# REPETITIVE PAROXYSMAL TACHYCARDIA

BY

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Paroxysmal tachycardia might be defined as sudden attacks of extreme acceleration of the heart that last from a few seconds to a few days and end abruptly as they begin. In the average patient with paroxysmal tachycardia the paroxysm is the exception: normal rhythm is the rule; the difficulty is to obtain an electrocardiogram during the attack. We have become interested in a special variety where recurrent paroxysms are the rule and normal rhythm is the exception; the difficulty now is to obtain the normal electrocardiogram. This variety is here described as *repetitive*. It is characterized by recurring short runs of auricular, nodal, or ventricular extrasystoles, runs or paroxysms of tachycardia in fact, almost constantly present for months or for years and only occasionally interrupted by the normal sinus rhythm. We do not include under this term the recurrent brief paroxysms of tachycardia which may happen to be portrayed on an isolated electrocardiogram. It is true that in a sense they are repetitive, but this is an incident, not a clinical state like that of repetitive paroxysmal tachycardia where the play of paroxysms almost displaces normal rhythm. Its separate consideration is justified on clinical grounds; for it more often affects children and young people in whom it presents a novel problem in prognosis. There are few conditions in children of which it can be said that they are likely to " grow out of it," but this appears to be one.

It is 20 years since one of us (J. P.) came across our Case 1, and since then we have records, more or less complete, of 40 cases of repetitive paroxysmal tachycardia. Cases to be quoted below have been reported singly or perhaps in twos or threes. Our plan was to place on record for the first time a large group of such cases. We have since found, however, in the monograph of Léon Gallavardin (1946), a study of this nature under the title " Extrasystolie auriculaire en salves," differing from ours in that it relates to auricular paroxysms only.

Lewis (1909) made the first observation of this kind in a man of 41 with an otherwise healthy heart. By continuous polygraphic record during 109 minutes he was able to count 54 short paroxysms of auricular paroxysmal tachycardia at a rate of 133–184. Paroxysms of over 1 minute duration were rare; the shortest attack consisted of a succession of a few ectopic beats, the longest lasted 7 minutes. Though occurring at rest, they were more frequent on exertion. Even at a slow rate the pulse was hardly ever regular because of single or multiple auricular extrasystoles.

White (1920) drew attention to the " markedly rapid but not immediate " increase in rate and similar offset during the short and continuously recurring attacks of auricular paroxysmal tachycardia in his patient observed for five years. Scott (1922) abolished with quinidine the short and continuously recurring attacks of ventricular tachycardia which were precipitated by exertion and were the cause of serious invalidism in this patient.

Louis Gallavardin (1922, *a*, *b*, and *c*) insisted on their distinction from ordinary paroxysmal tachycardia and described the ventricular variety in one case (Gallavardin, 1922*b*) and the auricular variety in three others (Gallavardin, 1922*c*), under the name of " Extrasystolie

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241

á paroxysmes tachycardiques." Later with Dumas (Gallavardin and Dumas, 1924) he used the name of " tachycardie en salves " in adding three more supraventricular cases. He also noticed in some that effort produced the paroxysms (Gallavardin, 1922a) and that the ventricular form frequently caused attacks of giddiness comparable with those in Stokes-Adams disease. Later with Veil (Gallavardin and Veil, 1929a and b) he added four similar cases which also include the one already published in 1922; after 15 years of observation the patient was still having the short and continuously recurring paroxysms of ventricular tachycardia.

Cassidy (1924) recorded an " incessant " paroxysmal tachycardia in an otherwise healthy child of 8, who had short bursts of auricular paroxysmal tachycardia every few minutes, constantly recurring during two years of observation.

In Gilchrist's (1925–6) Case 5 with ventricular tachycardia mostly on exertion " an intimate mixing of short ventricular paroxysms, multiple and isolated ventricular extrasystoles and occasional sinus beats " were continuously recorded for six months until they were abolished with quinidine.

Wenckebach and Winterberg (1927) reported two instances of almost continuous auricular paroxysmal tachycardia with varying A-V block, watched for two and three months respectively. In three other cases short bursts of ventricular paroxysmal tachycardia were constantly found, and in two of these effort produced longer attacks. They conclude that it would be better to distinguish a persistent form of paroxysmal tachycardia from the usual one occurring in attacks, as one does in flutter.

Single cases of the ventricular variety were described by Jones and White (1926–7), Schliephake and Graubner (1928), Lutembacher (1929), Andersen (1931), and Bérard (1931). In Gallavardin and Froment's (1930) case salvos of ventricular tachycardia co-existed with auricular paroxysmal tachycardia of the classical type. In McMillan and Bellet's (1931–2) patient, also with the ventricular form, a Cæsarean section was successfully performed while she was in the state of paroxysms. Wilson and others (1932) in adding four similar cases insist on the absence of structural heart disease, the relation of the paroxysms to exertion, their brevity, and the effectiveness of quinidine. This was also successfully employed in other ventricular cases quoted above.

Froment (1932) dedicates part of his admirable monograph on ventricular paroxysmal tachycardia to the " Extrasystolie ventriculaire bénignes á paroxysmes tachycardiques." He draws a sharp distinction between these and the " grave," " terminal," or " prefibrillatory " ventricular tachycardias and insists that the denomination of ventricular paroxysmal tachycardia should be reserved for these only while the former benign condition should be called extrasystolia. Transitional forms, however, are described where prolonged observation was necessary before placing the case into the one or the other group. Diagnostic criteria are given in favour of " extrasystolia," namely, the uniform shape of the extrasystolic complexes forming the tachycardiac runs, the normality of the intervening sinus beats, the youth of the patient, the integrity of the cardiovascular system, and the long evolution of the arrhythmia. Twenty cases are reported from the literature and no original cases are added.

The prognosis in general has been reported good. An exception was the 56-year-old patient of Clerc et al. (1933) with habitual ventricular tachycardia who died suddenly. Then, Elliott and Fenn (1934) described a 29-year-old woman in whom ventricular paroxysmal tachycardia with shorter or longer intermissions lasted for more than three months, after which she died of congestive heart failure. Each of these patients had an enlarged heart and an abnormal cardiogram between the attacks. Maddox (1947) has added a case in which there was a sudden mortal termination. Routier and Puddu (1936) and Routier (1937), describing three cases of benign ventricular extrasystoles in the form of paroxysmal tachycardia, also stress the good prognosis, provided there is no cardiac enlargement and the cardiogram is normal between the attacks. Campbell and Elliott (1939) also believe that the type " of very short

paroxysms recurring frequently, may be found with a normal heart and seems to be of no serious significance, even though it is ventricular."

Fine and Miller (1940) describe an "orthostatic" auricular paroxysmal tachycardia observed for two years, in whom the attack could be produced at will by change of posture, though it was also found sometimes at rest and in the supine position. Four years later the patient, who had congenital lues, died suddenly during malaria therapy (Miller and Perelman, 1945). On the other hand a similar patient, a healthy girl of 14, with "chronic auricular tachycardia" at rest and especially on change of posture, was able to live an active life during three years of observation (Miller and Perelman, 1945). Peters and Penner (1946) describe an orthostatic paroxysmal ventricular tachycardia successfully treated with quinidine. Graybiel and White (1946), in commenting on a cardiogram of recurrent auricular paroxysmal tachycardia, find it decidedly uncommon and believe that it is "in itself no evidence of heart disease."

Léon Gallavardin (1946) in an exhaustive study of the auricular extrasystole has collected 31 cases of "extrasystolie auriculaire en salves," partly original, partly previously published by his father (Louis Gallavardin) and others, with observation periods ranging from a few months to 29 years. In two-thirds of the cases the arrhythmia was the only cardiac abnormality while in the remaining one-third heart disease was present. He insists on distinguishing the arrhythmia characterized by the short and continuously recurring attacks from ordinary paroxysmal tachycardia, and gives a complete description of its clinical and electrocardiographic peculiarities.

In addition to these references mostly describing repetitive paroxysmal tachycardia separately, a number of examples are included in papers dealing with paroxysmal tachycardia in general. Williams and Ellis (1943) in analysing 36 cases of ventricular tachycardia found 12 belonging to the "intermittent" type. This group which included their only patient with no organic heart disease carried a less serious prognosis than the other group with persistent ventricular tachycardia. In Cooke and White's (1943) list of 32 cases of ventricular paroxysmal tachycardia, Cases 24, 25, and 27, and Freundlich's (1946) Case 3, probably belong to this variety, and so do several of Barker, Wilson, and Wishart's (1943) cases; their Case 1 is our Case 12, and Campbell's (1945) Case 2 is our Case 2.

The various names under which this kind of paroxysmal tachycardia has been described indicate the uncertainty hitherto felt on the subject. While some hold that the relation to exertion or posture is the most characteristic feature, others stress the "incessant," "chronic," or "habitual" nature of the paroxysms. The name "repetitive" seems to us to express best the very frequent recurrence of the paroxysms.

## BASIS OF INVESTIGATION

The following account is drawn from a series of 40 patients with repetitive paroxysmal tachycardia. Most of these were seen personally at the Cardiac Department of the London Hospital or at the National Heart Hospital; some were seen privately and others are added through the courtesy of friends. Sometimes the unusual character of the pulse, sometimes the routine cardiogram itself, led to the recognition of this variety of paroxysmal tachycardia. Apart from clinical and electrocardiographic examination, observations were frequent to obtain confirmation of its repetitive nature. The war has made a follow-up more difficult, but some have responded (see Tables).

## CLINICAL FEATURES

Ages extended from the youngest aged 4 years to the oldest aged 75. The auricular form is common in those under 40 and particularly so in children. Flutter and fibrillation in this form are commoner in the adult than in the young, as would be expected. The sex incidence is males : females about 2 : 1.

Observations ranging from 18 years to 3 months was possible in more than half the cases (see case notes and Tables).    The longest the paroxysmal state persisted was 10 years.    Eleven patients were seen once or twice only.    The diagnosis was here based on medical reports of almost incessant tachycardia or arrhythmia, on their long-standing symptoms, and of course on the characteristic electrocardiogram.

*Symptoms* and clinical features were similar in all groups irrespective of the electrocardiographic pattern.    The common symptom was palpitation, usually continuous and independent of exertion; a few complained of slight breathlessness in addition.    Six had syncopal attacks, which in three (all ventricular) constituted the main feature (Cases 32, 33, and 39).    Unfortunately we never had an opportunity of observing the patient when they occurred.    In the absence of evidence of heart-block at any time, we do not regard them as examples of Stokes-Adams attacks (Parkinson, Papp, and Evans, 1941).    Symptoms were often present over many years (Table I), and were severe enough in seven (Cases 3, 7, 12, 15, 19, 32, 39) to restrict

TABLE I

CASES OF REPETITIVE AURICULAR PAROXYSMAL TACHYCARDIA

| Case No., Sex, and Age | Duration of symptoms when first seen | Under observation | Repetitive paroxysms observed | Follow-up |
|---|---|---|---|---|
| 1. M.  6 | 1 month | 18 years | 10 years | Well, no paroxysms. |
| 2. M.  7 | — | 6 years | 5 years | — |
| 3. F.  22 | 5 years | 6 years | 6 years | — |
| 4. M. 20 | 2 years | 5 years | 5 years | — |
| 5. M. 11 | — | 7½ years | 3 years | Still paroxysms. |
| 6. F.  30 | — | 3½ years | 7 months | — |
| 7. M. 25 | 2 years | 2 years | 2 years | — |
| 8. M. 11 | — | 2¼ years | 2¼ years | — |
| 9. M. 46 | — | 2¼ years | 2¼ years | Still paroxysms. |
| 10. M.  6 | 2 years | 9 months | 9 months | Still paroxysms. |
| 11. F.  11 | — | 3 months | 3 months | Well after 4 yrs.    No paroxysms. |
| 12. M. 39 | 9 years | 5 years | 6 months | — |
| 13. M.  9 | 5 years | — | ? 9 years | Well after 13 yrs.    No paroxysms. |
| 14. M. 59 | 10 years | 4 years | 4 years | Still paroxysms. |
| 15. F.  50 | 20 years | 8 months | 8 months | Still paroxysms. |
| 16. F.  23 | 14 months | 10 months | 3 months | Well, no paroxysms. |
| 17. M. 35 | — | 5 years | 5 years | Still paroxysms. |
| 18. M. 18 | — | 4 years | 4 years | Still paroxysms. |
| 19. F.  20 | — | 3 years | 3 years | — |
| 20. M. 18 | — | 1 year | 1 year | Still paroxysms. |
| 21. M. 18 | — | 6 months | 1 month | Well, no paroxysms. |
| 22. M. 19 | — | 4 years | 2 months | Well, ? paroxysms. |
| 23. M. 39 | — | Seen once | — | — |
| 24. M. 37 | 3 weeks | Seen once | — | — |

TABLE II

CASES OF REPETITIVE FLUTTER (CASES 25–28) AND OF AURICULAR FIBRILLATION (CASE 29)

| Case No., Sex, and Age | Duration of symptoms when first seen | Under observation | Repetitive paroxysms observed | Follow-up |
|---|---|---|---|---|
| 25. F.  34 | months | 3 years | 3 years | — |
| 26. M. 55 | — | Seen once | — | — |
| 27. M. 52 | months | Seen once | — | Well after 16 yrs.    ? paroxysms. |
| 28. M. 75 | 5 years | Seen once | — | — |
| 29. M. 56 | 2 years | Seen once | — | Failure after 4 years. |

TABLE III

CASES OF REPETITIVE NODAL PAROXYSMAL TACHYCARDIA

| Case No., Sex, and Age | Duration of symptoms when first seen | Under observation | Repetitive paroxysms observed | Follow-up |
|---|---|---|---|---|
| 30. F. 22 | 3 months | 2 years | 2 years | Less palpitation. |
| 31. F. 56 | 13 years | 3½ years | 3½ years | — |

TABLE IV

CASES OF REPETITIVE VENTRICULAR PAROXYSMAL TACHYCARDIA

| Case No., Sex, and Age | Duration of symptoms when first seen | Under observation | Repetitive paroxysms observed | Follow-up |
|---|---|---|---|---|
| 32. F. 35 | 1 year | 13 years | 2½ years | Well after 12 yrs. No paroxysms. |
| 33. M. 20 | 4 years | 1 year | 1 year | Still paroxysms. |
| 34. F. 4 | 2 months | 11 months | 11 months | Still paroxysms. |
| 35. M. 36 | 4 years | 4 months | 4 months | Well after 10 years. No paroxysms. |
| 36. M. 57 | 1 year | Seen once | — | Well after 4 yrs. No paroxysms. |
| 37. M. 57 | 12 years | Seen once (hypertension) | — | Died in failure after one yr. |
| 38. F. 60 | 1 year | Seen twice | — | Well after 9 yrs. No paroxysms. |
| 39. F. 67 | 1 year | Seen twice | — | Well after 5 yrs. No paroxysms. |
| 40. F. 45 | 2 years | 1½ months | 1½ months | Still paroxysms. |

their physical activities. In the others the disability was minimal, though of six who were in the Services, three (Cases 4, 7, 33) had to be discharged. Seven children and seven adults were symptomless.

It is a remarkable fact that evidence of organic heart disease was found only in three patients. Case 37 with the ventricular form had hypertension and cardiac enlargement; he died suddenly three years later in failure. Case 40, also with the ventricular form, had slight cardiac enlargement of obscure origin and inversion of T I. Case 8 with the auricular form had congenital heart disease (atrial septal defect). Case 14 and Case 15, both with the auricular form, had toxic goitre; and Case 32, with the ventricular form for 5 years, had only single ventricular extrasystoles when six years later she was found to have a goitre.

ELECTROCARDIOGRAPHIC FEATURES

The 40 cases were classified according to the electrocardiographic findings as follows:

(1) Repetitive auricular paroxysmal tachycardia, 24 cases.
(2) Repetitive flutter, 5 cases.
(3) Repetitive nodal paroxysmal tachycardia, 2 cases.
(4) Repetitive ventricular paroxysmal tachycardia, 9 cases.

Repetitive auricular fibrillation might have formed another group, but it was seen only once and then only a single cardiogram was obtained. This case is included in Group 2.

(1) REPETITIVE AURICULAR PAROXYSMAL TACHYCARDIA

In 16 patients—they include the six cases of latent and partial block—the same type of extrasystole always formed the runs whether short or long (Fig. 1). P was inverted in leads I and II in one case, in leads II and III in seven, and was upright in six; it was bifid in one case and diphasic in one. If upright, the P always differed in some respect from the normal P of the sinus beat.
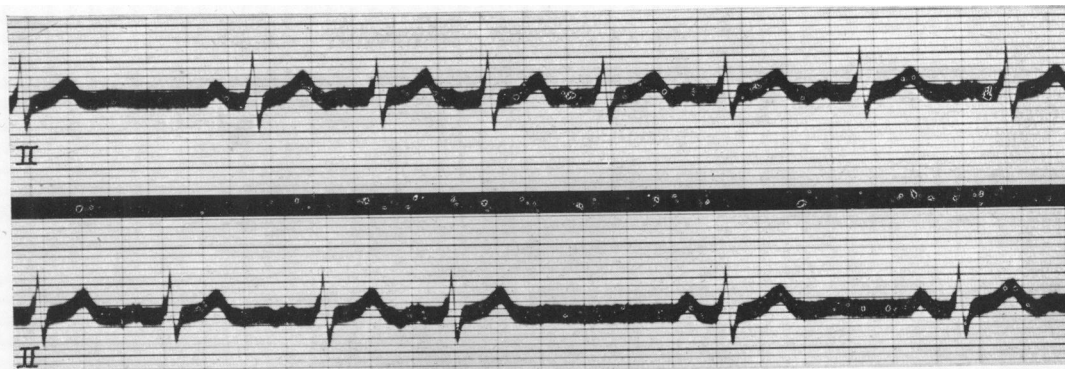
FIG. 1.—Repetitive auricular paroxysmal tachycardia (R.A.P.T.).   Case 23.   Beginning and end of paroxysm which is irregular, rate 90.   Note change in P.
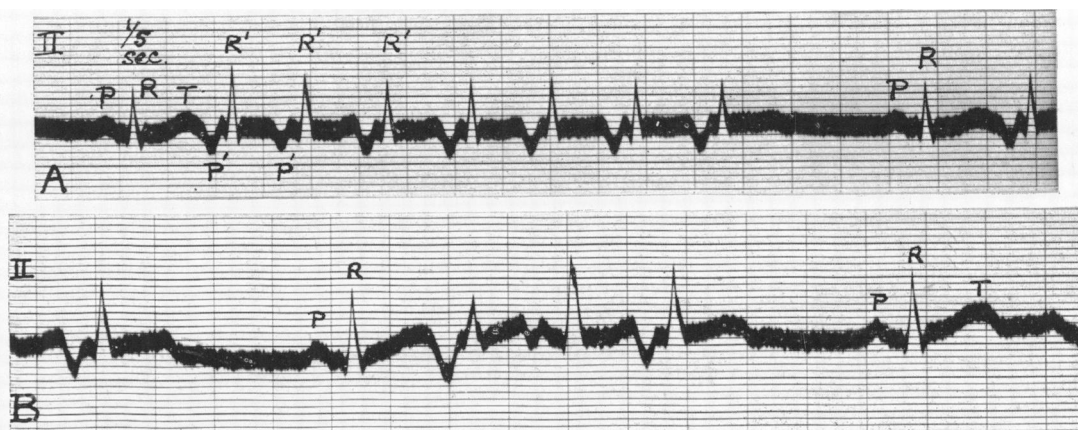


FIG. 2.—R.A.P.T.   Case 1.   (A) Run of auricular tachycardia, rate 170, regular.   (B) Short run of multiform auricular extrasystoles at a similar rate, but irregular.

In five patients, besides the recurrent short paroxysms like ordinary paroxysmal tachycardia as described above (Fig. 2A), there were many other brief paroxysms of an extrasystolic type—apparently runs of multiform auricular extrasystoles, irregular and some with bizarre ventricular complexes (Fig. 2B).

In two patients (Cases 7 and 14) these extrasystolic runs alone were recorded (Fig. 3). In 4 out of 24 cases, blocked auricular extrasystoles added to the disorder of the rhythm (Fig. 4). They were frequent during digitalis treatment.   The rate in children was around 150.   The rate in adults was noticeably lower; the highest was 150 in Case 3, the lowest 90 in Case 5, and on the average it was 120–130.   The rhythm during paroxysms of paroxysmal tachycardia type was regular in 15, more or less irregular in 7 where the paroxysms were of a low rate (around 100) and of a short duration (3–7 beats) (Fig. 1).   Even if regular, a slight slowing might occur towards the end of the paroxysm through a slowing of the last beats or through a slight prolongation of the P–R interval.

*Repetitive Auricular Paroxysmal Tachycardia with Block.*   During paroxysms latent block and higher grades of A-V block were found in 7 cases.   The P–R interval during paroxysms was prolonged to 0·26 sec. in Case 13;   Case 18 had Wenckebach periods (Fig. 5A), and Case 9
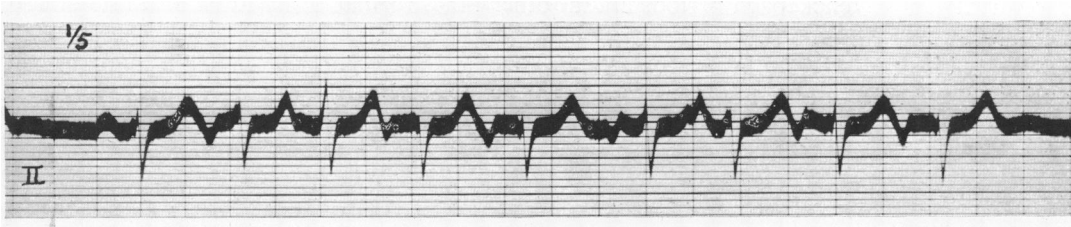
FIG. 3.—R.A.P.T. Case 14. Run of multiform auricular extrasystoles, rate 145, very irregular, forming repetitive paroxysms.



FIG. 4.—R.A.P.T. Case 6. Lead I: run of auricular paroxysmal tachycardia, rate 145, irregular. Lead II: groups of multiform auricular extrasystoles. Lead III: blocked auricular extrasystole.
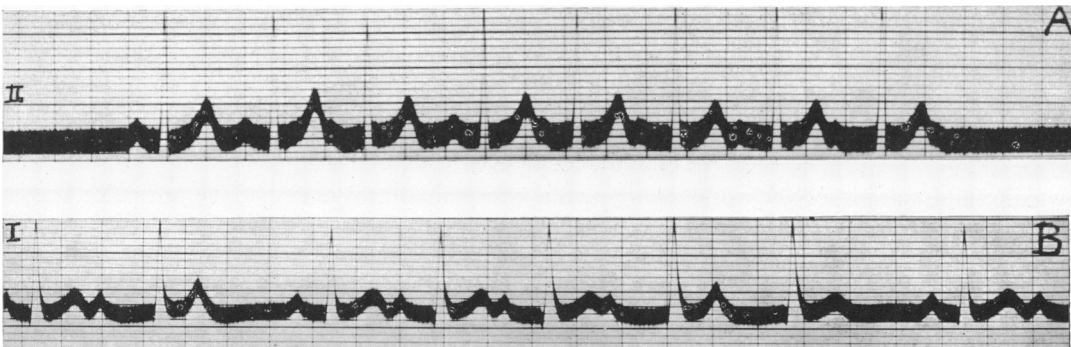


FIG. 5.—R.A.P.T. with Wenckebach periods. (A) Case 18, prolongation of P–R interval at the end of paroxysm from 0·16 to 0·24 sec. (B) Case 9, progressive prolongation of P–R interval from 0·18 to 0·36 sec. during paroxysm. Dropped beats in both cases; rate 120, slightly irregular.

showed at times Wenckebach periods and at other times regular tachycardia at a rate of 130 with P–R 0·2 sec. (Fig. 5B).

Heart block was found in 4 cases. In Case 2, 2 : 1 A-V block developed as soon as the auricular rate rose to about 300, in Case 16 when about 200 (Fig. 6). In Case 12, block of
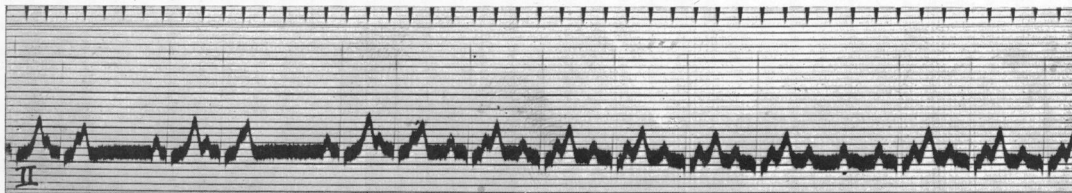


FIG. 6.—R.A.P.T. with 2 : 1 A-V block. Case 16. Auricular rate 200, regular, ventricular rate half.

2 : 1 and of higher degree might be present at a rate of 160–170, while at other times normal conduction was found at a rate of 200. In Case 8, 2 : 1 block was recorded all the time except when quinidine reduced the auricular rate from 180 to 105.

Case 8 had congenital heart disease and he always had a prolonged P–R interval of 0·22; Case 2 when normal rhythm became re-established also had a P–R interval of 0·24 sec. and a right bundle branch block of the wide S type in addition. The P–R interval was also pro-longed and P became bifid in Case 12 when seen in sinus rhythm two and five years later. In Case 16, 2 : 1 A-V block was found only on one occasion.

After the observations of Evans (1944) who found 2 : 1 block in 27 consecutive cases of auricular paroxysmal tachycardia, it is remarkable that among our 24 patients with repetitive auricular paroxysmal tachycardia only three had 2 : 1 A-V block. Admittedly the $CR_1$ chest lead was not applied, but in most of our records (e.g., Fig. 2A) it is obvious that it is a 1 : 1 and not a 2 : 1 rhythm. The slower auricular rate may be a partial explanation.

## (2) REPETITIVE AURICULAR FLUTTER

Repetitive flutter is very rare and few examples have been reported. Ritchie (1912) described one as intermittent auricular flutter; Semerau (1918) and Wolferth (1925) each reported two cases of intermittent impure flutter, and East and Bain (1936) one further case (Fig. 135). The physiological mechanism of impure flutter was explained by Lewis (1920) as a variation in the path, or in the speed, of the circus movement from cycle to cycle.

Ordinary flutter in the repetitive form must be exceptional. We observed it in Case 8 with repetitive auricular paroxysmal tachycardia and 2 : 1 A-V block once, and then for a short time only.

In our four cases of repetitive flutter the short paroxysms of 1–3 sec. duration are always introduced by an auricular extrasystole; this is followed by more or less irregular auricular activity at a rate of 200–370 with a completely irregular ventricular activity of 120–200. In Case 28, in whom the paroxysms could be produced at will by swallowing (Fig. 7), there is a waxing and waning of the auricular rate, the approximate distances of the auricular waves during an attack of 8 cycles being 34, 30, 12, 16, 18, 18, 28, 30, and 34 hundredths of a second, corresponding to an auricular rate 180–370. The paroxysms in Cases 26, 27, 28 are followed by a pause, but not in Case 25 (Fig. 8). Case 28 in other records on the same occasion had an almost regular auricular tachycardia (or flutter) with 2 : 1 block.

In the only instance of repetitive auricular fibrillation (Fig. 9) the paroxysm in lead II is introduced by an auricular extrasystole, followed by another which initiates auricular fibril-lation. Lead I of the same record shows auricular fibrillation; lead III sinus rhythm with auricular extrasystoles. The history favoured the diagnosis of repetitive auricular fibrillation,

FIG. 7.—Repetitive flutter; each paroxysm on swallowing. Case 28. Waxing and waning of auricular rate from 340 to 200 (dotted), ventricle irregular 200–100.
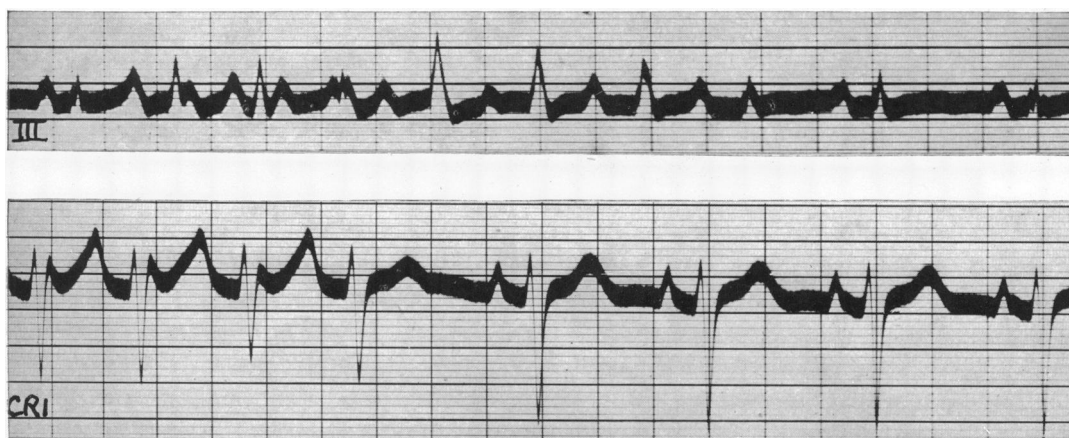


FIG. 8.—Repetitive flutter. Case 25. Auricular rate 380, slightly irregular; ventricular rate 160, irregular. $CR_1$ chest lead: flutter waves.

but the only record obtained of it is that here described. We have since seen another case of this kind with a similar electrocardiogram.

### (3) REPETITIVE NODAL PAROXYSMAL TACHYCARDIA

Though Case 1 was once seen in inferior nodal tachycardia, and isolated nodal extrasystoles occasionally appear in records of repetitive auricular paroxysmal tachycardia, repetitive nodal paroxysmal tachycardia itself is rare and only two examples were found.

Case 30 shows regular nodal rhythm at a rate of 64, with occasional sinus beats while at rest. Sinus rhythm at rest was recorded only once during two years of observation. Moderate effort produced nodal tachycardia at a rate of 130–140 with no visible P waves. In Case 31 an inverted P follows R at a short distance, and gradual slowing occurs at the end of paroxysms (Fig. 10).

Fig. 9.—Repetitive auricular fibrillation. Case 29. Lead I: auricular fibrillation. Lead II: auricular extrasystole initiating period of fibrillation. Lead III: normal rhythm with auricular extrasystoles.
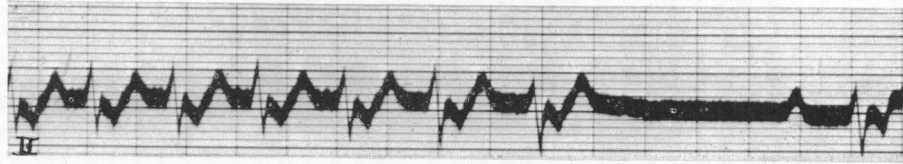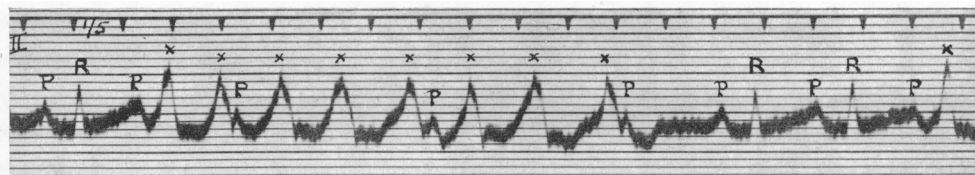


Fig. 10.—Repetitive nodal paroxysmal tachycardia. Case 31. End and beginning of paroxysm. P–R interval, 0·24 sec. after paroxysm.

### (4) Repetitive Ventricular Paroxysmal Tachycardia

Case 32 sometimes showed ventricular paroxysmal tachycardia with uniform ventricular complexes at an approximate rate of 200 and an auricular rate of 150 (Fig. 11A), and sometimes multiform ventricular extrasystoles in groups separated from each other by one or two sinus beats (Fig. 11B). Case 33 had regular ventricular tachycardia at a rate of 120 when seen twice in 1943 and once in 1944. Under digitalis treatment sinus rhythm reappeared for a short time; a few days later, however, in spite of continued digitalis treatment, runs of ventricular extrasystoles were again found, and after one month these repeated runs were still present (Fig. 12). Extrasystoles of different origin can be seen in the records of Case 39, but runs of ventricular tachycardia are always produced by the same interpolated ventricular extrasystole and are not followed by a pause (Fig. 13A and B). Case 38 (Fig. 14) with incessant bouts of irregular ventricular tachycardia at a maximum rate of 260, the highest in the series, was well and free from attacks when seen nine years later at the age of 69. The irregularity is even more obvious in Case 40 (Fig. 15) where retrograde conduction is evident as it is in Case 33 (Fig. 12) and Case 34. Except for Case 37, who died three years later, Case 40 is the only one with an enlarged heart and an abnormal cardiogram which here showed an inversion of T I of the sinus beat (Fig. 15). In all other records of this series the intervening sinus beats were normal and even during prolonged repetitive paroxysmal tachycardia the T of the sinus beats never seemed to change from upright to inverted. This is surprising in

FIG. 11.—Repetitive ventricular paroxysmal tachycardia (R.V.P.T.). Case 3. (A) Run of ventricular tachycardia, rate 240, almost regular; auricular rate 150. (B) Run of multiform ventricular extrasystoles at a similar rate, but irregular.
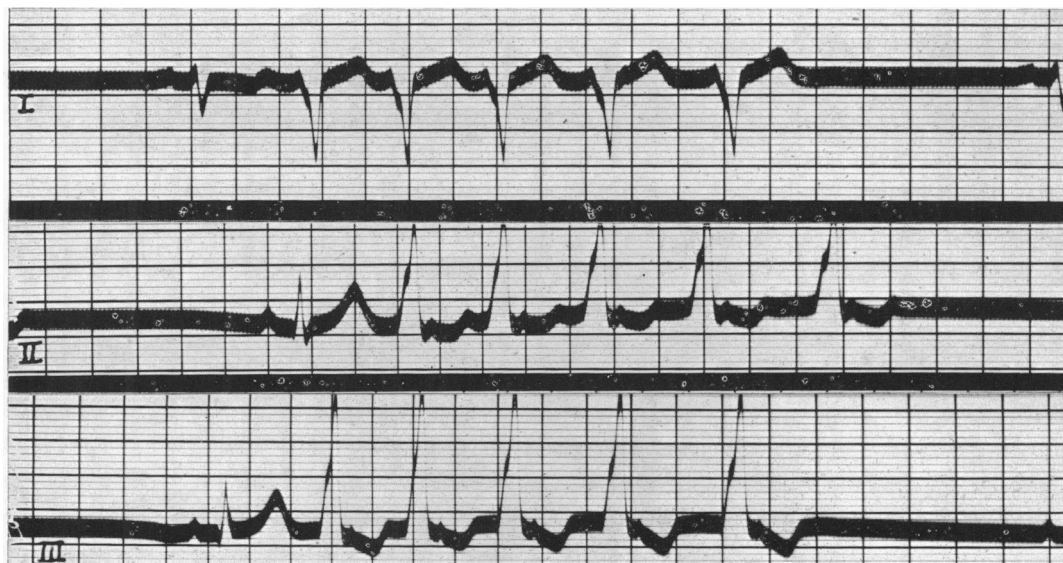


FIG. 12.—R.V.P.T. Case 33. Gradual waning of ventricular rate from 140 to 110. Retrograde auricular conduction seen in leads II and III.
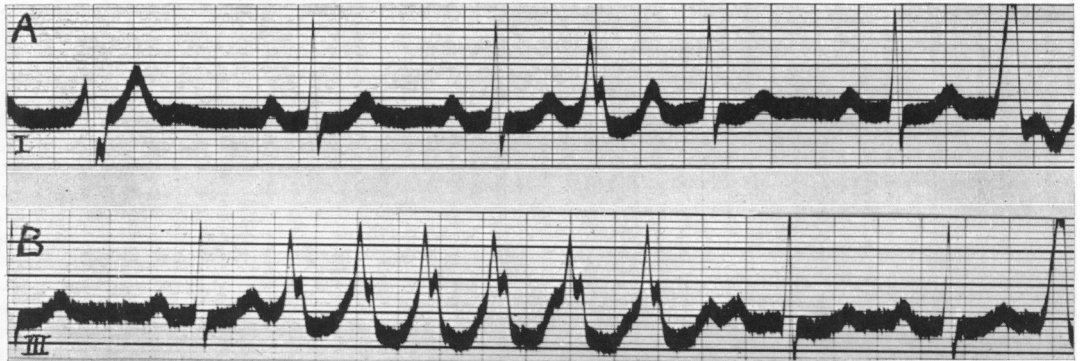
FIG. 13.—R.V.P.T.  Case 39.  (A) Multiform ventricular extrasystoles, one interpolated, identical with those in B.  (B) An interpolated paroxysm at a regular rate of 200.  P–R interval prolonged to 0·26 sec. after paroxysms.
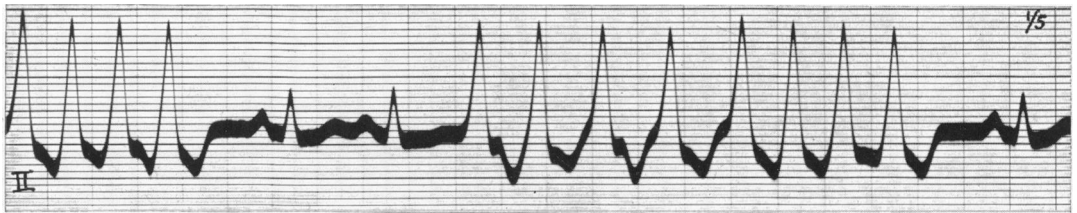


FIG. 14.—R.V.P.T.  Case 38.  Sudden change of rate during paroxysms from 210 to 270.
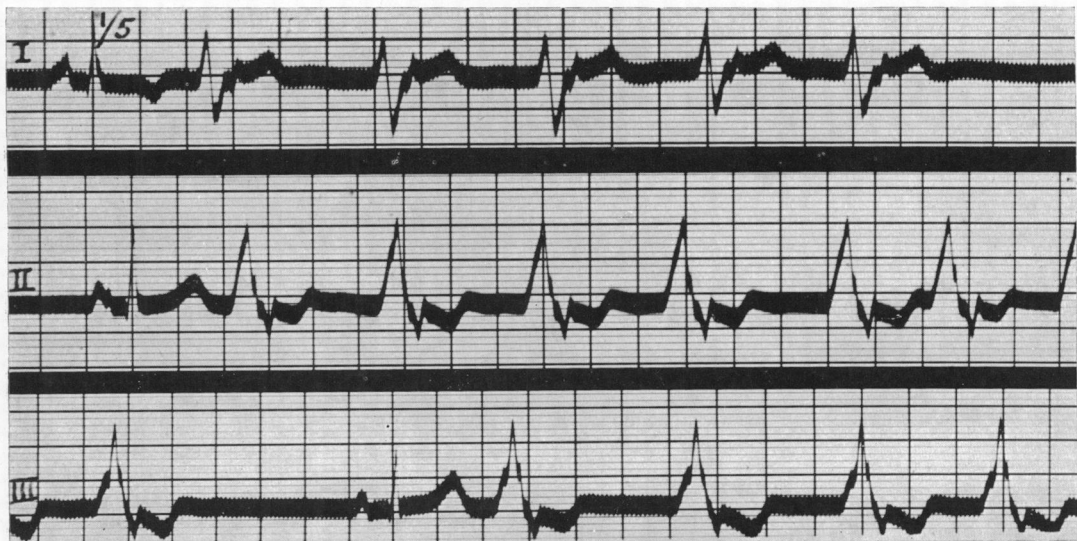


FIG. 15.—R.V.P.T.  Case 40.  Rate 80–150: irregular.  Retrograde auricular conduction.  T I inversion of sinus beat.

view of the frequency with which T inversion during normal rhythm following prolonged paroxysmal tachycardia has been reported.

## THE NATURE OF THE CONDITION

Although the single beats of ordinary paroxysmal tachycardia closely resemble those of extrasystoles, the relation between the two is not as close as it may seem. Thus, extrasystoles are the most frequent of arrhythmias while paroxysmal tachycardia is uncommon. Further, the majority of patients with ordinary paroxysmal tachycardia do not show extrasystoles in their normal rhythm. Campbell and Elliott (1939) found them in only 16 cases of their 42 cases with recorded paroxysms, and then they were often of a type different from the beats constituting the paroxysm. Isolated ventricular extrasystoles were found between attacks both of supraventricular and ventricular tachycardias.

In repetitive paroxysmal tachycardia short spells of extrasystoles, varied in form and irregular in rhythm, often recur constituting tachycardiac runs. These, however, are not the only constituents in repetitive paroxysmal tachycardia; even more often there are typical short paroxysms of tachycardia with all the beats similar in form and exactly like ordinary paroxysmal tachycardia except in their brevity and constant recurrence. The rhythm is mostly regular, though during brief and low-rate paroxysms arrhythmia (? sinus) may be found (cf. " nomotopic tachycardia," Maddox, 1937). Many years after the repetitive paroxysmal tachycardia has ceased, frequent single extrasystoles are still often to be found. As we see it, repetitive paroxysmal tachycardia provides a connecting link between extra-systoles and ordinary paroxysmal tachycardia, because it is composed both of runs and salvos of extrasystoles and of short ordinary paroxysms. Lewis (1909) expressed the same view about his relevant case of paroxysmal tachycardia already cited, and Katz (1946) illustrates the common genesis of extrasystoles and paroxysmal tachycardia in published records (Fig. 385, 386, 391, 395, and 396). Similar links can be established between auricular extrasystoles —e.g., repetitive auricular tachycardia—and auricular flutter and fibrillation. In Case 20 short periods of flutter are mixed with the repetitive runs of auricular tachycardia; in Case 28 auricular paroxysmal tachycardia was recorded at a time when the patient exhibited repetitive impure flutter, and the reverse occurred in Case 8 with repetitive auricular tachycardia and 2 : 1 A-V block who once showed pure flutter. Case 14 with repetitive showers of multiple auricular extrasystoles was twice recorded in auricular fibrillation. Repetitive flutter was always introduced by an auricular extrasystole, and so was repetitive auricular fibrillation in our only case. Repetitive auricular tachycardia illustrates better than any other condition the interdependence of arrhythmias. The relationship between the single groups may at times be so close that electrocardiographic distinction may be impossible—as it often is between auricular paroxysmal tachycardia with 2 : 1 A-V block and flutter (Evans, 1944).

*Ætiology.* Repetitive paroxysmal tachycardia apparently is not due to inflammatory, degenerative, or toxic causes. Though two of our patients gave a history of rheumatic fever or chorea, none had rheumatic heart disease; and the infections preceding the arrhythmia in a few, e.g., diphtheria in Case 31, glandular fever in Case 7, may have been incidental. Only one of the nine with repetitive ventricular tachycardia had hypertensive heart disease, and none of them had angina pectoris in any form. Heavy smokers and drinkers were scarce in our series. An objection to organic heart disease as a cause is that the basic cardiogram between the paroxysms and after an interval of years, with two exceptions always remained normal. In three cases an abnormal thyroid may have been the source of this arrhythmia. Case 32 was found to have a goitre with slight hyperthyroidism when seen five years after she had been under observation for repetitive ventricular paroxysmal tachycardia. Case 15, with the auricular form, had an enlarged thyroid. Case 14 had a slight thyroid enlargement and a fine tremor, and mild hyperthyroidism was diagnosed by us and the next year by another

observer who proposed a subtotal thyroidectomy which was refused: the next year paroxysmal fibrillation appeared in addition to repetitive paroxysmal auricular tachycardia as before.

The relative rarity of paroxysmal tachycardia of any type, its occurrence in infancy, child-hood, and early adult life, its occasional association with congenital heart disease (Case 8), in default of any other convincing cause at least permits us to suggest that it may be due to a congenital peculiarity in the conducting (specific) system of the heart. A recent case of congenital paroxysmal tachycardia diagnosed during fœtal life and confirmed after birth is described by Garvin and Kline (1947). Congenital heart block is already fully accepted. The current opinion about the short P–R bundle branch block condition (the so-called W.P.W. syndrome) is that it depends upon a congenital anomaly in the conducting system, and if so the high incidence of paroxysmal tachycardia in that syndrome points to a common congenital ætiology for both. That is also the view of Fine and Miller (1940). Admittedly the disturb-ance may arise in the nervous control of the heart and possibly from a congenital anomaly there. The relation to emotion or exertion (Cases 19, 30), and in one particular instance to swallowing (Case 28) (Fig. 7), intimates that the mechanism is at least under nervous control.

*Diagnosis.* When first clinically examined a patient was often thought to have auricular fibrillation or flutter, though sometimes the unusual nature of the case was suspected from the pulse. In general the condition cannot be recognized clinically; and the diagnosis rests upon the cardiographic recording of constantly recurring short paroxysms of tachycardia separated from each other by single or several sinus beats, sometimes more. This paroxysmal state continues for weeks or months or even years. In a proportion including some of the ventri-cular variety, there are also numerous short runs or salvos of multiform extrasystoles, irregular in rhythm. Contrary to the opinion of Froment (1932) we think that the cardiogram of such cases (Fig. 11B) in no way differs from that of " grave " or " terminal " ventricular tachy-cardias except that normal sinus beats are also in evidence. Sometimes short extrasystolic runs alone constitute the repetitive brief paroxysms.

Certain allied conditions will be discussed shortly.

(1) *Persistent paroxysmal tachycardia* (or ectopic auricular tachycardia) seems to be inter-minable and every record shows nothing but " paroxysmal tachycardia " for months and years. Such has been described by Weiss and McGuire (1936). Of this condition we have records of two cases, both children, one (P. T.) aged 2½, and the other (R. W.) aged 6 years. In the first the " paroxysm " seems to have persisted for almost three years at a regular rate of 170 and the cardiogram has always shown inverted P I, bifid P II, and diphasic P III; in the second with a similar observation period and rate there was always a diphasic P I with inverted P II and P III. Prolonged observation in hospital was possible in the second child,
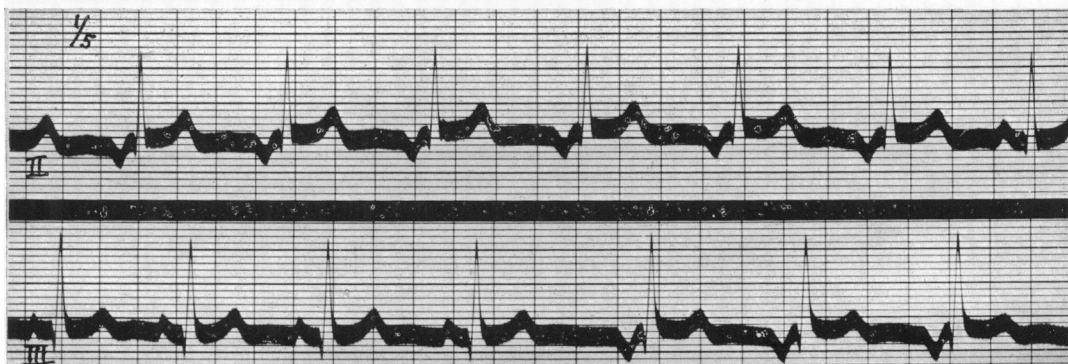


FIG. 16.—" Auricular rhythm." Case G. F. Change in rhythm: inversion of P and slight shortening of P–R interval—without change in rate.

and under digitalis treatment sinus beats and transient sinus rhythm appeared, but the ectopic tachycardia recurred as soon as the effect of the drug wore off.   Herson and Willington (1947) made a similar observation.   In Cases 11, 15, and 33, digitalis broke up the apparently persistent paroxysms into repetitive paroxysmal runs which persisted after treatment was stopped.   Thus the difference between persistent and repetitive paroxysmal tachycardia, based on the frequent appearance of sinus beats in the latter, is not great because persistent paroxysmal tachycardia under hospital observation and digitalis treatment can often be transformed, at least temporarily, into the repetitive variety.

It is worthy of note that runs of irregular and multiform extrasystoles always seem to be short and do not in nature compose the long attacks that characterize ordinary paroxysmal tachycardia as distinct from repetitive paroxysmal tachycardia.

(2) *Auricular rhythm without tachycardia.*   The term auricular rhythm is here used in contrast with sinus rhythm, and it is comparable with nodal rhythm.   It comprises instances in which the P of the sinus beat suddenly changes to an abnormal P without any increase in rate.   This condition, described by Scherf and Harris (1946) as " coronary sinus rhythm," was observed in three cases, not included in our series for there was no tachycardia during the abnormal rhythm marked by the sudden inversion of P I and P II (Fig. 16).   The close relationship of auricular rhythm to repetitive paroxysmal tachycardia is shown by our Case 12 (vide Fig. 1 (C) in Barker *et al.* 1943), and the one of Maddox (1937); in both of them an ectopic P identical with the one during paroxysms continued when the paroxysms were over and the rate was slow.   Normal sinus rhythm was not seen.

(3) *Sinus arrhythmia.*   The short and low-rate irregular paroxysms of repetitive auricular paroxysmal tachycardia may be mistaken for sinus arrhythmia.   Distinctive features are the ectopic P waves of all but the first complexes of the runs, and the pause that follows the runs (Fig. 1).

<center>COURSE, TREATMENT, AND PROGNOSIS</center>

The case notes (see appendix) will illustrate the course of repetitive paroxysmal tachycardia. Children often grow out of it at the time of adolescence.   The state of paroxysms subsided in Cases 1 and 13 at the age of 14 and 15 years, and Case 11, observed in a state of paroxysms for three months when 11, was found in normal rhythm four years later.   Case 5 forms an exception; observed for sinus tachycardia since the age of 11, he had repetitive auricular paroxysmal tachycardia when 15 and still had it when last seen at the age of 18.   Among 14 adults who could be followed up, six (Cases 9, 14, 17, 18, 20, and 33) had repetitive paroxysmal tachycardia one, four, and five years after it was first detected; while eight had either a normal cardiogram when re-examined or had stated they were free from palpitation when questioned by letter.   Among them were Cases 32, 35, 36, and 38 with repetitive ventricular tachycardia in whom the state of paroxysms was known to have lasted from $2\frac{1}{2}$ years (Case 32) to 4 months (Case 35).   The state of paroxysms in most patients did not cease abruptly, but the paroxysms gradually became less frequent, then occurred at long intervals, eventually perhaps disappearing altogether.

The disability arising from repetitive paroxysmal tachycardia differs according to the patient, and the rate and duration of the paroxysms.   Children tolerate almost continuous tachycardia at a rate of 160–180 surprisingly well, being generally symptomless; and so do adults if not subjected to excessive physical strain.   Case 15 sustained successfully the operation of bilateral salpingo-oöphorectomy complicated by postoperative bronchopneumonia, while in the paroxysmal state with a pulse rate of 120–160.

The course and duration of repetitive paroxysmal tachycardia cannot be much modified by treatment, though the paroxysms disappeared in Case 6 after two months' quinidine.   An almost continuous ventricular tachycardia in Case 33 and a continuous auricular tachycardia in Cases 11 and 15 were transformed into a repetitive one by digitalis.   Unsuccessful attempts

with both digitalis and quinidine were far more numerous (Cases 1, 3, 4, 13, 7, and 12).   Nor had vagal compression and deep breathing any effect when tried during prolonged paroxysms.

The prognosis in general is certainly better than one might at first suppose.   Though therapeutic attempts usually fail there is good reason to expect that in time the state of recurrent tachycardia will pass.   Meanwhile most patients are able to live an almost normal life provided they avoid great physical activity.   Our series of prolonged observations includes only three cases where heart disease was already evident; of these, two were little affected but one of them (Case 37) was mortally aggravated by repetitive tachycardia—ventricular in this case.

## SUMMARY

The name " repetitive " is here applied to a special variety of paroxysmal tachycardia in which short paroxysms separated by sinus beats constantly recur over months or years.

Forty new cases of repetitive paroxysmal tachycardia have been collected, nineteen of them with prolonged observation periods ranging from two months to eighteen years, and their clinical and electrocardiographic features were studied.   Ages extended from 4 to 75 years, with two-thirds of the patients (including seven children) under 40.   Males were affected twice as often as females.

The usual symptom was palpitation, and four patients with the ventricular form had syncopal attacks in addition.   With few exceptions the disability caused by the recurrent paroxysms was surprisingly slight.   Fourteen patients—seven children and seven adults—had no symptoms, and all but one of these had repetitive auricular tachycardia.

Of the forty cases only three had evidence of organic heart disease.   One case with the ventricular form had hypertension and cardiac enlargement, another had cardiac enlargement of unknown origin, the other with the auricular form had congenital heart disease.   Toxic goitre was found in two with the auricular form and in one with the ventricular form.   Two gave a rheumatic history but had no rheumatic heart disease.

An irregular pulse was the only clinical sign.   The diagnosis was based on finding in a patient at numerous consecutive examinations a particular kind of electrocardiogram.   This showed on almost every occasion, short paroxysms of tachycardia composed of several beats or more, and separated by normal sinus beats.   The electrocardiogram in its rhythm was divided as follows.

(a) Repetitive auricular paroxysmal tachycardia, 24 cases.   In 16 there were short runs of the ordinary pattern; in 5 cases there were in addition salvos of multiform auricular extrasystoles which in 3 of these formed alone the brief irregular paroxysms.   The rate was lower than in ordinary paroxysmal tachycardia, being on the average 150 in children and 130 in adults.

(b) Repetitive auricular flutter, 5 cases.   This differed from ordinary flutter in that the auricular rhythm was irregular (impure flutter).   One here included was probably fibrillation.

(c) Repetitive nodal paroxysmal tachycardia, 2 cases.

(d) Repetitive ventricular paroxysmal tachycardia, 9 cases, in brief, repeated paroxysms, identical with ordinary ventricular tachycardia (including the grave variety) in respect of the electrocardiogram, except that the sinus beats between paroxysms were physiological, giving no evidence of organic heart disease.

Repetitive paroxysmal tachycardia provides a connecting link between extrasystole and paroxysmal tachycardia because it may be composed both of runs of extrasystoles and of short ordinary paroxysms.   The extrasystolic type is irregular and the paroxysmal tachycardia type is more often irregular than is ordinary paroxysmal tachycardia.   The ætiology of repetitive paroxysmal tachycardia is unknown though something can be said for the view that it arises from a congenital anomaly of the conducting system.

The paroxysmal state is uncertain in its duration, but it often subsides.   In children it

may last until adolescence; in adults it may last for years and then disappear as it did in 8 out of 14 cases long observed. The prognosis as regards disability and length of life is good both in the auricular and the ventricular form; naturally there are exceptions. Quinidine may be tried especially in the ventricular form, but the results are meagre; digitalis is also disappointing; neither is likely to terminate the state of paroxysms. Repetitive paroxysmal tachycardia may be regarded as a distinctive disorder of rhythm rather than a cardiac disease of consequence.

## ILLUSTRATIVE CASES

### List of Abbreviations

A.Ex.        = auricular extrasystole(s).
A.P.T.       = auricular paroxysmal tachycardia.
B.B.Bl.      = bundle branch block.
B.P.         = blood pressure.
EC.          = electrocardiogram(s).
N.R.         = normal rhythm (sinus rhythm).
R.A.P.T.     = repetitive auricular paroxysmal tachycardia.
R.V.P.T.     = repetitive ventricular paroxysmal tachycardia.
V.Ex.        = ventricular extrasystole(s).
V.T.         = ventricular tachycardia.

*Case 1, aged 6.*
   *Repetitive tachycardia in a boy aged 6, almost continuously present for ten years without symptoms or signs other than a rapid irregular pulse. Electrocardiogram (EC.) almost always showed runs of regular auricular tachycardia interspersed with sinus beats; also groups of multiform auricular extrasystoles. Digitalis and quinidine unsuccessful. Normal school life, then gradual and spontaneous disappearance of the arrhythmia after the age of 16. Well and at work, free from paroxysms at the age of 27 (21 years' observation).*
   *History.* He was a twin and weighed only 4¼ lb. at birth. He had always been fit until the last few weeks when his mother noticed that the heart was beating fast, and his father, a doctor, found the pulse to be uncountable. The child did not complain and had always been particularly active.
   *Examination* (1925). Well-developed, of good colour, not distressed. No abnormal signs except for irregular tachycardia varying from 120–180. Heart sounds clear; no cardiac enlargement on radioscopy. EC. (Fig. 2): short runs of A.P.T. and multiple A.Ex. interspersed with occasional normal beats.
   *Course.* From 1925 till 1943 he was examined at frequent intervals; he was also under close observation during the first five years by his father, who for one year charted the pulse three times a day. The child had hardly a day without paroxysms or very frequent extrasystoles, so that it was uncommon to find the pulse completely regular; neither absolute rest in bed nor moderate exertion made any difference, and during sleep at times the pulse was found as irregular. Quinidine had no effect when given for two months, nor had digitalis when given for three weeks, both in moderate doses. Climatic change was without effect, for the pulse was as irregular in the Highlands as it was at the seaside. Intercurrent diseases left the paroxysms unaffected: in 1926 during a period of acidosis with renal glycosuria, pyrexia, and vomiting, the paroxysms were as before. From 1929 onwards he lived an almost normal life for a schoolboy of his age. He took part in light games, drill, and physical exercises, first at public school, later at the university, avoiding only hard games and competitive sports. In 1932, when 13, the paroxysms became less frequent, and when seen in 1933 there were no extrasystoles or paroxysms. He still had occasional palpitation, and in 1935 runs of paroxysmal tachycardia were again recorded. The EC. was normal during the last two visits in 1939 and 1943 when he was working as a curate, and only occasionally had rapid and irregular heart action. EC. were recorded first at monthly, later at 3–6 monthly periods till 1932, once in 1933, twice in 1935, and again in 1939 and 1943. Outside the paroxysms they are remarkable for the constant pattern all

X

through the 18 years of recorded observation and except for Q III are normal. During the paroxysmal attacks either runs of multiform A.Ex., mostly 3–5 in number, follow each other at irregular intervals at a rate of 160–180, or longer stretches of regular A.P.T. composed of the same elements persist at a rate of 140–150. These two features can be found side by side on the same record (Fig. 2). Once a short paroxysm of nodal tachycardia was recorded. In January 1947 he was reported to be well and at work, and he had no recurrence of palpitation; this was at the end of 21 years' observation.

### Case 2, aged 7.

*Arrhythmia found on routine examination in a boy of 7, constantly present till the age of 12. No complaints and no organic heart disease. EC. almost always showed repetitive auricular paroxysmal tachycardia with 2 : 1 A-V block and an auricular rate of 300. After 5 years, spontaneous reappearance and persistence of sinus rhythm with P–R interval of 0·24 and right bundle branch block in the EC.*

*History.* Tonsils and adenoids removed 8 months ago. No complaints, energetic, not short of breath.

*Examination* (October 1930). Short systolic murmur at the apex. No cardiac enlargement. Pulse completely irregular, at first regarded as extreme sinus arrhythmia. EC. short and long runs of A.P.T. interspersed with periods of normal rhythm. During A.P.T. the ventricular complex is modified and resembles that of right bundle branch block; closer inspection shows the deep S in lead II to be due to an inverted P and thus during paroxysm 2 : 1 A-V block persists with an auricular rate of approx. 300 and a ventricular rate of 150.

*Course.* Under observation (Dr. Maurice Campbell) in Guy's Hospital in November and December 1930, because of otitis media. EC. were always the same, a basic rate of 90–110 suddenly interrupted by a run of A.P.T. at a ventricular rate of 140–170. Effort and deep breathing had no effect on the paroxysms. Seen at frequent intervals from 1930–5; transient sinus rhythm was recorded only twice, the next record on the same day again showed A.P.T. In 1936 sinus rhythm seemed to be re-established at a rate of 75; P–R of 0·22 sec. and right bundle branch block (deep S type) persisted in the EC. He felt well and did not complain.

### Case 3, aged 22.

*Young woman complaining of attacks of faintness, actual faints, and palpitation for five years. Normal heart except for irregular tachycardia. EC. showed repetitive auricular paroxysmal tachycardia. During five years of observation only twice was transient sinus rhythm recorded. All treatment failed.*

*History.* Five years ago " fainting " attacks rarely producing unconsciousness; in hospital for 3 weeks. For 3 years frequent recurrence of faint feelings accompanied by palpitation; also slightly breathless on exertion.

*Examination* (March 1931). Varying, irregular pulse, 80–110. B.P. 125/70. Clear heart sounds; radioscopy normal. EC. sudden runs of slightly irregular A.P.T. at a rate of 130–140, preceded by a normal beat and followed by a pause. P II and P III inverted.

*Course.* Heart Hospital in-patient from March till July 1931. During this period the pulse was hardly ever found to be regular, though the rate occasionally dropped to 70. Usual rate at rest 120–140. Quinidine, quinine, digitalis, and atropine had no effect on the rhythm. EC. were almost always of R.A.P.T.; when the paroxysms were short the rate was irregular because of slowing towards the end, when they were long it was regular, 130. Normal rhythm was recorded only three times, at a rate of 80–100. Seen again in February 1932 because of fainting in the street while having palpitation; EC. was found unchanged except for a prolongation of the P–R interval to 0·22 sec. towards the end of the paroxysms. Under observation as out-patient from April 1935 till January 1936 still complaining of giddiness and faintings; heart and B.P. were again found normal, and EC. showed R.A.P.T. on every occasion.

### Case 4, aged 20.

*Man, aged 20, discharged from the Army for D.A.H. complaining of palpitation for many years. Normal heart except for arrhythmia, rate 120. EC. showed repetitive runs of auricular paroxysmal tachycardia. During 5 years of observation not once seen in sinus rhythm. Quinidine ineffective.*

*History.* Discharged from the Army in 1918 because of disordered action of the heart. Complains of continuous palpitation, breathlessness, and slight cough, but at work.

*Examination* (April 1920). Pulse 130, regular. Heart sounds normal. No cardiac enlargement. EC., A.P.T., 132, regular, P II and P III inverted.

*Course.* Observed as an out-patient till February 1925. EC. taken first at monthly then at three-monthly intervals always showed either continuous, regular A.P.T. at a rate of 120–130, or short runs of the same pattern preceded by a normal beat and followed by a pause of 1·2 to 1·4 sec. Occasional normal beats were followed by a blocked A.Ex.; at one time single V.Ex. were seen. Quinidine was ineffective.

## Case 6, aged 30.

*Woman, aged 30, complaining of palpitation. No heart disease. EC. during 9 months' observation always showed repetitive runs of auricular paroxysmal tachycardia and groups of multiform auricular extrasystoles. Disappearance of paroxysms under quinidine.*

*History.* Frequent attacks of sudden palpitation.

*Examination* (December 1938). No heart disease. Radioscopy normal. EC. runs of nodal and auricular Ex., some with aberrant ventricular complex; at times periods of irregular ventricular activity at a rate of 170 with no visible P waves (? fibrillation). Paroxysm often begins with a nodal beat and is always followed by a pause (Fig. 4).

*Course.* Observed as an out-patient till August 1939 at monthly intervals, from then till June 1942 at six-monthly intervals. EC. always similar, with shorter or longer runs of Ex. and A.P.T. till May 1939. Quinidine was tried and when seen in July she had only single A.Ex.; quinidine 3 grains t.d.s. continued for three months; from August 1939 onwards, she was only seen in sinus rhythm, though still having occasional palpitation.

## Case 8, aged 11.

*Boy of 11 with congenital heart disease, probably atrial septal defect, and a rapid irregular pulse. EC. repetitive auricular paroxysmal tachycardia with 2 : 1 A-V block, auricular rate 200. Persistence during 2½ years of observation; once in flutter, never in sinus rhythm. Quinidine reduced auricular rate, otherwise ineffective.*

*History.* Delicate from birth; as a baby had blue lips, and as a child was unable to play games because of shortness of breath. When aged 5, school doctor sent him to special school because of heart disease.

*Examination* (February 1924). High colour and slight cyanosis of lips; no dyspnœa. Pulse 100, very irregular, with frequent long pauses. Soft systolic murmur loudest at the pulmonary area and over 3rd and 4th left spaces; no thrill. Pulmonary second sound heard. Radioscopy: prominence and increased pulsation of pulmonary artery with large hilar branches, right ventricle enlarged. EC. runs of·A.P.T. with P II and P III inversion; 2 : 1 A-V block. Auricular rate 180–220 irregular, ventricular rate half. Very occasional aberrant beats.

*Diagnosis.* Congenital heart disease, probably atrial septal defect.

*Course.* Out-patient (National Heart Hospital) from February till December 1924, seen at frequent intervals; EC. always the same. In-patient from January 31st till March 14th, 1925. During this period of close observation not once was sinus rhythm recorded and the paroxysmal state persisted all the time. The auricular rate was usually 190 with 2 : 1 block; only occasionally were higher grades of block recorded. The short paroxysms usually ended with a pause of 1·2 sec. or less, succeeded by a sinus beat and then a new run. The P–R interval of the sinus and paroxysmal beats was identical and slightly prolonged to 0·22 sec. Once a paroxysm of flutter was recorded, the auricular rate being 308, the ventricular half this and quite regular.

*Vagal pressure* had no effect on the EC. *Exercise*, paroxysms became shorter, otherwise no effect. Atropine, 1/100 of a grain subcutaneously, increased average auricular rate from 190 to 220. Quinidine reduced the auricular rate of 105 and abolished 2 : 1 block, but only temporarily.

From January till October 1926 seen frequently as an out-patient; the same abnormal rhythm was recorded on every occasion.

## Case 14, aged 59.

*Man, aged 59 complaining of irregular heart beating. Pulse irregular; thyroid enlarged with mild toxic signs; no cardiac enlargement. During 3½ years of observation EC. almost constantly showed runs*

*of multiform auricular extrasystoles at a rate of* 140–160 *and irregular. Sinus rhythm was exceptional; transient auricular fibrillation twice recorded. Digitalis ineffective.*

*History.* B.P. found raised 10 years ago when for the first time felt " fluttering and quivering " in the chest at night. For three months missed beats, coupling, or else a completely irregular heart. Feels fit and walks any distance.

*Examination* (October 1942). Pulse irregular, 70–80. B.P. 160/95. Normal heart sounds; no cardiac enlargement. Isthmus of thyroid gland easily seen; fine tremor of hands; toxic goitre suspected. EC. (Fig. 3) runs of A.Ex. (8–10) of different origin at an irregular rate 140–160, followed by pause. Basic rate, 66 regular.

*Course.* June 1942 observed at Liverpool Royal Infirmary (Dr. Wallace-Jones). EC. invariably the same. B.M.R.+27 per cent. Iodine treatment begun; reported August 1943, less palpitation under iodine, but EC. unchanged. Subtotal thyroidectomy considered but not performed. From May 5th till June 9th, 1944, in Manchester Royal Infirmary (Prof. Crighton Bramwell); diagnosis of toxic goitre doubted. B.M.R.+5 per cent. Among 28 EC. there recorded, 23 show runs of multiform A.Ex. sometimes interspersed with sinus beats, 3 sinus rhythm, and 2 auricular fibrillation. This last appeared under digitalis which was ineffective in reducing the paroxysms but increased the number of blocked A.Ex.

Seen again in July 1944, February and September 1945, and March 1946; paroxysms recorded on each occasion.

*Case 18, aged 18.*

*Healthy and active man of* 18, *no complaints. Referred by Medical Board because of irregular pulse. This found at the age of* 14 *on routine examination. EC. then as now, showed repetitive auricular paroxysmal tachycardia with Wenckebach periods.*

*History.* Played games at school. On admission as naval cadet, aged 14, doctor noticed irregular pulse. EC. (inspected) then showed short runs of A.P.T. similar to the one described below. Completed full physical training of cadet, and never complained of palpitation. Referred by Medical Board because of irregular pulse.

*Examination* (February 1947). Pulse very irregular with series of rapid beats then pause; persistent, no change on exertion. B.P. 130/80. Normal heart sounds. X-rays, no enlargement EC. continuous short runs (3–7 beats) of A.P.T. at a slightly irregular rate of 120–130 with gradual increase of P–R interval from 0·18 to 0·24 (Fig. 5A). The first two P waves of the paroxysms appear normal, and are small and upright; then P changes, often gradually, to flat-diphasic (lead II). The paroxysm sometimes ended with a blocked P wave, always followed by a pause of varied length.

*Case 25, aged 34.*

*Woman, aged 34, complaining of palpitation. Irregular pulse found with short paroxysms of tachycardia; otherwise normal heart. Three years later EC. showed repetitive flutter.*

*History.* Sudden attacks of rapid heart-beating for a few seconds, minutes, or hours. More frequent when lying, occasionally a dozen a day. Referred by a doctor because of extrasystoles.

*Examination* (October 1941). Pulse 120 with occasional irregularities. At times 5–6 beats in paroxysms at a rate of 160–180. Otherwise no cardiac abnormality. No EC. then recorded.

In December 1944 she had the same complaints. EC. basic rate 90–100, regular, with tall P II. Sudden bursts of slightly irregular, apparently independent ventricular activity of 200, lasting for 7–12 auricular cycles, after which the normal rhythm resumes without intervening pause. $CR_1$: typical flutter waves (Fig. 8).

*Case 30, aged 22.*

*Woman of* 22 *had irregular heart when aged* 6; *normal adolescence. Palpitation on effort. EC. during two years of observation always showed nodal tachycardia on exertion, nodal rhythm or sinus rhythm at rest. No other signs of heart disease. Spontaneous improvement with little acceleration on exertion, but persistence of abnormal rhythm.*

*History.* When aged 6, heart was found irregular: she was allowed to play games only after the age of 13. For 2–3 months, palpitation and increasing dyspnœa on exertion.

*Examination* (July 1944). On standing, pulse 120, regular; after ½–1 minutes irregular, then 76, regular. Exercise increases rate to 130, persisting for several minutes. No other abnormal signs; radioscopy normal. EC. nodal tachycardia.

*Course.* EC. were recorded first at fortnightly, then at monthly, later at three-monthly intervals from July 1944 till June 1946. At rest: nodal rhythm, no P waves visible, rate 60–70, regular. Sinus rhythm found only once. On exertion: nodal tachycardia, 130–140, regular; when rate drops few sinus beats appear. At the last examination there was sinus rhythm in lead I, nodal rhythm in leads II and III while at rest, nodal rhythm at a rate of 96 on exertion. She was then better and able to exert herself without much palpitation or dyspnœa.

*Case 32, aged 35.*

Woman, aged 35, *complaining of sudden faintness and palpitation. Pulse rapid, irregular; otherwise normal heart. EC. during* 2½ *years of observation constantly showed repetitive ventricular tachycardia and multiform extrasystoles. Quinidine ineffective. Ten years later mild toxic goitre, yet no recurrence of paroxysms.*

*History.* Well until one year ago when she collapsed in the street with sudden palpitation, weakness, and dizziness; many similar attacks since.

*Examination* (April 1921). Except for irregular pulse nothing abnormal. No cardiac enlargement on radioscopy. EC., R.V.P.T., rate 220 reg.; aur. rate 150, reg. Shorter runs of 2–3 V.Ex. Intervening sinus beats at a rate of 110 (Fig. 11A and B).

*Course.* EC. recorded twice in February 1922, June 1922, October 1923, always showed R.V.P.T. at a rate of 200–250. Quinidine at first reduced the number of attacks, but was later ineffective. Seen again January 1931, still complained of palpitation. Examination then showed small toxic goitre, with a pulse rate of 96. B.P. 160/90, slight tremor; normal heart on physical and radiological examination. EC. showed N.R. except for single ventricular extrasystoles. She improved with rest and sedatives and the signs of toxic goitre subsided. When last seen in 1934, she felt well; single V.Ex. were still present in the EC.

*Case 33, aged 20.*

Man *of* 20, *served as air pilot during the war; discharged from R.A.F. because of heart disease. Complains of palpitations and fainting attacks. Normal heart except for tachycardia. Three EC. during* 10 *months of observation as an out-patient showed regular ventricular tachycardia, rate* 120. *While in hospital and on digitalis, transient sinus rhythm appeared, followed by repetitive ventricular tachycardia, always present during the remaining* 14 *months of observation.*

*History.* Two years ago when volunteering for the R.A.F. pulse was found " queer." Examined several times before being accepted Grade 1. Trained as a pilot, apt for his duties, flying at times at 20,000 feet without oxygen. Tonsillitis 6 months ago; kept for 3 months in hospital where X-rays and EC. were taken. Discharged from R.A.F. because of " congenital heart disease." Complains of frequent palpitation and dizziness. Fainted four times during the year while on parade.

*Examination* (November 1943). Pulse 110, reg. B.P. 105/80. Heart sounds normal. Radioscopy: full-sized heart within normal limits. EC., V.T. (Right B.B.Bl.), 125 reg., no P waves seen Five days later, EC. similar.

*Course.* Seen again September 1944; having frequent palpitation even at night, usually short attacks 1–2 min. duration. EC., V.T. as before. Under observation at the London Hospital 9th October till 11th November 1944. V.T. persisted while at rest, rate 110–130. Under digitalis leaf 1½ grains t.i.d. pulse became irregular and the rate decreased occasionally to 50 when the pulse became regular and the EC. normal. Digitalis, 1 grain bis die, later t.i.d.; successive EC. always showed V.Ex. singly or in short runs (3–5), at a rate of 120–130, preceded by a sinus beat and followed by pause with a basic rate of 90–96 (Fig. 12). One month later the EC. was the same. In 1946 it was heard by chance that he had committed suicide.

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