Paroxysmal Tachycardia
Experiences with Massive Doses of Quinidine Intravenously in a Refractory Case

By George O. Bell, M.D., Robert B. Bradley, M.D., and Lewis M. Hurxthal, M.D.

An interesting case of recurrent paroxysmal tachycardia is presented. The patient had had a previous coronary thrombosis, leaving in its wake a residual left bundle branch block. Three episodes of paroxysmal tachycardia were observed, the longest one of sixty-three days’ duration. Although ventricular origin of the tachycardia was thought to be most likely, a nodal origin was also considered. All forms of therapy were tried but only intravenous quinidine proved to be successful. Experiences with massive doses of quinidine intravenously are presented.

Paroxysmal tachycardia occurring in patients with organically sound hearts is always a burden and especially so if it is prolonged. When such an abnormally rapid heart rate occurs in patients who have previous myocardial damage the threat to life is indeed real. The conversion of the tachycardia to normal sinus rhythm must be accomplished as quickly as possible to avoid a fatal outcome. The case herein reported proved to be extremely refractory to all treatment and only after heroic measures were used was success attained.

Although the exact nature of the tachycardia in the present case remains uncertain, the therapeutic procedures used may be of some interest to others who are confronted with the problem of treating patients with stubborn cases of paroxysmal rapid heart action.

Case Report

R. T., a 44 year old white, married clerk, was first seen at the Lahey Clinic on October 22, 1946, complaining of rapid heart action of eighteen days’ duration. His past history revealed the occurrence of a coronary occlusion in December 1941. Electrocardiographic changes were characteristic of an anterior myocardial infarction. Recovery from the episode was prompt and he was able to return to his usual work three months later. Following the attack of 1941 until October 1946, six episodes of paroxysmal rapid heart action had occurred, each lasting about twelve hours and responding to simple measures such as administration of digitalis, morphine, or sodium pentobarbital and a night’s sleep. On October 4, 1946, he suffered a sudden attack which failed to respond to the usual measures.

On examination, on October 22, the heart was found to be beating rapidly (180) and regularly but with some slight unevenness of the apical sounds. Vagal stimulation by pressure on both carotid sinuses produced no slowing. Electrocardiograms showed a tachycardia with QRS interval of 0.12 second (fig. 1, A).

The administration of 20 mg. Mecholyl subcutaneously produced flushing and substernal distress, but no change in the heart rate.

The next day 0.6 Gm. quinidine sulfate given intravenously over a period of fifteen minutes slowed the apex rate from 180 to 140. The same dose was repeated and slowed the apex rate to 130. Four hours later 2 cc. (1.0 mg.) Prostigmine methyl sulfate administered intramuscularly produced no change in the abnormal rhythm.

During the next four days (October 24–27, 1946, inclusive) the patient remained in bed at home and was given quinidine sulfate orally, 0.4 Gm. every three hours. At times his apical rate slowed to 90 beats per minute.

First Hospital Admission. On October 28, 1946, the patient was hospitalized for further treatment. An electrocardiogram verified the presence of the abnormal rhythm with a ventricular rate of 160. He was given 1.5 mg. Prostigmine, followed in five minutes by 5 mg. Mecholyl administered subcutaneously. This combination of drugs produced vigorous vagal stimulation characterized by profuse sweating, rhinorrhea, lacrimation, nausea, vomiting, severe abdominal cramps, diarrhea and micturition, but no change whatever in the tachycardia. Atropine sulfate (1.2 mg. given intravenously) terminated this reaction.

The following day, October 29, 1946, 20 cc. of a 25 per cent solution of magnesium sulfate given intravenously had no effect on the tachycardia.

During the next three days treatment consisted of a daily ration of quinidine sulfate (1.0 Gm.)
and potassium chloride (10 Gm.) given by mouth. Because of marked discouragement on the part of the patient he was discharged on November 2 to admitted to the hospital with congestive heart failure. He was critically ill, with an apical heart rate of 172, cardiac enlargement, basal pulmonary

![Fig. 1.—A, October 22, 1946. Paroxysmal tachycardia. Rate 208. B, November 10, 1946. One day following conversion to sinus rhythm. Rate 110. P-R, 0.20 second. Delayed intraventricular conduction. QRS, 0.13 second. T, inverted.](image)

continue the same dosage of quinidine and potassium chloride at home.

Second Hospital Admission. On November 9, 1946, one week after discharge, the patient was re-

![Fig. 2.—December 1946. Left ventricular enlargement. Cardiothoracic ratio, 15.2 to 30.5 centimeters.](image)

Quinidine hydrochloride* (1.8 Gm. in 300 cc. of 5 per cent glucose solution) was administered intravenously over a period of one and one-half hours. The heart rate slowed from 172 to 120 beats per minute. Potassium acetate (0.5 Gm.) was then given every two hours, and after the third dose the heart rate dropped to 88. The electrocardiograms (fig. 1, B) showed a normal sinus rhythm with a rate of 110, P-R interval of 0.2 second, QRS complex 0.13 second in duration, and inverted T wave in Lead I. Total duration of this attack of tachycardia was thirty-seven days.

During the remainder of this hospital stay the patient was maintained on 0.8 Gm. quinidine sulfate and 4 Gm. potassium acetate per day. The congestive heart failure responded to low-salt diet and diuretics. Digitalis was avoided because of the possible danger of reinstigating the abnormal tachycardia. The patient was discharged on November 26, on a program of markedly restricted activity, 0.2 Gm. quinidine sulfate three times daily, and 0.033 Gm. phenobarbital three times daily.

The patient was seen at varying intervals during the next four months. In December 1946 a roentgenogram of the chest showed clear lung fields and left ventricular enlargement (fig. 2). He maintained a normal sinus rhythm with an average apex rate of 90 to 100. Because of easy dyspnea, digoxin

* Brewer and Company, Inc.
therapy was started and maintained for the next three months. The patient returned to work in January 1947 and remained fairly well, except for mild angina on effort.

Third Hospital Admission. On March 10, 1947, the patient was admitted to the hospital for the third time because of rapid heart action which had started suddenly the day before without obvious precipitating cause. The apex rate fell from 206 to 88 at which point the mechanism reverted to a normal sinus rhythm. Total duration of this paroxysm was two days. The patient was maintained on oral quinidine sulfate (0.2 Gm. four times a day) and potassium acetate (1.0 Gm. four times a day). Electrocardiograms made following conversion to normal rhythm showed a rate of 72 with a P-R interval of 0.28 second and QRS wave of 0.17 second’s duration (fig. 3, A). The patient’s general condition was good and there were no signs of cardiac decompensation.

Vagal stimulation by means of eyeball pressure, carotid sinus pressure, gagging, and the Valsalva maneuver were without effect. Heavy dosage of morphine given subcutaneously provided the patient with a good night’s sleep but was without influence on his tachycardia.

The next day he was given 180 mg. procaine (18 cc. of 1 per cent solution) intravenously within four minutes. Sodium pentothal was given concomitantly to control the toxic effects on the central nervous system. There was no change in cardiac rhythm although the rate fell from 200 to 156 per minute.

Several hours later the patient was given 0.2 Gm. quinidine orally, and potassium chloride (4 Gm. initially and 2 Gm. every two hours) and quinidine hydrochloride (1.8 Gm. in 200 cc. of 5 per cent glucose solution) intravenously over a period of one and one-half hours. The apex rate fell from 206 to 88

Fig. 3.—A, March 10, 1947. Paroxysmal tachycardia. Rate 204. B, March 11, 1947. Following intravenous quinidine therapy. Normal sinus rhythm. Rate 72, delayed A-V and I-V conduction, P-R, 0.28 second. QRS, 0.17 second.
tion in his work. Physical examination showed no abnormality except for slight cardiac enlargement and an apical rate of 160 per minute. Slight variation in intensity of heart sounds and irregularity in the rhythm were again noted. Signs of congestive failure were absent.

During the first four days quinidine was given orally in doses of from 2.4 to 4.8 Gm. per day along with 24 Gm. potassium acetate per day. Morphine sulfate was given at various times, usually 15 mg. subcutaneously and occasionally intravenously. The abnormal rhythm persisted although the apical rate was slowed from 160 to 110-120.

![Fig. 4.—September 7, 1948. Lead II only. (A), Paroxysmal tachycardia, (B), during intravenous quinidine therapy showing (C), conversion to sinus rhythm.](image)

On September 7, 1948, quinidine was given intravenously in a dose of 1.8 Gm. in 300 cc. of 5 per cent dextrose in saline solution. Potassium chloride was administered throughout the period of treatment in 2-Gm. oral doses every two hours. Normal sinus rhythm (fig. 4) was established after 1.2 Gm. quinidine had been administered. One hour after the cessation of intravenous quinidine therapy, however, tachycardia reappeared, despite continued oral administration of quinidine and potassium, given to the point of nausea.

The next day, September 8, the electrocardiogram showed a perfectly regular although abnormal rhythm (fig. 5, A) which suggested a supraventricular tachycardia occurring in the presence of a left bundle branch block. In view of this cardiogram and also since the quinidine therapy had failed, it was decided to give digitalis a trial. Cedilanid administered intravenously in a dosage of 1.6 mg. had no influence on the electrocardiogram nor on the clinical findings.

On September 9, intravenous administration of quinidine (1.8 Gm.) again failed to establish a normal rhythm.

After these two failures of intravenous quinidine therapy it was decided to administer digitoxin to its fullest effect. Digitoxin was given orally in a dose of 0.2 mg. every eight hours for the next two days, September 11 and 12. On the second day of this program the apical rate steadily increased from the average basal rate of 120 up to 180 per minute. With this manifestation of digitalis toxicity it was apparent that further pursuance of this program would not only be unsuccessful but would also be dangerous. Digitoxin therapy was therefore abandoned and the oral administration of quinidine was resumed in a dosage of 0.4 Gm. every four hours. The ventricular rate was maintained between 120 and 140 per minute on this program.

On September 14, prostigmine methyl sulfate (1.0 mg. given intramuscularly) and prolonged pres-
sure first on both eyeballs and then on both carotid sinuses had no effect.

On September 15, acetylcholine was administered intravenously rapidly (within one second) in doses of 35 mg., then 60 mg., and finally 100 mg. with no effect on the tachycardia.

On September 17, the intravenous quinidine program was repeated. Potassium chloride was given in dosage of 2 Gm. every hour for four doses. A total of 1.8 Gm. quinidine over a period of one hour and fifty-five minutes failed to convert the tachycardia to normal sinus rhythm. At the completion of the quinidine injection, atropine sulfate (1.2 mg. given intravenously) had no effect.

On September 18, 0.1 Gm. Papaverine given intravenously produced a marked generalized flush but no change in cardiac rate or rhythm. A roentgenogram of the chest showed the same findings as previously noted, but for the first time calcification was noted in the pericardium of the left ventricle (fig. 6).

On September 25, a fourth trial of intravenous quinidine was given as before. Three grams of potassium chloride were given in a single dose ninety minutes after beginning the quinidine. The patient received 1.8 Gm. quinidine in one hundred and sixty-five minutes. Normal sinus rhythm appeared one hundred and twenty-five minutes after the start of quinidine and thirty-five minutes after the dose of potassium chloride. Maintenance therapy consisted of 0.4 Gm. quinidine sulfate and 1.0 Gm. potassium chloride every four hours. Normal sinus rhythm remained for a period of twelve hours at which time the patient vomited, the ventricular rate increased to 115 beats per minute, and the abnormal rhythm reappeared.

On September 28, three days later, the fifth trial of intravenous quinidine was given in the same manner. This attempt was unsuccessful in spite of reduction of the ventricular rate to a low of 84 per minute.

On October 2, thirty days after the onset of his tachycardia, a bilateral procaine block of the inferior cervical ganglia plus the first four thoracic sympathetic ganglia was performed.* Good physiologic effect was obtained, as manifested by bilateral Horner's syndrome, increased warmth of the arms and hands, and absence of sweating on face and arms. Vagal stimulation was also done by means of eyeball and carotid sinus pressure. There was no change demonstrable either clinically or by electrocardiogram.

On October 6, four days later, the bilateral procaine block was repeated. This time the patient was given 0.6 Gm. quinidine orally every four hours for forty-eight hours prior to the block. Again, there was no effect on the tachycardia. Atropine sulfate (2.0 mg.) given intravenously did not change the abnormal rhythm. Four hours later Prostigmine (1.5 mg. given intramuscularly) was likewise unsuccessful.

On October 7, the intravenous quinidine regimen was applied for the sixth time. The electrocardiogram showed the appearance of normal sinus rhythm after 1.0 Gm. had been administered. Sinus rhythm alternated with the tachycardia repeatedly during the next one hundred and ten minutes, during which time an additional 0.8 Gm. quinidine was given.

![Fig. 6.—September 18, 1948. Calcification in pericardium over left ventricle.](http://circ.ahajournals.org/)

Forty-five minutes after the discontinuance of the quinidine injection the electrocardiogram showed that the abnormal tachycardia had persisted. That night the patient retained 2.0 Gm. potassium given orally every two hours, except for two doses, and 0.6 Gm. quinidine given every four hours. An electrocardiogram taken the next morning (fig. 7) suggested the possibility of potassium intoxication although the patient showed no clinical signs of this condition.

From October 8 to October 13 the patient was maintained on quinidine sulfate (0.2 Gm. every four hours). The ventricular rate averaged about 140 per minute or less. During this time, diarrhea from medication increased to eight or ten stools per day, and anorexia was moderately severe.

* Suggested by Dr. Samuel A. Levine.
On October 13 the patient received 20 cc. of a 25 per cent solution of magnesium sulfate intravenously in a period of two minutes. This produced severe flushing and sweating, but had no effect on his heart except for slight slowing of the ventricular rate (140 to 120 per minute).

On October 15 the same quinidine regimen as that used on October 7 was tried. Conversion to a sinus rhythm occurred after slightly less than 1.0 Gm. had been given. On this occasion, the abnormal rhythm reappeared between one and two hours after the treatment had been terminated.

On October 16 the patient developed pleuritic pain in the lower portion of the right side of his chest. A roentgenogram of the chest showed a high diaphragm on the right side, mottled density throughout the base of the right lung, and slight shift of the mediastinum to the right; these findings were interpreted as evidence of atelectasis and a superimposed pneumonitis. Fever, chest pain, and rales at the right base responded slowly to penicillin therapy. On October 25 the roentgenograms showed an increase in the density at the base of the right lung; this was interpreted as evidence of pneumonia, pleuritis, and fluid, although the possibility of a pulmonary infarct could not be entirely excluded.

On October 30 the patient showed evidence of congestive heart failure. Mercurial diuretics, xanthises, and acid-ash, salt-free (1.5 Gm. salt or less) diet were effective in relieving the symptoms to some extent. During this interim the tachycardia continued without change. The patient’s daily quinidine ration kept the rate at an average level of 120 to 130.

It became evident that the patient was showing increasing dependence on morphine. His daily intake of morphine had been limited to 15 mg. at bedtime with occasionally an extra dose during the day. Attempts at withdrawal were accompanied by mental depression, anxiety, apprehension, paranoid tendencies, and increase in diarrhea.

On November 3 the intravenous quinidine regimen was attempted for the eighth and final time. Since the previous successful conversions to normal rhythm had failed to establish the sinus rhythm for more than twelve hours, the plan adopted on this trial was to give quinidine until normal sinus rhythm appeared and then to maintain this rhythm by constant slow intravenous drip of quinidine over a period of several hours. In this way, we hoped to depress the irritability of the ectopic focus long enough to permit the sinus node to recapture its dominance.

Quinidine hydrochloride, 1.8 Gm. in 300 cc. of normal saline solution, was given intravenously. Sixty-five minutes after starting the quinidine and twenty minutes after the oral administration of 2.0 Gm. potassium chloride, the electrocardiogram showed a normal sinus rhythm with a ventricular rate of 106 per minute.

The initial 1.8 Gm. quinidine was administered in a period of two hours, after which an additional 1.8 Gm. was started. At this time the apex rate was 88 per minute. The flow of quinidine solution was reduced to a rate of 5 to 8 drops a minute.

Four hours later an electrocardiogram (fig. 8) showed normal sinus rhythm with a rate of 70. The patient at that time responded poorly to commands and his breathing became irregular. The flow of quinidine solution was stopped, a total dose of 3.2 Gm. having been administered. Five minutes later he was completely unresponsive, his breathing ceased, and his heart sounds could not be heard. An electrocardiogram (fig. 8) showed loss of P waves and marked widening of the QRS complex. Neo-nephrine (3 mg.) was given intravenously and the heart sounds became audible with an apical rate of 96. An additional 10 mg. Neo-nephrine and 0.5 Gm. caffeine were administered intravenously followed by a marked improvement in the heart sounds.

The patient was placed in an oxygen tent and within an hour he regained full consciousness, cyanosis decreased, normal sinus rhythm remained with an apex rate of 90 to 100, but the blood pressure was still unobtainable.

During the ensuing twenty-four hours the blood pressure gradually rose to 80/60. The sinus rhythm persisted. Anuria occurred and the patient complained of marked thirst. Edema appeared in the feet and ankles. The nonprotein nitrogen rose to 78 mg. per 100 cc. of blood, the serum carbon-dioxide combining power was 25 volumes per cent, and the serum chloride was 446 mg. per 100 cc. of serum.
The patient was given fluids freely by mouth, 350 cc. of human plasma intravenously, and additional caffeine and Neosynephrine intramuscularly. On November 4 a total of 55 cc. of urine was obtained by catheterization during the twenty-four hours. On November 5 urinary output returned to normal levels. The duration of the urinary suppression was forty-two hours.

From this point on the patient began to improve slowly and steadily. The serum nonprotein nitrogen, chloride, and carbon-dioxide combining power returned quickly to normal levels. Edema decreased and the blood pressure rose to around 100/60-70. The final electrocardiogram (fig. 9) showed a normal sinus rhythm. The addiction to morphine was the only problem of significance and it was gradually corrected after withdrawal of the drug. The patient was discharged from the hospital on November 16, seventy-five days after admission. The duration of his paroxysmal tachycardia was sixty-three days.

Fig. 8.—November 3, 1948. Lead II only. During intravenous quinidine therapy (3.2 Gm. total). A, Paroxysmal tachycardia, B, sinus rhythm, C, D, E, F, progressive changes showing quinidine toxicity. G, recovery from toxicity. H, eight and one-half hours later, normal sinus rhythm.

Fig. 9.—November 15, 1948. Normal sinus rhythm. Rate 92. P-R, 0.18 second. Delayed intraventricular conduction. QRS, 0.13 second. (The most recent electrocardiogram, January 10, 1949, showed normal sinus rhythm. Rate 110. Delayed A-V conduction. P-R, 0.22 second. Left bundle branch block. QRS 0.16 second in duration.)
Following his discharge from the hospital the patient was maintained on 1.0 Gm. quinidine sulfate daily and an occasional injection of Mercuhydrin.

Activity was gradually increased and well tolerated until he contracted an acute upper respiratory infection in January 1949. A severe paroxysmal cough developed and interfered with sleep. Dyspnea became progressively worse and because of this he was readmitted to the hospital where he remained for fifteen days. Because of hydrothorax on the right side, secondary to congestive heart failure, the pleural cavity was tapped twice and 1,850 cc. of straw-colored, transudative fluid were removed. While the patient was in the hospital acute and severe pleurisy involving the lower left side appeared and slowly cleared up. Although this was considered to be inflammatory, the possibility of a small pulmonary infarct could not be excluded.

The patient was discharged on February 3 and seen at the clinic on February 17, 1949. His heart had maintained its normal sinus rhythm, and there were no signs of congestive heart failure. Physical activity was being resumed very slowly and the patient was apparently getting along satisfactorily.

**DISCUSSION**

The diagnosis of this patient's tachycardia has been in doubt. We do know that the patient has a left bundle branch block and the tachycardia has been engrafted upon this. Retrograde conduction of the impulse from ventricles to auricles seems to be definite and inverted P waves are found to occur at varying intervals following the QRS complexes. In figure 5, A an inverted P wave follows each QRS complex. In figure 5, B an inverted P wave follows the QRS wave at progressively increasing intervals—the “reverse Wenckebach” with a 4:3 and 3:2 retrograde block. Regularly occurring P waves independent of the ventricular rhythm have not been identified. In the absence of this proof, ventricular tachycardia cannot be diagnosed with certainty in spite of the fact that in many ways the tachycardia behaved very much like a ventricular tachycardia. The possibility that the ectopic focus was located somewhere in the junctional tissue must be entertained. The configuration of the QRS wave when sinus rhythm was restored is quite similar to the QRS wave noted during the tachycardia and this fact favors a supraventricular origin of the tachycardia. Whatever is the mechanism of the abnormal rhythm, the results of various forms of treatment are quite interesting.

Treatment of this patient's tachycardia included practically every form of therapy that has been recommended for such disorders of rhythm. Most forms of therapy were used more than once and in sufficiently high dosage to guarantee their full pharmacologic effect with the possible exception of intravenous morphine, in which our dosage was much below that recommended by Gonzales Sabathie.4

Methods of increasing vagal tone were totally ineffective. The administration of Prostigmine, Mecholyl, and acetylcholine, and the application of carotid sinus pressure were ineffective. The administration of Prostigmine followed within five minutes by Mecholyl produced such an array of symptoms that there was little doubt that vagal stimulation of marked degree was obtained and yet there was no significant alteration in the tachycardia.

Blocking the vagus by the intravenous administration of 2.0 mg. atropine after the patient had received heavy doses of quinidine8, 12 orally likewise had no effect.

Blocking the sympathetic nervous pathways by the injection of procaine solution into the inferior cervical and first four thoracic ganglions on both right and left sides1 was without effect. Several days before the first procaine block, oral quinidine therapy had purposely been discontinued to allow the ventricular rate to return to its maximum level. It was reasoned that the higher initial ventricular rate might serve as a better control for any change resulting from the procaine block.

Continuous electrocardiographic tracings and constant auscultation over the heart during the procedure disclosed no alteration in the abnormal cardiac rhythm. Carotid sinus pressure after this block produced no change.

The second sympathetic block was performed at a time when the patient was under the influence of high quinidine dosage. Again there was no noticeable effect on the rhythm and this time the intravenous administration of atropine was ineffective. Finally, the administration of prostigmine resulted in no change. These procedures demonstrated to us that blocking all cardio-accelerator impulses
while at the same time blocking the vagus chemically, and later stimulating the vagus chemically produced no change in the abnormal rhythm.

The use of a potassium salt in conjunction with quinidine during the first episode of tachycardia led us to believe that it was of definite value in terminating the abnormal rhythm. We suspect, however, as a result of the numerous failures during the last bout of tachycardia, that potassium salts played a questionable role in the successful conversion to normal rhythm.

Magnesium sulfate was administered intravenously on two separate occasions and although there was severe flushing and sweating and some slowing of the ventricular rate, the basic rhythm remained unchanged. The dosage was adequate (20 cc. of 25 per cent solution) and the speed of injection was rapid (two minutes).

We do not wish to imply that all the above-mentioned forms of therapy may be ineffective in other cases of paroxysmal tachycardia. They simply did not work in our patient.

The experience with digitalis in this patient was the same as noted by others. There was no beneficial effect on the tachycardia from the standard 1.6 mg. lanatosid C and as digitoxin was further administered there was a steady rise in the ventricular rate. It was apparent that further administration of digitalis was definitely hazardous.

Intravenous procaine administration had no effect on the tachycardia except to produce some slowing of the ventricular rate.

The only drug of any value in our patient was quinidine. Quinidine was given orally to the point of tolerance (4.8 Gm. a day). It was effective in slowing the ventricular rate from 170 or 180 to 110 or 120. Anorexia, nausea, occasional vomiting, and excessive diarrhea interfered with attempts to increase the dose by mouth. Slowing the ventricular rate even by that amount was of considerable value in delaying the onset of congestive heart failure. However, conversion to normal rhythm by oral administration of quinidine was not accomplished.

Quinidine hydrochloride given intravenously has been the only successful form of therapy in this patient. The first two attacks of paroxysmal tachycardia were terminated after a dosage of 1.8 grams. During the last episode, quinidine was administered intravenously on eight separate occasions. Four of these trials were total failures and four were marked by conversion to normal rhythm for variable periods of time. Previous administration of digitalis could possibly account for lack of success in at least two trials. A third trial with the intravenous quinidine failed to terminate the tachycardia in spite of slowing the ventricular rate to 84. The administration of more quinidine at this point seemed unwise. The fourth failure to convert was marked by a long period of alternating paroxysmal tachycardia and normal sinus rhythm. Perhaps in this instance a larger dose of quinidine might have been successful.

Of the times when intravenous quinidine therapy was at least temporarily successful, there were two occasions when the abnormal rhythm was converted to normal sinus rhythm after the administration of only 1.0 Gm. of the drug. In each case, however, an additional 0.8 Gm. was injected, with the intention of depressing the irritable focus long enough to permit full recovery of the sinus node. Normal sinus rhythm persisted for one hour and for two hours respectively. The third partly successful result was obtained with 1.8 Gm. quinidine. In this instance, maintenance oral quinidine was given in a dosage of 0.4 Gm. every four hours. Normal sinus rhythm remained for twelve hours, after which the paroxysmal tachycardia reappeared. A higher maintenance dose of quinidine may have prevented the return of the tachycardia. The fourth and last intravenous injection of quinidine terminated the tachycardia and established a normal sinus rhythm which has persisted to date. On that occasion the patient received 3.2 Gm. of the drug over a period of four hours. At the end of that time he developed clinical signs of toxicity which included a state of coma during which he was completely unresponsive for a period of one hour, and had marked irregular respirations, cyanosis, and extreme weakness. The cardiac manifestations of toxicity included...
pulselessness for a short period of time; cardiac sounds were not audible and the only way it was possible to ascertain whether or not he was still alive was by means of the electrocardiogram. His blood pressure fell until it was unobtainable, and only gradually returned to its usual level during the following twenty-four hours. Urinary suppression lasted forty-two hours and although this may be a manifestation of quinidine toxicity alone, it is more likely that the low blood pressure and a disturbed electrolyte balance were largely responsible.

The electrocardiogram (fig. 8) revealed a progressive loss of the P wave, widening of the QRS complex to 0.28 second, and an increase of the Q-T interval to 0.5 second. The Q-T interval had been lengthened by about 39 per cent which is much beyond the 25 per cent range of safety usually recommended. This risk was deliberately taken since in this patient we found that the average intravenous dose of quinidine (1.8 Gm.), although successful on some occasions, failed completely on other trials. It, therefore, appears to us that the intravenous dosage of quinidine may vary not only from patient to patient, but also may vary in the same patient from time to time. It furthermore appears that when quinidine is used intravenously for the treatment of paroxysmal tachycardia it should be given in a dosage that is large enough to terminate the tachycardia. This may range from 0.6 Gm. to 1.8 Gm. or higher. If early recurrence of the tachycardia within the first twenty-four hours cannot be prevented by oral maintenance doses of quinidine, then the use of quinidine intravenously should be considered.

The final point of interest is the development of pericardial calcification over the left ventricle. It is quite possible that the area of calcification may involve the myocardium as well. Presumably this represents calcification following the original myocardial infarction. In time this may possibly further embarrass the heart. Whether or not it is playing a part in the recurrent tachycardia it is not possible to say.

CONCLUSIONS

A case of recurrent paroxysmal tachycardia is reported. Three separate attacks have been observed, one of thirty-seven days', another of two days', and a third of sixty-three days' duration.

Intravenous administration of quinidine was successful in terminating each attack. The dosage used was 1.8 Gm. quinidine hydrochloride in two attacks and 3.2 Gm. in the third.

Methods aimed at altering the nervous control of the cardiac rhythm were ineffective. Intravenous administration of magnesium sulfate and of procaine were also of no value. Digitalis failed to terminate the tachycardia and actually increased the heart rate.

The diagnosis of the type of tachycardia is uncertain although we consider it either a paroxysmal nodal or paroxysmal ventricular tachycardia in the presence of a left bundle branch block.

Calcification in the pericardium made its appearance during the two-year period of observation and the question was raised as to its possible role in the recurrent episodes of tachycardia.

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