The Use of Digitalis in Spite of the Presence of Ventricular Tachycardia

By J. S. Gilson, M.D. and F. R. Schemm, M.D.

The authors' observations throw some doubt on the validity of objections to the use of digitalis in the presence of ventricular tachycardia. These observations demonstrate that digitalis can be used liberally and that it does not appear to interfere with conversion to a normal mechanism. They suggest that digitalis may even set the stage for reversion by aiding the failing heart.

The presence of ventricular tachycardia has been regarded as one of the major contraindications to the use of digitalis.1-8 Our clinical observations, however, have led us to use digitalis somewhat more freely in cardiac disease in general than is usually recommended.9-18 The four observations on three patients presented in this paper illustrate instances of the intensive use of digitalis in the presence of ventricular tachycardia.

It is not assumed that the digitalis in these instances stopped the tachycardia, but its use, along with other supportive measures, was followed by marked clinical improvement and the cessation of ventricular tachycardia. These results were not to be expected from the usual literature on digitalis or ventricular tachycardia.1-8 In our observations signs and symptoms which could be attributed to digitalis intoxication (nausea, vomiting, diarrhea and ventricular tachycardia) disappeared with the intensive use of digitalis, indicating that those signs, including the ventricular tachycardia, were not due to digitalis effect or to digitalis intoxication.

Gratifying experiences9 with the use of digitalis in patients with disabling ventricular premature beats, heart block, and with severe acute myocardial infarction, all rather commonly regarded as relatively unsuitable conditions for the use of digitalis, led to its use in the first case reported in this paper, when rapidly progressing heart failure seemed to demand digitalis in spite of a persisting ventricular tachycardia.

Observation I. Case 1: L. G., age 53, a rancher. Admitted May 21, 1940. The tachycardia began thirteen days after admission for symptoms and findings diagnostic of an acute myocardial infarction in an anterior position (fig. 1, 5/22/40). The post-infarction period was characterized by one week of low grade fever, leukocytosis and by recurrent anjinal pain up to the onset of the ventricular tachycardia.

On the evening of the thirteenth day following admission (at 8 P.M. on June 3, 1940), after sitting up in bed for the first time that afternoon, the patient noted palpitation and discomfort in the chest with pain. There were no significant physical findings. The rectal temperature was 98.4 F. The heart rate was 128 per minute; the rhythm was regular.

Before the electrocardiogram was read the patient was given 10 cat units of digitalis in twenty-four hours. When ventricular tachycardia was recognized as the mechanism (fig. 1, 6/3/40), quinidine sulfate was administered. A total of 3.5 Gm. was used in forty-eight hours. The quinidine was discontinued when tinnitus, vomiting and a broadening of the QRS complex developed. Because the signs of heart failure were progressive and pulmonary edema developed, digitalis was resumed on the evening of June 7, four days after the onset of tachycardia. A total of 24 cat units was given by vein, with improvement in the signs of failure after 16 cat units and cessation of the tachycardia four hours after the twenty-fourth cat unit, 72 hours after the digitalis had been resumed. The tachycardia had lasted 176 hours, ending at 4 A.M. June 11. Nausea and vomiting ceased twenty-four hours after the ventricular tachycardia had stopped, in spite of the continuation of a maintenance dose of digitalis.

Digitalization was maintained for the next six years until death resulted from another acute myocardial infarction. No subsequent episodes of ventricular tachycardia were observed. The autopsy demonstrated an extensive old, and a smaller recent, infarction.
Comment: As in the other observations which follow, this patient developed his ventricular tachycardia following a myocardial infarction. Unlike the others he had received no digitalis prior to the appearance of the ventricular tachycardia. Quinidine was given to the point of toxic manifestations, yet the tachycardia was unaffected and signs of heart failure were progressing. Digitalis was given primarily because of ominous progression of the heart failure, but it was given cautiously in view of the presence of both an infarction and ventricular tachycardia. The heart failure, the tachycardia, and the vomiting all disappeared with the use of digitalis in full dosage. The patient survived, without recurrence of tachycardia, for six years, during which he received a daily dose of digitalis.

In spite of the free use of digitalis in 87 of 189 cases of acute myocardial infarction seen in the intervening eight years, no other instances of ventricular tachycardia were encountered on this service until 1948. Others which doubtless occurred were probably of too short duration for recognition.

Observation 2. Case 2: J. B., age 72, a retired cattle buyer. Admitted July 8, 1948. The episode of tachycardia began on July 9, 1948 at about 9 P.M., twenty-one hours after admission to the hospital. Admission had followed a two day history of pressure-like chest pains lasting up to three hours and only partially relieved by rest, but totally relieved by morphine. During the first twenty-one hours of admission he was asymptomatic and showed a heart rate of 62 to 69 beats per minute with no irregularity. The blood pressure was 112/88. The lung fields were clear. While reaching for a glass the patient noted a sudden strange sensation, totally dissimilar to his previous anginal pain. He was conscious of rapid beating of his heart, felt giddy, faint, and nauseated. Apprehension was marked.

Four years previously an acute myocardial infarction (fig. 2, 4/21/44) had been associated with a complete heart block and syncopal attacks, which were treated not only with adrenaline, but also with digitalis because of myocardial failure. The heart block disappeared and he was discharged on a “maintenance” dose of digitalis. He had been admittedly somewhat lax in taking it in the intervening four years.

At the beginning of the tachycardia the heart rate was 180 per minute and regular, although the radial pulse was so uneven in force as to simulate auricular fibrillation. This unevenness of the radial pulse was noted in Observation 4 also. Heart tones were dis-
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The blood pressure was 102/70. The lung fields were clear.

The electrocardiogram (fig. 2, 7/9/48), taken one hour after the onset, showed a characteristic ventricular tachycardia although clear-cut P waves could not be identified. With the previous case in mind, 0.4 mg. of digitoxin was given intravenously. Thirty minutes later a normal rate had reappeared and a normal mechanism was shown by the tracing (fig. 2, 7/10/48, 8:00 A.M.) together with changes compatible with a posterior myocardial infarction of indeterminate duration. Two hours after this electrocardiogram and twelve hours after the first episode another attack of tachycardia occurred, only to cease spontaneously in thirty minutes (fig. 2, 7/10/48, 9:30 A.M.).

The patient was then digitalized, receiving 1.0 mg. of digitoxin by vein in 18 hours, in addition to the 0.4 mg. given by vein with the first paroxysm of tachycardia. The nausea and vomiting associated with the attacks subsided during this completion of redigitalization. The following evening, on July 11, after another 0.2 mg. of digitoxin had been given by vein earlier in the day, another episode of tachycardia occurred. The patient was terrified; a reversion to normal rate occurred after morphine was given.

Digitalis was continued without further incident. He was treated as a case of recent myocardial infarction and discharged on his twenty-ninth hospital-day to continue his convalescent care at home. Digitalization has been maintained with from 1.0 mg. to 1.4 mg. of digitoxin per week, faithfully taken. During the past year and a half there has been no recurrence of palpitation or chest pain.

Comment: It seems quite obvious that digitalis neither caused, aggravated nor stopped these intermittent episodes of ventricular tachycardia. They began while the patient was taking a maintenance dose, yet subsided several times in the face of increased dosage. They have not recurred with the increased maintenance dose.

With the first myocardial infarction in 1944, digitalization coincided with the disappearance of heart block and associated Adams-Stokes syncope. The patient has now survived the original infarction six years and has survived his ventricular tachycardia more than one and one-half years in spite of an increase in his maintenance dose of digitalis.

Observation 3: Case A.T., age 54, crane operator. Admitted Nov. 18, 1948. Extreme palpitation

Fig. 2.—Case J.B. Observation 2. The first tracing of 4/21/44 shows a diagnostic pattern for posterior infarction with Q waves, arched S-T and inverted T waves in Leads II and III. The second taken four years later shows the ventricular tachycardia; independent P waves were not identifiable. The 8 A.M. tracing taken ten hours later, on 7/10/48, shows a normal sinus mechanism with a QRS-T pattern in Leads II and III diagnostic of a posterior infarction of recent origin. The fourth record, taken twenty-four hours later, was limited to a long strip of Lead II and shows one of the recurrences of the ventricular tachycardia. The last record shows return to sinus rhythm.
and malaise, sweating and apprehension, without pain, began about 18 hours before admission.

Fourteen months earlier, in September 1947, a severe myocardial infarction had occurred after one month of anginal pain. A pericardial friction rub persisted for ten days; arrhythmias, including auricular fibrillation and multiple ventricular premature beats, demonstrated by electrocardiogram, occurred intermittently; fever, without other demonstrable cause than the infarction and pericarditis, persisted for one month. Leukocytosis and the elevated sedimentation rate gradually subsided over a 4 week period. The

Fig. 3.—Case A.T. Observation 3. The first tracing, 9/10/47, was taken at the time of the original infarction and shows changes in both anterior and posterior myocardial surfaces. The next three records show the episode of ventricular tachycardia, occurring fourteen months later, during which time digitalis was being given. The record of 3 A.M. on 11/17/48 is the tracing taken on admission; that of 6 P.M., fifteen hours later, was taken after 1.8 mg. of additional digitoxin. The record of 8 A.M. on 11/18/48, four hours later, was obtained two hours after the total dose of digitoxin had reached 2.2 mg., a few moments after the mechanism had reverted to normal. The last tracing shows a return to a pattern very similar to that obtained fourteen months earlier.

ECG was compatible with a diagnosis of anterior myocardial infarction (fig. 3, 9/10/47). He was digitalized in the first week and was maintained for the intervening year on 1 cat unit of digitalis leaf taken with religious regularity.

This first episode of ventricular tachycardia began on Nov. 16, 1948, one week after returning to very light work. On examination the temperature was found to be 101.2 F., the heart rate 160 and regular, the blood pressure 120/100. His face was ashen and covered with cold sweat. Respirations were rapid and grunting but the lung fields were clear. The heart sounds were faint and rapid. The white blood count was 23,000. The electrocardiogram showed a ventricular tachycardia (fig. 3, 11/17/48, 3 A.M.)

He was given the usual supportive measures for myocardial infarction, and dicumarol after the diagnosis of a fresh infarction was established. No quinidine was used. On the contrary, he was redigitalized because of our experience in the preceding observations and our belief that patients frequently become insufficiently digitalized on the smaller daily doses of digitalis. Digitoxin, 2.0 mg., was given by vein over a thirty hour period without any effect on the tachycardia. During the entire thirty hours, sweating, ap-
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(fig. 3, 9/10/47 and 11/18/48). He was maintained on digitoxin, 0.2 mg. daily, and discharged on the twenty-third hospital day, the hospital program being continued at home.

Comment: We felt certain that this patient had received every dose of digitalis prescribed following the infarction in 1947. During the nine months immediately preceding the ventricular tachycardia he received a "maintenance" dose of 1 cat unit daily. Nevertheless, during redigitalization he tolerated an additional 2.2 mg. of digitoxin given rapidly by vein in thirty hours. Furthermore the symptoms of nausea and vomiting which could have been attributed to digitalis intoxication subsided instantly with the reversion to normal rhythm; this suggested that a therapeutic and not a toxic amount of digitalis had been given. Finally, the ventricular tachycardia itself, which some would feel had been caused by the preceding "maintenance" dose of digitoxin, ceased despite the additional "full digitalizing dose" of digitoxin.


He had been on markedly restricted activity for six months since recovery from the first attack of ventricular tachycardia and his second myocardial infarction described above. He had been faithfully taking digitoxin, 0.2 mg. daily, since the previous attack.

This second episode of ventricular tachycardia began with palpitation and "uneasiness" about 11 p.m. April 28, 1949 following a quiet visit with two friends. There was no pain.

On examination (April 29) the rectal temperature was 100 F. The heart rate at the apex was 170 and regular, but the radial pulse exhibited irregular force as noted in Observation 2. The heart sounds were of fair quality. No rales were heard at the bases of the lungs. The white blood count was 15,500. The electrocardiogram showed a ventricular tachycardia (fig. 4, April 29, 1949, 5 p.m.).

Because the effect of quinidine, in what was thought to be a third infarction, was feared even more than the effect of digitalis and because the

![Fig. 4.—Case A.T. Observation 4. The initial tracing on this plate and the final tracing of figure 3 are both interval records between the first and second severe episodes of ventricular tachycardia. The second tracing shows the ventricular tachycardia. The third shows the pattern after 2.6 mg. of digitoxin and prior to the use of quinidine. The tracing of 5/2/49 was taken after two doses of 1.3 Gm. of quinidine; the P waves are clearly seen. The tracing of 5/4/49 shows that the ventricular tachycardia was superseded by auricular flutter (arrows) after totals of 3.7 mg. digitoxin and 5.7 Gm. of quinidine had been given. The next tracing, twenty-four hours later, shows reversion to sinus rhythm. The last tracing shows persistence of the sinus rhythm and also that the QRS-T pattern following recovery from the attack is similar to the pattern preceding the attack.](http://circ.ahajournals.org/doi/10.1161/01.RES.4.1.282)
previous attack of ventricular tachycardia had responded coincident to rapidly given digitoxin in a total dose of 2.2 mg., digitoxin was again given intravenously when morphine and sodium phenobarbital had given no relief. During the first fifty-three hours 2.6 mg. of digitoxin were given intravenously with no effect on the tachycardia. He vomited three times after the first dose of digitoxin but had no nausea or vomiting thereafter. During this fifty-three hour period he became febrile, complained of substernal heaviness, and finally developed pulmonary edema, which subsided in three hours coincident to the use of tourniquets, phlebotomy, and a positive pressure mask. The pulmonary edema recurred sixteen hours later. At this recurrence (just after the record shown in fig. 4, May 1, 1949, 10 p.m.) quinidine lactate* was begun but was halted after 0.4 Gm. had been given because of rapidly increasing irregularity in pulse, cyanosis, and coma. He gradually improved in the succeeding twelve hours.

Although digitoxin was continued, quinidine lactate was again given intravenously on May 2 and 3 (the fourth and fifth days of the tachycardia). Doses of 1.3 Gm., followed in eight hours by 2.0 Gm., and in twenty-four hours by 2.0 Gm. were given intravenously in 5 per cent dextrose solution at a rate of about 0.5 Gm. of quinidine lactate per hour. The heart rate slowed progressively with each administration of quinidine, and an apparent broadening of the QRS complexes and reversal of the major deflections of QRS in Leads I and II occurred. In one record taken during these transient (one to two hours) slowings, P waves are clearly seen at a rate of 68 with a ventricular rate of 150 per minute (fig. 4, May 2, 1949). He became distinctly and progressively irrational during each administration of quinidine; and was controlled only for one to two hours by phenobarbital or morphine. Basal pulmonary rales persisted; cyanosis increased. One Gm. of potassium chloride was given intravenously on the evening of May 3 with no apparent effect.

He had received 3.7 mg. of digitoxin, 5.7 Gm. of quinidine, and 1.0 Gm. of potassium chloride when, on May 4, at 8 a.m., 112 hours after admission and 128 hours after symptoms had begun, his rate suddenly dropped from 200 per minute with a regular rhythm, to 92 with an irregular rhythm, shown by the tracing to be auricular flutter with a variable block (fig. 4, May 4, 1949, 9 a.m.). This occurred twenty hours after the last dose of quinidine and twenty-four hours after the last dose of digitalis.

The next day, five and one-half days after admission, his rhythm was regular and the electrocardiogram (fig. 4, May 5, 1949) showed a sinus rhythm with a rate of 90 per minute. The tracing of May 7, 1949 showed changes indicating a fresh infarction.

Within sixty hours after reversion to normal rhythm his general improvement was marked. Digitoxin, 0.2 mg., and quinidine, 0.6 Gm. daily, were continued as he convalesced uneventfully. In the nine months since his discharge on the thirty-sixth hospital day, there has been no recurrence of heart failure or tachycardia with 0.6 Gm. of quinidine daily and a heavy maintenance dose of 1.8 mg. of digitoxin per week.

Comment: This patient survived a third myocardial infarction, a second bout of persistent ventricular tachycardia which lasted almost six days, and two episodes of profuse pulmonary edema. It is, of course, impossible to judge with certainty to what extent the numerous adjuncts used (penicillin, vitamins, phenobarbital, morphine, oxygen, quinidine etc.) contributed to the recovery. He did, however, survive, and did revert to a normal mechanism while receiving large doses of digitoxin.

As in the preceding observation the nausea and vomiting, which might have been attributed to digitalis intoxication, cleared in the face of large amounts of additional digitalis. The ventricular tachycardia itself, occurring while the patient was receiving a daily dose of 0.2 mg. of digitoxin, might also have been regarded as a toxic effect of that digitalis dosage. The amounts of digitalis given should, according to widely accepted ideas, have prolonged the tachycardia if the digitalis were a precipitating cause. Yet reversion occurred at a time when a large amount of digitalis had been given in a relatively short time. If the reversion occurred as a result of the use of quinidine or any of the other measures employed, at least the digitalis given did not prevent the reversion.

Digitalis, which is accepted as valuable in the presence of acute myocardial failure with pulmonary edema, may well have determined survival in this case, in spite of the presence of ventricular tachycardia, even if the effect of the digitalis on the myocardium did not indirectly bring about the cessation of the ventricular tachycardia.

**DISCUSSION**

Authoritative interdictions to the use of digitalis in ventricular tachycardia abound in the

* Courtesy of Eli Lilly & Co.
literature. Gold	extsuperscript{1} says that there is "... only one drug which can abolish it, namely quinidine. Digitalis may make the condition worse. In any case it cannot abolish it." Goodman and Gilman	extsuperscript{2} state: "Digitalis should not be prescribed if tachycardia is of ventricular origin." Leaman	extsuperscript{3} says: "Digitalis is valueless, in fact it may even increase the rate." And in his therapeutic chart (after ventricular tachycardia) there appears the warning: "Digitalis dangerous." Levine	extsuperscript{4} states that, while "... in most cardiac conditions in which there is heart failure digitalis is indicated ...," the presence of ventricular tachycardia is a special exception and that, "when this arrhythmia is present digitalis will not only fail to improve the situation but may well worsen it." Burch and Reaser,	extsuperscript{5} in speaking of digitalis in paroxysmal tachycardia in general, say: "It should be used cautiously, if at all, in the presence of ventricular tachycardia as there is a possibility that the tachycardia will be converted to ventricular fibrillation." A few authorities, however, mention a limited use for digitalis in ventricular tachycardia. Thus, Herrmann and Hejtmanek	extsuperscript{6} state that "when digitalis is the precipitating factor (of ventricular tachycardia) it should, of course, be withheld. Occasional cases respond only to quinidine and digitalization ... and administration of digitalis is indicated in moderate to severe decompensation when it can be ruled out as a causative factor." And White	extsuperscript{7} says "... Digitalis intravenously ... should be tried only if other measures in the control of an obstinate attack have failed or if there are symptoms and signs of congestive failure." However, the usual view remains that of Williams and Ellis	extsuperscript{8} who warn that "Digitalis is best omitted if it has been used before the attack. Moreover, because we believe that digitalis was a causal factor in some of our cases, we were doubtful of the wisdom of using it in the treatment of an attack even when no digitalis has been taken."

In each of the four observations reported in this paper the ventricular tachycardia occurred following severe injury to the myocardium from a myocardial infarction. In each instance, the clinical picture was alarming and was associated with the characteristic objective findings of severe myocardial injury and a grave prognosis. In the patient who died six years after the observation reported here, the autopsy verified the extensive nature of the myocardial injury. Upon this severe damage in each case were superimposed episodes of ventricular tachycardia, which were prolonged (except in Observation 2 in which the tachycardia was intermittent). In three of the observations the tachycardia occurred while digitalis was in use because of antecedent or coincident myocardial failure. In all four observations the patients received amounts of digitalis after the onset of the ventricular tachycardia equivalent to, if not in excess of, what is commonly considered an average full digitalizing dose. Yet none died during the attack, all reverted to a normal mechanism and all survived their tachycardias for long periods. The reported survival rate is low in ventricular tachycardia: Williams and Ellis	extsuperscript{8} show about 35 per cent of 25 cases alive after one month, and Herrmann and Hejtmanek	extsuperscript{6} report only 3 out of 8 cases recovering. That death did not occur in the four instances reported here suggests that the digitalis did no harm and may actually have been helpful. If digitalis in proper dosages actually induces ventricular tachycardia the incidence of ventricular tachycardia in our series of cases of acute myocardial infarction should have been high. However, in the nine years from 1940-49, 87 of 189 cases were digitalized both for outspoken signs of congestive failure and for suspected myocardial failure without classical signs.	extsuperscript{9} Yet the uncorrected first admission mortality rate was only 10 per cent. There was no increased incidence of ventricular tachycardia as the four observations reported here are the only instances of ventricular tachycardia recognized in the 189 cases.

The fear of digitalis in the presence of ventricular tachycardia appears to have arisen chiefly from the observation that digitalis given continuously in large (presumably toxic) amounts, increases myocardial irritability and can produce ventricular tachycardia in both experimental animals and man. Apparently this led to the assumption that a patient getting the usual, or more than usual, amounts of digitalis has been poisoned by the drug if a ventricular...
Tachycardia appears. This assumption ignores the fact that the heart disease itself, and particularly myocardial infarction, can increase myocardial irritability and can produce a variety of disturbances of rhythm and rate, including ventricular tachycardia. About half the instances of ventricular tachycardia in many series reported in the literature had received no digitalis at all.

Our observations suggest that the patient may actually need more, rather than less, digitalis if heart failure is present or is developing during a ventricular tachycardia. The tachycardias ceased and the signs of digitalis “intoxication” as well as the symptoms and signs of heart failure disappeared when digitalization was achieved or re-enforced. The beneficial effect of the digitalis in these cases seemed to be in its support of the failing heart. In the first observation the dyspnea and râles began to subside before the tachycardia ceased; in the third observation, the signs of heart failure subsided with redigitalization and the cessation of the tachycardia; and in the fourth observation the two episodes of prolonged profuse pulmonary edema were survived while liberal amounts of digitalis were being superimposed on a liberal maintenance dose.

It cannot, of course, be stated that digitalis actually stopped these tachycardias, yet it seems pardonable to speculate that there may have been some beneficial effect from the digitalis on the uninfarcted portion of the failing myocardium which lessened its irritability and favored the cessation of the tachycardia. In any event it is our impression that a freer use of digitalis for the failing heart in the presence of ventricular tachycardia is justified.

Summary

1. Four observations on three patients with ventricular tachycardia who were given large doses of digitalis are reported. The occurrence of remote and/or recent severe myocardial infarction was established in each instance.

2. In the first observation the patient received 10 cat units of digitalis in two days and, after two days of quinidine, another 24 cat units in three days. In the second observation, 1.4 mg. of digitoxin were given in thirty hours; in the third, 2.2 mg. of digitoxin in thirty hours; and in the fourth, 3.7 mg. of digitoxin in 112 hours. In the last three observations the doses shown were superimposed on “maintenance doses” of digitalis which the patients were receiving when the ventricular tachycardia began.

3. The ventricular tachycardias ceased with, or in spite of, these doses of digitalis. Heart failure was controlled with the digitalis in spite of the presence of the tachycardia. Symptoms, which could have been interpreted as being due to digitalis intoxication, disappeared in spite of its continued use.

4. The observations suggest that digitalis is not as harmful in the presence of ventricular tachycardia as the literature leads one to believe. The survival of these patients for prolonged periods suggests that support of the failing myocardium with digitalis may be an important aspect of treatment in prolonged ventricular tachycardia.

References

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J. S. GILSON and F. R. SCHEMM

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