No. of Episodes Reverting</th>
<th>Total Amount before Reversion in Grams</th>
</tr>
</thead>
<tbody>
<tr>
<td>0.2</td>
<td>6</td>
<td>0.2-1.6</td>
</tr>
<tr>
<td>0.3</td>
<td>6</td>
<td>0.3-0.9</td>
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<tr>
<td>0.4</td>
<td>4</td>
<td>0.4-1.0</td>
</tr>
<tr>
<td>0.6</td>
<td>8</td>
<td>0.6-2.4</td>
</tr>
<tr>
<td>0.7</td>
<td>4</td>
<td>2.2-2.5</td>
</tr>
<tr>
<td>0.8</td>
<td>9</td>
<td>0.8-6.5</td>
</tr>
<tr>
<td>1.0</td>
<td>3</td>
<td>2.1-4.3</td>
</tr>
<tr>
<td>1.2</td>
<td>2</td>
<td>1.8-3.7</td>
</tr>
<tr>
<td>1.4</td>
<td>1</td>
<td>5.6</td>
</tr>
<tr>
<td>1.5</td>
<td>2</td>
<td>7.2-8.0</td>
</tr>
<tr>
<td>2.5</td>
<td>1</td>
<td>45.8</td>
</tr>
</tbody>
</table>

and the patient expired subsequently. Quinidine was not a factor in the fatalities.

The time of reversion following the last oral dose of quinidine was accurately noted in thirty-four episodes, the shortest time being twenty-five minutes and the longest four hours. The usual time was two to four hours. The usual method of the oral administration of quinidine was to start the patient on 0.2 Gm. and increase the dosage 0.2 Gm. each four hours until reversion took place. The first dose also served as an indicator of any sensitivity to the drug. In several patients requiring intravenous quinidine therapy, the dose necessary to produce reversion was less than the largest previous oral dose which had been inadequate.

Ten patients with intermittent episodes of ventricular tachycardia were given quinidine orally. The amount used was 0.2 to 0.6 Gm. three times a day. Under these conditions, the episodes in 6 patients were completely controlled and in 3 the treatment was of doubtful value.
In thirty-one persistent episodes of ventricular tachycardia, quinidine was administered intravenously. The treatment was successful in 20 cases. The single intravenous doses varied from 0.2 to 1.5 grams. In six of the twenty episodes, reversion was not effected on the first intravenous dose but was accomplished on the second. The second intravenous dose varied greatly in amount. There were eleven failures with this method. In 2 of these 11 patients, reversion to normal sinus rhythm followed a larger single oral dose of quinidine, in one it followed the administration of magnesium sulfate, in one, failure followed three injections of quinidine in fifteen hours (total 3.4 Gm.) and reversion took place spontaneously the next day. In one patient the therapy was stopped after 0.3 Gm., in 2 patients death was the result of ventricular tachycardia, and in 4 death was due to quinidine toxicity. The first of the four fatalities due to quinidine occurred in a 55 year old woman, with mitral stenosis and mitral insufficiency, who was in a moribund state with pulmonary edema and shock. She died after 0.6 Gm. quinidine been given in 100 cc. of 5.0 per cent glucose solution in twenty minutes. The second patient was a man, 63 years of age, with hypertension, aortic stenosis, aortic insufficiency, and an acute myocardial infarction. He was in uremic coma and had congestive heart failure. He was given 0.3 Gm. quinidine in a few minutes, when respirations suddenly ceased. The third, a woman, 58 years old, with an acute myocardial infarction, in extreme shock, was given 0.6 Gm. quinidine in 200 cc. of solution in twenty-five minutes. The heart rate fell from 137 to 100 beats per minute. She then received 0.6 Gm. quinidine in one to two minutes and respirations ceased two minutes later. The fourth, a woman 51 years old, with mitral stenosis, mitral insufficiency, auricular fibrillation, and marked congestive failure, was in extremis with a blood pressure of 80/60. After 0.1 Gm. quinidine in 30 cc. of solution had been given slowly, she expired. It is clear from these 4 cases that all fatalities occurred in patients who were in extremis. In each instance it did not appear that they would have survived for many minutes or more than a few hours.

Thirteen patients were treated with magnesium sulfate given intravenously in doses varying from 2 to 5 grams. One patient received only 1 gram. Nine received two or more injections, usually within thirty minutes of each other. In 2 cases, reversion to normal sinus rhythm took place and in one case the ventricular tachycardia was converted to 2:1 flutter. The side effects noted were similar to those described by Boyd and Scherf, namely, a feeling of intense heat, flushing, and nausea. The value of magnesium sulfate is not great but the drug is relatively safe and therefore can be tried early. It proved effective rarely when quinidine failed.

In 7 patients, atropine sulfate was given either intravenously or intramuscularly in doses of 1 or 2 milligrams. The atropine was usually given after maximum slowing of the ventricular rate had been brought about by quinidine. The most dramatic effect occurred in the patient previously described by Salley, in whom the ventricular rate was slowed from 160 to 112 following 2.0 Gm. quinidine given by mouth; then 2 mg. of atropine were given intramuscularly and almost immediately complete heart block with a ventricular rate of 30 developed. There were 2 other patients in whom atropine may have been instrumental in causing reversion of the tachycardia to normal sinus rhythm. One patient had had ventricular tachycardia for twenty-one days and had failed to respond to magnesium sulfate, quinidine either orally or intravenously, and potassium salts. Finally 1 Gm. of quinidine was given intravenously and the rate fell from 180 to 110; 2 mg. of atropine were given and normal sinus rhythm occurred almost at once. The second patient had had ventricular tachycardia for twenty days and had not responded to any form of therapy. She was given 2.5 Gm. of quinidine by mouth in a single dose. After the heart rate slowed, 1 mg. of atropine was given and reversion took place in about two hours. The atropine may not have played any part in the reversion in this case. In the remaining cases atropine was of no value. Salley suggested that atropine breaks up the circus of ventricular tachycardia by paralyzing the vagus and that this effect lengthens the refractory period of the ven-
tricles. Wilburne and collaborators\textsuperscript{31} produced ventricular tachycardia in animals by the intravenous injection of epinephrine and found that the arrhythmia could be prevented by the use of atropine prior to the administration of epinephrine.

Potassium salts were used only in 2 patients and proved to be of no value. Morphine (30 mg.) was given intravenously to one patient and failed to cause reversion.

The drug of choice in the treatment of ventricular tachycardia, in our opinion, is quinidine. The route of administration of the quinidine depends on the clinical condition of the patient. If the condition is critical, the drug should be given by the intravenous route. This is a matter of individual judgement and difficult to express in very definitive terms. In general, if it appears that the patient is likely to succumb in minutes or hours the intravenous route is preferable, whereas if he is likely to survive for days the oral method might be employed. Quinidine hydrochloride, diluted (0.6 Gm. with 200 cc. of 5 per cent glucose in distilled water), may be given by slow drip over a period of about one hour.\textsuperscript{*} Electrocardiograms, made continuously or frequently during the injection, are helpful in that the procedure can be stopped instantly if reversion takes place or if any threatening disturbance in the cardiac mechanism occurs. If the first intravenous injection fails, a similar or larger amount may be given within several hours.

There are many difficulties in appraising and predicting the effect of oral quinidine dosage. One common error in general practice is to continue the same oral dosage day after day. This applies not only to the treatment of paroxysmal ventricular tachycardia but to auricular fibrillation and other arrhythmias. If a certain program, such as 0.2 to 0.3 Gm. three times a day, is not effective in a day or two it is not likely to be any more effective if continued indefinitely.\textsuperscript{42} The maximum effective blood level of the drug is reached about an hour or two after oral administration and there is practically none left in the blood stream twenty-four hours later.\textsuperscript{43, 44} On the other hand, the blood level need not be the determining factor in causing an effect on the heart or reversion to normal sinus rhythm. This is well illustrated by the experiences we have had in which auricular fibrillation has reverted to normal rhythm six hours or more after the last dose had been given. In such cases the blood level must have passed its peak when sudden regularization took place.

In general it is of little importance to estimate the total amount of the drug employed. The significant factor is the largest single dose that is required to effect reversion. Many patients have had 10 or 20 Gm. of quinidine given as a total dose over many days without any beneficial effect on their arrhythmia, while a total of 1 or 2 Gm. when given in increasing doses over the course of one or two days was effective. In administering quinidine either orally or intravenously it is helpful to observe a slowing of the ventricle which occurs quite frequently. This indicates a partial effect in the right direction and makes one think that further dosage might produce the desired regularization.

Another value of quinidine is that even if the drug does not cause reversion of the ventricular tachycardia, it may keep the ventricular rate at a slower rate and improve the general state of the patient.\textsuperscript{45} Such an effect has been noted in many of the patients in this series. Quinidine has also been reported as being valuable in controlling bidirectional ventricular tachycardia.\textsuperscript{46}

Much has been said and written about the toxic effects of quinidine. In the treatment of ventricular tachycardia these matters are of very little importance as the condition under consideration is already critical and often fatal if uncontrolled. Excessive quinidine may produce shock but these patients are not infrequently already in shock. In many cases marked distortion of the electrocardiograms resulted from the large doses of quinidine. Had such abnormalities been used as a criterion for cessation of therapy the attacks would not have been controlled. It is difficult to define the exact indication for discontinuing the drug. It is reasonable to regard sudden syncope or some other obviously desperate untoward complication such as convulsions.

\textsuperscript{*}Quinidine hydrochloride supplied through the courtesy of Brewer & Company, Worcester, Mass.
as sufficient reason to discontinue the treatment. The fact that the patient becomes nauseated, dizzy, weak, or develops diarrhea or ringing of the ears should not discourage the physician from persisting with this therapy, when the alternative is likely to be a fatal termination.

Summary

1. The following observations were made from a study of 107 patients with paroxysmal ventricular tachycardia, varying in age from 13 to 83 years, with the majority between 50 and 70 years.

2. The diagnosis can often be suspected by simple bedside examination but is better confirmed by electrocardiographic studies. On clinical examination an occasional case can be overlooked because only one heart sound may be present to each cardiac cycle so that the physician estimates the rate at one-half its actual value.

3. In 79 patients (74 per cent) the underlying cause of the heart disease was coronary artery disease, generally with recent or old myocardial infarction. Nine patients had rheumatic heart disease, 13 no heart disease, and 6 miscellaneous conditions.

4. There were 27 patients who had recurrences of prolonged attacks over the course of months or years.

5. The duration of the prolonged attacks varied from hours to twenty-four days.

6. It seemed that digitalis played a role in the precipitation of the paroxysmal ventricular tachycardia in only a small number of instances.

7. Although the attack of tachycardia may be symptomless, it is frequently accompanied by substernal pain, sudden collapse, shock, dyspnea, or syncope.

8. The immediate prognosis of the attack of tachycardia is very good, since in all but a few subjects normal rhythm was resumed following appropriate therapy. The ultimate prognosis is grave in those who have underlying coronary or valvular disease; however, a fair number of these were able to carry on a useful occupation for years.

9. In the group of subjects with no organic heart disease the prognosis is generally excellent. Even in this group the threat of sudden death is present; death during a paroxysm occurred in one patient who had a structurally normal heart post mortem.

10. In this study, magnesium sulfate administered intravenously was occasionally of value, potassium salts given orally were of no value in the few instances in which they were employed, and morphine given intravenously failed on the one occasion it was tried. The treatment of choice was quinidine therapy. The oral administration was successful in forty-six of fifty-seven episodes. The intravenous route, used in the more desperate cases, was successful in twenty out of thirty-one attacks.

11. The intelligent use of quinidine was found to be of great value in this condition and not infrequently life saving.

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