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PAROXYSMAL VENTRICULAR TACHYCARDIA

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Paroxysmal ventricular tachycardia is an uncommon disorder of cardiac rhythm, the incidence, according to Campbell (1947), being 4 per cent of all paroxysmal arrhythmias. Since it usually occurs in association with serious organic heart disease, and since there is always the danger of the development of terminal ventricular fibrillation, it is commonly regarded as a serious condition in which early diagnosis and treatment are important.

Lewis (1909) first established electrocardiographic proof of the arrhythmia originally suspected by Mackenzie (1908) on polygraphic records, and the diagnosis of paroxysmal ventricular tachycardia, although it may be suspected clinically, can be made only with certainty after careful analysis of the electrocardiogram. Using strict electrocardiographic criteria a review of 83 cases occurring in a large teaching hospital has been undertaken.

Electrocardiographic Diagnostic Criteria

The diagnostic criteria originally proposed by Robinson and Herrmann (1921) stressed the importance of three features.

(1) The presence of runs of abnormal ventricular complexes varying slightly in contour, due to occasionally superimposed atrial complexes.

(2) The establishment of atrial complexes, occurring independently and usually at a slower rate, rarely retrogradely.

(3) The finding of isolated ventricular complexes, before and after a paroxysm, that have the same form as the ventricular complexes recorded during the paroxysm. The isolated premature contractions must bear the same relation to the normal ventricular complex as do the onset and offset complexes of the tachycardia.

However, the electrocardiographic distinction between supraventricular and ventricular tachycardia is often difficult and indeed not always possible. Abnormal ventricular complexes may be present during a supraventricular tachycardia due to pre-existent bundle-branch block, aberrant ventricular conduction consequent upon the rapid rate or by the availability of an alternative pathway of atrio-ventricular (A-V) conduction as in the pre-excitation phenomenon. Conversely, paroxysmal ventricular tachycardia may be present with ventricular complexes of normal or near normal appearance (Simon and Langendorf, 1944). As shown by Pick and Langendorf (1960), supraventricular as well as ventricular tachycardia may cause complete or incomplete A-V dissociation. Also retrograde activation of the atria may occur from an ectopic impulse arising in the A-V node as well as from the ventricles. Therefore, the presence of abnormal ventricular complexes and the identification of an independent atrial rhythm at a slower rate or in a retrograde fashion cannot by themselves be considered as diagnostic of paroxysmal ventricular tachycardia, and further electrocardiographic clues must be sought. The finding of isolated ventricular complexes before or

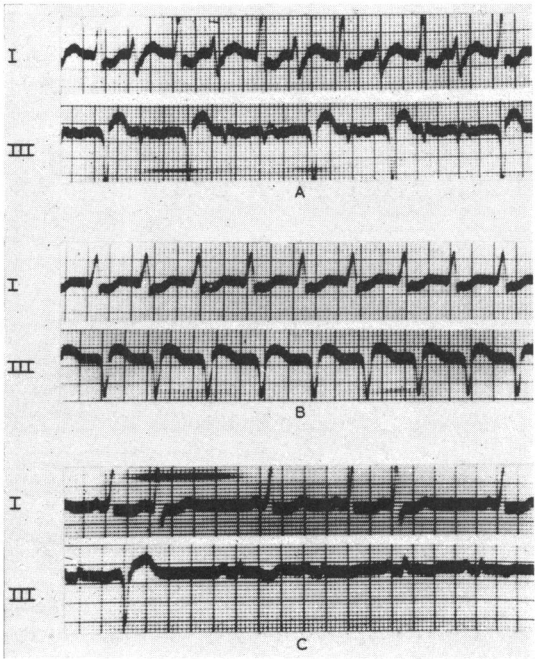

FIG. 1.—The tachycardia present in tracing A shows an alternating type of ventricular complex. No definite P waves can be determined. The diagnosis was considered to be paroxysmal ventricular tachycardia, which was possibly induced by digitalis. Tracing B was taken during treatment with procaine amide and shows the development of stable ventricular complexes from one focus. The ventricular extrasystoles shown in tracing C after conversion to normal rhythm have a similar configuration to those recorded during the paroxysm, and help to substantiate the diagnosis.

after a paroxysm, the third point suggested by Robinson and Herrmann, can be of great help in diagnosis as illustrated in Fig. 1. Perhaps the best evidence suggesting the presence of a paroxysmal ventricular tachycardia is the finding of partial ventricular captures, leading to ventricular fusion beats as shown in Fig. 2. In these circumstances there is competition for ventricular control between a normally conducted sinus impulse and the ventricular ectopic focus itself. Such ventricular fusion beats will obviously only occur when the sinus and ectopic impulses do not share a common pathway of conduction through the A-V nodal tissue and the ectopic impulse must therefore arise below the A-V node.

MATERIAL

The records of the Department of Cardiology, Royal Infirmary, Edinburgh for the years 1920 to 1961 inclusive were examined. There were 83 acceptable cases of paroxysmal ventricular tachycardia, and these form the basis of this report. All case records were available for review. Follow-up records were also available in most instances, and the remainder (with few exceptions) were either personally followed up by one of the authors or by communication with the general practitioner. Six patients originally reported by Ritchie (1926) and five patients by Gilchrist (1926) are included. An arbitrary division of the initial episode of paroxysmal ventricular tachycardia in each patient was made into a persistent and an intermittent variety. The

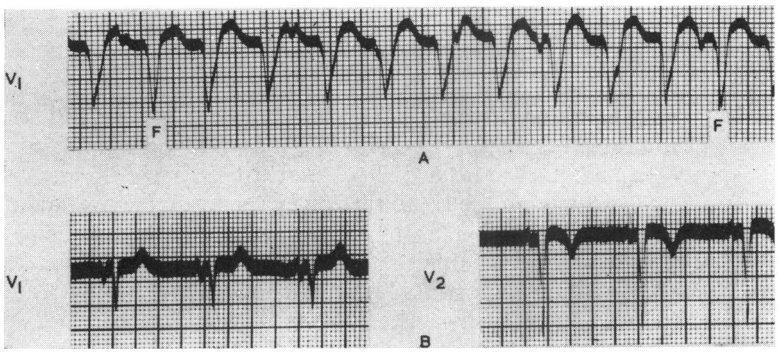

FIG. 2.—Paroxysmal ventricular tachycardia. The ventricular origin of the paroxysm is substantiated by (1) bizarre widened QRS complexes (rate 158/min.); (2) the differentiation of an independent atrial rhythm at a slower rate of 107/min.; (3) probably the best evidence is that of the identification of partial ventricular capture beats (fusion beats, F), where the QRS complexes are less broad and less bizarre, and approach the configuration of the QRS complex during normal A-V conduction as shown in the lower tracing, taken after the paroxysm. (See text.)

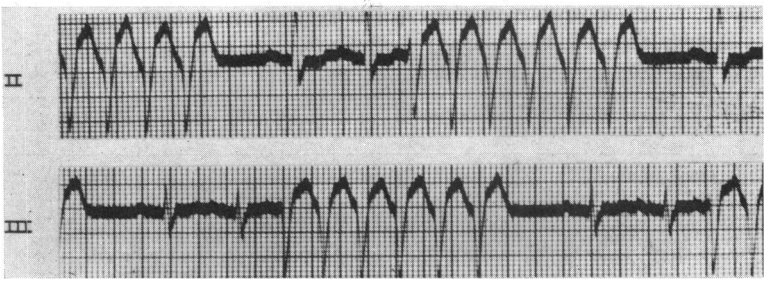

FIG. 3.—Intermittent ventricular tachycardia. Although P waves cannot be identified, the stability of the coupling and configuration of the QRS complexes are points in favour of the ventricular origin of the paroxysms.

tachycardia was classified as persistent if the arrhythmia was consistently present throughout the cardiogram, and intermittent if the tracing presented short bouts of five or more ventricular ectopic beats interspersed with runs of sinus rhythm as shown in Fig. 3. It was not unusual for patients to have further bouts of tachycardia during the following few days after successful conversion to normal rhythm. For the purpose of discussion these repeated episodes during the acute illness and occurring over a few days were not regarded as recurrent episodes. Recurrent attacks are defined as further episodes that occur following discharge from the initial hospital admission. There were 110 episodes in these 83 patients available for review.

RESULTS

The age distribution in decades of these 83 patients is shown in Fig. 4: the peak incidence is between the fifth and seventh decades. There were 56 male and 27 female patients whose ages ranged from 14 to 84 years. The initial episode of tachycardia was persistent in 45 and intermittent in 38, and the ventricular rate during the episodes varied from 136 to 220 per minute, the duration of attacks varying from a few seconds to 18 days in one instance. The type of underlying heart disease present is shown in Table I.

Ischæmic Heart Disease. Ischæmic heart disease was the underlying disorder in 60 patients (72%).

Acute Myocardial Infarction. The occurrence of paroxysmal ventricular tachycardia was associated with an acute infarct in 31 patients (37%). The diagnosis of infarction was based on either post-mortem or unequivocal electrocardiographic evidence of recent transmural myocardial damage. There were 24 men and 7 women of whom the average age was 64 years (range 32–84 years). The tachycardia was persistent in 17 and intermittent in 14. The time of onset of paroxysmal ventricular tachycardia relative to the occurrence of infarction is shown in Fig. 5. Of the 31 episodes 15 occurred on the same day as the infarct and, with the exception of 4 patients, all occurred within 12 days of infarction. Two episodes occurred much later (23 and 42 days after the initial acute infarct), but

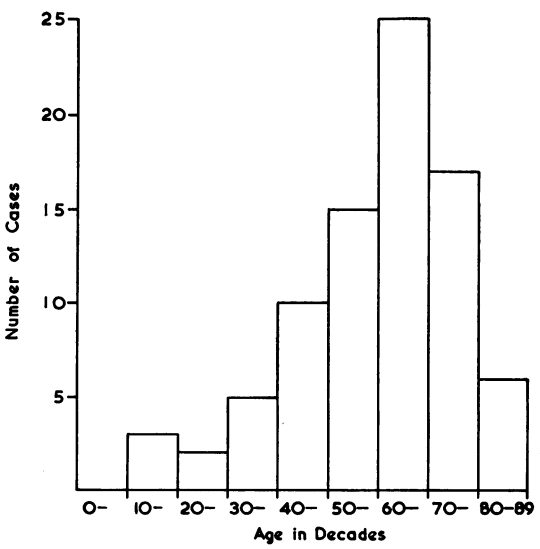

FIG. 4.—The age distribution in decades of 83 patients (56 male and 27 female) with ventricular tachycardia.

TABLE I

TYPES OF UNDERLYING HEART DISEASE

Underlying heart disease	No. of cases	Percentage
Ischæmic heart disease		
Recent acute myocardial infarction	31 } 60	37 } 72
Other evidence of ischæmic heart disease	29 }	35 }
Rheumatic heart disease	9	11
Miscellaneous		
Hypertension and congestive failure	5	
Myocarditis	2	
Secondary neoplasm	1	12
Thyrotoxicosis	1	
Pulmonary stenosis	1	
No heart disease	4	5

in both instances the patients had suffered further pain and fresh infarction proven electrocardiographically immediately before the onset of the tachycardia. The development in a third case, occurring 25 days after infarction, was undoubtedly induced by excessive digitalis therapy, and the fourth, occurring 30 days after infarction, was also possibly induced by digitalis. The site of myocardial infarction was anterior in 9, antero-septal in 9, posterior in 11, and both posterior and antero-septal in 2. The duration of tachycardia varied from a few hours up to seven days in one instance. There were 35 episodes in these 31 patients.

Ischæmic Heart Disease Without Acute Myocardial Infarction. There was evidence of underlying ischæmic heart disease, but without recent acute myocardial infarction in 29 patients (35%). This evidence was based on a history of preceding angina or old myocardial infarction or post-mortem evidence of old infarction. There were 23 men and 6 women, whose average age was 64 years (range 40–80 years). The tachycardia was persistent in 17 and intermittent in 12, and its duration varied from a few minutes up to 18 days. There were 30 episodes in these 29 patients.

Rheumatic Heart Disease. Rheumatic heart disease was the underlying heart disorder in 9 patients (11%). There were 4 men and 5 women, with an average age of 43 years (range 24–67). The tachycardia was persistent in 5 and intermittent in 4, its duration varying from hours up to 4 days. There were 21 episodes in these 9 patients.

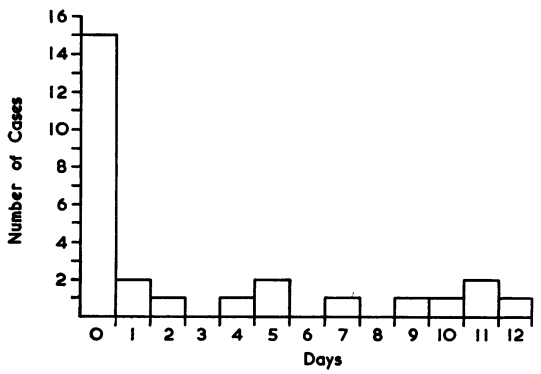

FIG. 5.—Onset in days of paroxysmal ventricular tachycardia after acute myocardial infarction. The onset of tachycardia was delayed beyond the twelfth day in 4 additional patients as described in the text.

No Evidence of Heart Disease. There was no clinical, electrocardiographic, or radiological evidence of underlying heart disease in 4 patients, 3 men and 1 woman (5%), whose ages were 18, 19, 41, and 43 years. The tachycardia was persistent in 2 and intermittent in 2 cases, and the duration varied from a few hours up to 24 hours.

The Ætiological Role of Digitalis. Digitalis is well known as a precipitating cause of paroxysmal ventricular tachycardia (Gilson and Schemm, 1950). The possible ætiological role of the drug is summarized in Table II. The criteria used for assessing the importance

TABLE II

THE ÆTIOLOGICAL ROLE OF DIGITALIS

	No. of cases	Percentage
Induced by digitalis	18	22
Possibly induced by digitalis ..	9	11
Digitalis dosage not excessive ..	15	18
Total number receiving digitalis ..	42	51
Total number without digitalis ..	41	49

of digitalis as a precipitating factor were as laid down by Lown, Marcus, and Levine (1959). At the time of onset of the tachycardia 18 (22%) had evidence of digitalis intoxication: in 8 of these the onset of the arrhythmia followed the administration of intravenous digoxin to patients already fully digitalized and taking an oral maintenance dosage. In a further 9 patients (11%) it may possibly have been induced by excessive digitalis dosage. In a further 15 patients (18%) to whom it had been given the drug was considered to have played no ætiological role. Of the 31 patients with an associated myocardial infarct 8 had received digitalis before the onset of the tachycardia, which was considered to have been induced by the drug in 2, and possibly induced in a further 3 cases. It was considered to have played no part in the ætiology of the remaining 3 cases.

Treatment. The effects of the various therapeutic agents tried are shown in Tables III, IV, and V.

Paroxysmal Ventricular Tachycardia Not Induced by Digitalis (Table III). No treatment was given in 14 episodes, and in 9 of these the arrhythmia settled spontaneously. It was of the intermittent variety in 7, and congestive cardiac failure was present in 1 of these 9 cases undergoing spontaneous remission. Congestive cardiac failure was present before the onset of the tachycardia in 4 of the 5 unsuccessful cases, and the arrhythmia was of the persistent variety in three. All cases spontaneously reverting to normal rhythm did so within 24 hours of the onset of paroxysmal ventricular tachycardia.

Sedation with either phenobarbitone or morphine was successful in 2 out of 3 episodes: congestive cardiac failure was present before the onset of the tachycardia in the unsuccessful case but was not present in either of the cases with a successful outcome.

Oral quinidine was successful in 20 of the 29 episodes in which it was tried. The therapeutic response to intravenous quinidine and procaine amide was identical. Fourteen episodes were treated with a conversion rate of 10 with each drug. In one instance the tachycardia was probably

TABLE III

TREATMENT OF VENTRICULAR TACHYCARDIA—NOT INDUCED BY DIGITALIS

Type of treatment	No. of trials	No. converted
No treatment	14	9
Morphine (intramuscular)	1	1
Sodium phenobarbitone (intramuscular)	2	1
Oral quinidine	29	20
Intravenous quinidine	14	10
Intravenous procaine amide	14	10
Oral procaine amide	1	1
Intravenous+oral procaine amide	1	0
Oral quinidine+intravenous procaine amide	1	1
Hydrocortisone	3	0

TABLE IV

TREATMENT OF DIGITALIS-INDUCED VENTRICULAR TACHYCARDIA

Type of treatment	No. of trials	No. converted
Digitalis continued	10	0
Digitalis stopped	7	7
Digitalis stopped+potassium	2	1
Digitalis stopped+prednisone	1	1
Digitalis stopped+oral quinidine	4	3
Digitalis+oral quinidine	2	1
Digitalis+oral quinidine+intravenous procaine amide	3	3
Totals	29	16

induced by oral quinidine therapy, as first described by Scott (1922), and quinidine was being given in an attempt to convert atrial fibrillation to normal rhythm: the drug was stopped and the paroxysm terminated. In another instance tachycardia developed during the treatment of atrial flutter with both digitalis and quinidine in excessive dosage: both drugs were stopped, and the paroxysm terminated.

Paroxysmal Ventricular Tachycardia Induced by Digitalis (Table IV). In 10 patients digitalis therapy was continued after the onset of tachycardia with uniformly fatal results. In 7, conversion to normal rhythm was achieved simply by discontinuing the administration of digitalis; in 2, oral potassium chloride was given in addition to stopping digitalis: this was successful in one, and in the second the tachycardia had followed the administration of intravenous digoxin to a patient already fully digitalized by the oral route. The patient died within 24 hours with paroxysmal ventricular tachycardia still present. Treatment with oral quinidine in addition to stopping digitalis was successful in 3 out of 4 patients. The administration of digitalis was continued in 5, but combined with either oral quinidine alone or in combination with intravenous procaine amide, and this therapy was successful in 4 patients. Despite this high rate of conversion, there was evidence to suggest that the continuation of digitalis perpetuated the tachycardia in these cases. The longest episode of paroxysmal ventricular tachycardia in this review lasted 18 days: this was a case of coronary insufficiency in which the tachycardia was possibly induced by digitalis, and treatment with oral quinidine and intravenous procaine amide, while continuing digitalis, was eventually successful.

Digitalis Therapy in Paroxysmal Ventricular Tachycardia not Induced by Digitalis (Table V). Oral digitalis was the sole drug given therapeutically in six patients, and none of these had had digitalis previously. It was successful in 3, 2 of whom were of the intermittent variety and without associated congestive cardiac failure. The remaining successful case was of the persistent variety and was in congestive cardiac failure before the onset of the tachycardia. It was present for

TABLE V

DIGITALIS THERAPY IN VENTRICULAR TACHYCARDIA—NOT INDUCED BY DIGITALIS

Type of treatment	No. of trials	No. converted
Digitalis alone	6	3
Digitalis+oral quinidine	4	4
Digitalis+intravenous+oral procaine amide	1	0
Digitalis+intravenous procaine amide+oral quinidine	3	3
	14	10

TABLE VI

MORTALITY IN VENTRICULAR TACHYCARDIA

Mortality	Persistent type (45 cases)		Intermittent type (38 cases)		Total (83 cases)	
	No.	%	No.	%	No.	%
Immediate	13	29	7	18	20	24
One year	29	64	18	47	47	57

one day in 2 and for two days in one. In the 3 unsuccessful trials of this therapy, 2 were of the persistent variety, 1 of which was in congestive cardiac failure. The remaining case was of the intermittent variety without associated cardiac failure. The tachycardia was present for one day in 2 and for three days in one.

Prognosis. The immediate mortality during a paroxysm and at the end of one year is less in the intermittent than the persistent variety of tachycardia (see Table VI). Congestive cardiac failure was present before the onset of the tachycardia in 47 per cent of patients in each group and consequently does not account for the difference in mortality. The relation between the type of underlying heart disease and mortality is shown in Table VII.

Twenty patients (24%) died without apparent conversion of the tachycardia although its actual presence was only proven electrocardiographically at the time of death in 3 patients. The development of terminal ventricular fibrillation was demonstrated in a further 4 patients. The part played by the presence of congestive cardiac failure in the immediate mortality is shown in Table VIII. Of the 20 patients, 16 were in congestive cardiac failure before the onset of the tachycardia and, in one, cardiac failure developed during the paroxysm. There were only 3 patients without congestive cardiac failure in whom the tachycardia was immediately fatal.

Acute Myocardial Infarction (31 patients). Five patients (16%) died during the episode of paroxysmal ventricular tachycardia. The over-all mortality one year after the tachycardia was 55 per cent (17 patients): of the remaining 14 patients, 1 was not available for follow-up; 9 were still alive at varying times after the tachycardia, i.e. 1 after five months, 1 after one year, 3 after two years, 1 after three years, 1 after six years, and 2 after seven years. Four patients had died: two, four, seven, and eight years after the tachycardia. Congestive cardiac failure was present before the onset in 9 patients, and all were dead within one year. Ten patients had had a previous myocardial infarct but without associated paroxysmal ventricular tachycardia. Four patients were known to suffer a further myocardial infarct without recurrence of tachycardia. Only one

TABLE VII

MORTALITY IN 83 CASES OF VENTRICULAR TACHYCARDIA

Mortality	Acute myocardial infarction (31 cases)		Ischæmic heart without acute disease (29 cases)		Rheumatic heart disease (9 cases)		Miscellaneous (10 cases)		Normal hearts (4 cases)	Total (83 cases)	
	No.	%	No.	%	No.	%	No.	%		No.	%
Immediate	5	16	8	28	3	33	4	40	0	20	24
One month	11	36	15	52	5	56	5	50	0	36	43
One year	17	55	17	66	5	56	6	60	0	45	57

TABLE VIII

THE ROLE OF HEART FAILURE IN THE IMMEDIATE MORTALITY OF VENTRICULAR TACHYCARDIA

Underlying heart disease	Cardiac failure before onset	Cardiac failure after onset	No heart failure	Total
Acute myocardial infarction	4	1	0	5
Ischæmic heart disease without acute infarction	6	0	2	8
Rheumatic heart disease	3	0	0	3
Miscellaneous	3	0	1	4
Normal hearts	0	0	0	0
	16	1	3	20

patient suffered from recurrent episodes and he had three further episodes of tachycardia without further infarction until he died suddenly five months after his initial episode.

Paroxysmal Ventricular Tachycardia Associated with First Episode of Myocardial Infarction. There were 21 patients in whom the first episode of infarction was associated with tachycardia. One patient could not be followed up. The mortality rate in this group one month after infarction was 24 per cent (5 patients). The one-year survival rate, starting follow-up one month after infarction, was 73 per cent.

Ischæmic Heart Disease Without Acute Myocardial Infarction. Eight (28%) of the 29 patients died with tachycardia apparently still present. The mortality rate one month after the episode was 52 per cent (15 patients). Congestive cardiac failure was present before the onset in 17, and 14 of these died within one year of the paroxysm. Two were not available for follow-up and the remaining patient was still alive two months after the tachycardia. Of the remaining 12 unassociated with heart failure, 5 died within one month of the episode and a further 3 died at one year, two years, and four years after the initial episode. One was not available for follow-up, and the remaining 3 patients were still alive four months, one year, and nine years after the initial episode. Only one patient suffered recurrent attacks: he had four further episodes of tachycardia in the following year and, at the time of writing, is still alive and well nine years after the initial one.

Rheumatic Heart Disease (9 patients). Of the 9 patients 3 died with paroxysmal ventricular tachycardia apparently still present. A further 2 died within one month of the episode. Of these 5 who died, 4 were in congestive cardiac failure before the onset of tachycardia, and the remaining one developed heart failure during the episode. One patient, whose tachycardia was induced by quinidine therapy, died 18 months after the episode. Another suffered repetitive attacks over a period of four years and finally died suddenly at home: these repetitive episodes were not induced by digitalis. The two remaining patients were not available for follow-up.

Miscellaneous Group. Five patients had hypertensive heart disease and all were in congestive cardiac failure before the onset of tachycardia. The arrhythmia was probably induced by excessive digitalis therapy in each of these.

Myocarditis was the underlying heart disease in two patients and both died during the paroxysm. Necropsy revealed a tuberculous ætiology in one and a necrotizing myocarditis and arteritis in the other. One patient was found at necropsy to have secondary myocardial deposits from an old reticulum cell sarcoma of the tibia. The tumour tissue had invaded the bundle of His, producing complete heart block, and the occurrence of paroxysmal ventricular tachycardia had been induced by digitalis.

A 55-year-old woman with thyrotoxicosis had an intermittent tachycardia of two days' duration, which was treated successfully with oral digoxin, quinidine, and carbimazole. She was still alive three years later on a maintenance dose of carbimazole and had had no further bouts of tachycardia.

A 14-year-old girl with congenital pulmonary stenosis developed paroxysmal ventricular tachy-

cardia during diagnostic cardiac catheterization. The tachycardia was present for a few minutes, and normal sinus rhythm was restored shortly after removing the catheter from the infundibulum of the right ventricle. She was still alive and well, having had no further bouts of tachycardia, five years later. She is included in the series as an example of the well-known liability of these patients to develop paroxysmal ventricular tachycardia during manipulation of a catheter in the right ventricle and also to illustrate the benign nature of the condition, once the catheter is removed.

No Evidence of Heart Disease (4 patients). Of these 4 patients, 2 have previously been reported by Gilchrist (1926). The third was a 43-year-old man when he suffered his first episode in 1951. He had recurrent bouts, often necessitating admission to hospital, with peripheral circulatory collapse during the following seven years. At the time of writing he has been completely symptom free for three years and is leading an active life, eleven years after his first episode of tachycardia.

The fourth patient was a girl with intermittent bouts of paroxysmal ventricular tachycardia since the age of 12 years. Emotional excitement appeared to be a precipitating factor. The tendency to recurrent episodes was largely controlled by prophylactic oral quinidine sulphate for a number of years. This therapy was stopped in 1957, and during the past four years she has had only one brief episode of palpitations. At the time of writing she is symptom free, 18 years after the first episode of tachycardia.

Discussion

The occasional occurrence of paroxysmal ventricular tachycardia in patients with no demonstrable evidence of heart disease is well known. The incidence of 5 per cent in the present review is less than that reported in comparable series. Armbrust and Levine (1950) reporting 107 cases and Herrmann, Park, and Hejtmancik (1959) reporting 60 cases observed an incidence of 12 and 10 per cent respectively. The type of underlying heart disease present and the age distribution by decades is in general agreement in all three series.

Since first described by Vaughan (1918) there have been numerous references to digitalis as a precipitating factor of paroxysmal ventricular tachycardia. Gilchrist (1926) was probably the first to demonstrate repetitive attacks in the same person unequivocally related to digitalis therapy. There were 2 patients in this review who developed repetitive attacks that appeared only upon the administration of digitalis and disappeared when the drug was withdrawn. The apparent ætiological role of digitalis has varied in the published series from a possible 10 per cent (Armbrust and Levine, 1950) to 22 per cent (Williams and Ellis, 1943). The high incidence of 22 per cent in the present series is probably explained by the fact that 8 of these 18 patients had been given intravenous digoxin while already taking oral maintenance digitalis dosage. Levine (1951) considers the observation of paroxysmal ventricular tachycardia with alternating bidirectional complexes to be pathognomonic of digitalis intoxication. There were 4 examples of this in the present series, which were all considered to be induced by digitalis. The importance of recognizing digitalis intoxication as a cause of paroxysmal atrial tachycardia with atrio-ventricular block has recently been re-emphasized (Harris Julian, and Oliver, 1960), and the lethal effects of continuing the drug in such patients pointed out. This is also true for patients with paroxysmal ventricular tachycardia. In 10 patients, in whom digitalis was not recognized as an important ætiological factor, the drug was continued without additional therapy, with uniformly fatal results. In striking contrast, the simple withdrawal of the drug in 7 patients was successful in all. The importance of assessing the possible ætiological role of digitalis will be immediately apparent. Gilson and Schemm (1950), on the other hand, have reported the value of digitalis in the treatment of paroxysmal ventricular tachycardia when the arrhythmia was not induced by the drug. Greenwood and Finkelstein (1961) and Brodwall and Knutsen (1961) have recently re-emphasized the value of this treatment, and Sokolow (1958) has advocated the use of digitalis when congestive cardiac failure arises during a paroxysm. In the present series digitalis was successful in 3 out of the 6 occasions when it was employed as the sole therapeutic measure. However, the tachycardia settled spontaneously in 9 of 14 episodes and simple sedation was successful in 2 out of 3 trials. While digitalis may have a part to play in the treatment of paroxysmal

ventricular tachycardia when not induced by the drug, no such conclusions are possible from our material.

Our results suggest that a high proportion of episodes, particularly of the intermittent variety, and without associated congestive cardiac failure, will revert spontaneously to normal rhythm within 24 hours. There is, however, a natural desire to start treatment due to the possible development of ventricular fibrillation (four proven cases in the present series). Spontaneous reversion is unlikely to occur after 24 hours. The value of oral quinidine in the treatment of paroxysmal ventricular tachycardia was first reported by Scott (1922), and there can be little doubt of its efficacy, since it was successful in 20 out of 29 trials. Intravenous quinidine and procaine amide were each tried on 14 occasions and were successful in 10. It should be pointed out that quinidine was always given to the point of toxicity before being considered a therapeutic failure. On the other hand, procaine amide was considered (with one exception) to have failed after an arbitrary dose of 1 g. had been given. In one case, which had proved resistant to all other therapeutic measures, normal rhythm was finally restored after 3·4 g. of procaine amide had been given intravenously. Embree and Levine (1959) and Wright and Davis (1961) have also reported the successful use of large doses of procaine amide in the treatment of resistant tachycardia.

Armbrust and Levine (1950) considered the immediate prognosis of the attack of tachycardia to be good since, in all but a few of their patients, normal rhythm was restored following the appropriate therapy. In contrast, 20 out of the 83 patients (24%) in the present series died during the acute episode. The poor ultimate prognosis of those cases associated with organic heart disease in this review is in agreement with all other series published. Cooke and White (1943) reported that 17 out of 23 patients with coronary heart disease were dead within a few hours to 18 months, and Williams and Ellis (1943) reported the death of 20 out of 36 patients within one month. Armbrust and Levine (1950) considered the prognosis to be particularly grave when associated with an acute myocardial infarct, and noted a mortality rate of 64 per cent after one month, compared with a rate of 43 per cent in association with coronary artery disease. Herrmann *et al.* (1959) report a mortality rate in association with myocardial infarction of 77 per cent and with coronary artery disease of 50 per cent. The present series reveals, after one month, a lower mortality rate of 36 per cent with myocardial infarction, and 52 per cent with coronary artery disease. Our results suggest that the presence of congestive cardiac failure (and therefore the severity of the cardiac disease) before the onset of paroxysmal ventricular tachycardia adversely affects both the immediate and ultimate prognosis more than the type of heart disease. Only 1 patient, in whom congestive cardiac failure was present before the onset of tachycardia, was known to be alive after one year, whereas, in the absence of cardiac failure, patients may live for many years after the initial episode, regardless of the underlying pathology.

The prognosis is good in those patients without evidence of heart disease in this and all other published series. Although these patients may be desperately ill during the episode of tachycardia, no patient in the present review developed congestive cardiac failure and none died.

SUMMARY

The ætiology, treatment, and prognosis of paroxysmal ventricular tachycardia in 83 patients has been reviewed.

Underlying ischæmic heart disease was present in 72 per cent. In 4 (5%) there was no evidence of heart disease.

The tachycardia was considered to be induced by digitalis in 22 per cent, and possibly induced by digitalis in a further 11 per cent. The importance of assessing the possible ætiological role of digitalis in each case is emphasized.

A high proportion of cases of the intermittent variety and without associated congestive cardiac failure will revert spontaneously to normal rhythm in the first 24 hours. Oral quinidine has been shown to be a valuable therapeutic agent. In the more resistant cases, the intravenous administration of either quinidine or procaine amide was equally successful.

The immediate and ultimate prognosis is poor in those patients with underlying heart disease. It is suggested that prognosis is adversely affected by the presence of congestive cardiac failure rather than the type of underlying heart disease.

The prognosis is good in those cases with no evidence of heart disease.

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