Selected clinical features of paroxysmal tachycardia

A prospective study in 120 patients

Myron H. Luria¹

From the Division of Medicine, Saint Luke's Hospital, Cleveland, Ohio, U.S.A.

One hundred and twenty consecutive patients with paroxysmal tachycardia were personally evaluated in a prospective study. Some outstanding clinical features included the association of paroxysmal tachycardia and primary myocardial disease; the lack of palpitations in some patients with paroxysmal tachycardia; the relation of angina pectoris with paroxysmal tachycardia and coronary artery disease; the association of inferior and strictly posterior wall myocardial infarction with paroxysmal tachycardia; the unique presence of urina spastica in 19.2 per cent of patients; and the value of 'hunkering down' and sighing respiration in aborting attacks of rapid heart action. In addition, the role of precipitating factors in the onset of tachycardia and the finding that true paroxysmal atrial tachycardia ('PAT') appears to be an infrequent phenomenon when the entire group of paroxysmal arrhythmias is considered, have been presented. The relative ineffectiveness of digitalis, quinidine, and pronestyl has also been evaluated in a selected group of patients with frequent episodes of rapid heart action.

Largely descriptive clinical studies have diminished in frequency. This may reflect the many excellent descriptive studies of the past as well as the present-day preoccupation with methodology. In the past 30 years clinical descriptions of the paroxysmal tachycardias have similarly been uncommon (Campbell and Elliott, 1939; Wolff, 1942; Armbrust and Levine, 1950; Nadas *et al.*, 1952; Jones, 1954; Lundberg, 1963; Mackenzie and Pascual, 1964; Haynal and Matsch, 1968), aside from textbook reviews and occasional reports of unusual forms of treatment and rare electrocardiographic rhythms. Unlike many of these previous reports, the present study was undertaken prospectively. Several distinctive clinical features were found and evaluated which have hitherto received insufficient attention.

Patients

From 1965 to 1969, 120 consecutive patients with paroxysmal tachycardia were personally evaluated by the author. A history of sudden onset of rapid heart action unassociated with untoward acute cardiac disease such as an acute myocardial infarction or acute rheumatic fever was used as

the criterion for the clinical diagnosis of paroxysmal tachycardia and was substantiated by electrocardiogram in 101 patients. Careful attention was given to 19 patients in whom a record of the tachycardia could not be obtained, particularly as to historical reliability. Each of these latter patients had repeated episodes of abrupt onset of tachycardia with symptoms of palpitations.

Seventy-five patients were male and 45 female. The patients have been divided into 5 broad groups aetiologically (Table 1): idiopathic, short PR syndromes, primary myocardial disease, rheumatic heart disease, and coronary heart disease. Hyperthyroidism was not a feature in any patient. The age at onset of paroxysmal tachycardia is listed in Table 2. A preponderance of tachycardia without a known cause reflects the clinically healthy population from which these patients were drawn. The benignity of paroxysmal tachycardia may be deduced from Table 3 wherein it is evident that recurrent rapid heart action may persist for many years.

Of the 15 patients with a short PR syndrome, 10 had the Wolff-Parkinson-White syndrome (1930): one patient had an atrial reciprocating tachycardia, the probable mechanism for tachycardia in most patients with a short PR syndrome (Luria and Hale, 1970); 6 patients had supraventricular tachycardia without such a reciprocating mechanism clearly evident; 2 patients had paroxysmal atrial fibrillation; and one patient had Ebstein's anomaly; however, an electrocardiogram could not be obtained during an episode of tachy-

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¹ Address for correspondence: Dr. Myron H. Luria, St. Luke's Hospital, 11311 Shaker Blvd., Cleveland, Ohio 44104.

cardia in this latter patient. A short PR interval with a normal QRS configuration, the Lown-Ganong-Levine syndrome (1952), was seen in 5 patients during sinus rhythm. In 2 of these patients, paroxysmal supraventricular tachycardia occurred, and in one patient paroxysmal atrial fibrillation was found. An electrocardiogram could not be obtained during an episode of tachycardia in 2 additional patients.

The diagnosis of primary myocardial disease was deduced by the clinical course and cardiac catheterization with left ventricular and coronary angiography in 4 patients who suffered from recurrent paroxysmal tachycardia as the chief clinical manifestation of their cardiac disease. The incidence of rheumatic heart disease was low. On the contrary, objective evidence of coronary artery disease was present in 14 patients. This perhaps reflects the changing pattern of cardiac disease in the United States.

Clinical observations

Precipitating factors The onset of tachycardia was often viewed mechanistically by the patients as occurring in relation to some specific event or movement. These may be divided into several general categories (Fig. 1). 1) Sustained exercise complicated by paroxysmal tachycardia was seen in 27 patients. Examples include marching, dancing, running, swimming, and climbing stairs.

2) Sudden movement was noted just before the onset of tachycardia in 31 patients. Examples include bending over, turning around quickly, standing up suddenly, and rapidly taking a deep breath.

3) Psychic factors were notable in 25 patients and include the occurrence of anger, fright, or intense excitement; looking at television or the movies; or reading a novel at the time of onset of the tachycardia. Dreams may have been of some importance in 10 patients who were awakened from sleep with tachycardia. In 2 of these patients a specific recurrent dream – falling off a cliff and a car accident, respectively – appeared to be directly related to the onset of paroxysmal tachycardia. In order to further evaluate the role of the central nervous system, an electroencephalogram was recorded in 7 patients. Minor non-specific abnormalities during both paroxysmal tachycardia and sinus rhythm were observed in 2 patients, the other records being entirely normal. Leptazol was infused in 2 patients during sinus rhythm, but no electroencephalographic or electrocardiographic alterations were elicited.

4) Chemicals such as poison gas (during a practice military drill), parenteral epinephrine, amyl nitrite, and alcohol were related to the onset of tachycardia in 9 patients.

Coffee could be related to the onset of tachycardia in only one of the present 120 patients and cigarettes in only two.

5) Infection was associated with the onset of tachycardia in 11 patients who had no clinical evidence of cardiac involvement such as myocarditis. In 6 patients this consisted of an upper respiratory infection; in 2 patients pneumonia was present; and in 3 patients an

TABLE I *Type and aetiology of paroxysmal tachycardia in 120 patients*

<i>Paroxysmal arrhythmia</i>	<i>Idiopathic</i>	<i>Short PR</i>	<i>Primary myo-cardial disease</i>	<i>Rheumatic heart disease</i>	<i>Coronary heart disease</i>	<i>Total</i>
Ectopic atrial tachycardia	6				2	8
Supraventricular tachycardia	25	8	1		3	37
Atrial reciprocating tachycardia		1				1
Atrial flutter	7		1		1	9
Atrial fibrillation	25	3	2	2	3	35
AV junctional tachycardia	2					2
Ventricular tachycardia	5				2	7
Repetitive tachycardia: atrial	1					1
ventricular	1					1
Uncertain (no ECG)	13	3			3	19
Total	85	15	4	2	14	120

TABLE 2 *Age at onset and aetiology of paroxysmal tachycardia in 120 patients*

<i>Age</i>	<i>Idiopathic</i>	<i>Short PR</i>	<i>Primary myocardial disease</i>	<i>Rheumatic heart disease</i>	<i>Coronary heart disease</i>	<i>Total</i>
0-9	5	1				6
10-19	14	3				17
20-29	21	4				25
30-39	12	2	2		3	19
40-49	18	3	2	1	3	27
50-59	9	1		1	5	16
60-76	6	1			3	10
Total	85	15	4	2	14	120

TABLE 3 *Duration of recurrent paroxysmal tachycardia*

<i>Age at onset</i>	<i>< 1 yr.</i>	<i>1-5 yr.</i>	<i>6-10 yr.</i>	<i>11-15 yr.</i>	<i>16-30 yr.</i>	<i>Total</i>
0-9		3	1		2	6
10-19	1	6	4	2	4	17
20-29	8	8	1	4	4	25
30-39		7	6	2	4	19
40-49	5	13	5	1	3	27
50-59	3	9	3	1		16
60-76	5	3	2			10
Total	22	49	22	10	17	120

ear infection, urinary tract infection with septicaemia, and recurrent prostatitis were present, respectively. Of considerable note was the association of an upper respiratory infection with all 8 episodes of supra-ventricular tachycardia in a 2-year-old girl whose paroxysmal tachycardia was first noted at the age of 2 months. A 53-year-old man also had 4 episodes of paroxysmal atrial fibrillation associated with upper respiratory infections within 3 years, and a 77-year-old man had 4 episodes of paroxysmal atrial flutter in 6 years, each associated with prostatitis.

6) Gastrointestinal upset was observed in 8 patients before the onset of paroxysmal tachycardia and was chiefly manifested by extreme bloating or belching, especially after a large meal.

Palpitations Palpitations are the *sine qua non* of tachycardia. However, their lack of full reliability was manifest in 8 patients with recurrent rapid heart action who had tachyarrhythmic episodes documented fortuitously by electrocardiogram unassociated with an awareness of the tachycardia. In addition, 4 of these patients had never had palpitations: 2 patients complained of chest pain (angina pectoris) during their attacks, one noted marked shortness of breath, and one became very weak. In the majority, the palpitations were readily noted and very bothersome, though patients with frequent attacks often continued in their daily jobs if no other symptoms arose. Moreover, the rapid heart action was often noted by acquaintances – cigarettes in a shirt pocket moving in time to the heart beat, or, as noted by the wife of a patient, movement of the bed in time to the rate of the heart.

Angina pectoris Chest pain frequently accompanied the tachycardia and usually consisted of a vague praecordial aching sensation. However, 17 of the 120 patients had pain typical of angina pectoris during their attacks and indeed 5 patients obtained relief with nitroglycerin during the tachycardia. One of these 17 patients, a 21-year-old man who was otherwise in good health, had paroxysmal ventricular tachycardia. Ten other patients had objective clinical and electrocardiographic evidence of coronary artery disease. Three of the 4 patients who died in this study are included in this group. Each died at home very suddenly without a clinical picture of rapid heart action at the time of death. Though overt manifestations of heart diseases were not present in the remaining 6 patients with

FIG. 1 Precipitating factors in 120 patients with paroxysmal tachycardia. GI, gastro-intestinal.

angina pectoris during paroxysmal tachycardia, their age range of 40 to 66 years is consistent with the likelihood of coronary artery disease, which might be brought out only by a pronounced tachycardia.

Electrocardiograms Contrary to popular usage, so-called 'PAT', or paroxysmal atrial tachycardia, was an uncommon phenomenon and was observed in only 8 patients. The majority of patients with paroxysmal tachycardia had either atrial fibrillation or supra-ventricular tachycardia of undetermined origin. The latter connotes a tachycardia wherein the ventricular rate is regular but in which P waves are not discernible. The site of origin of these supraventricular tachycardias may thus be either in the atria or AV junction. The differentiation of ectopic atrial tachycardia and atrial flutter was an additional difficult clinical problem and was often judged by the atrial rate. One woman had paroxysmal ectopic atrial tachycardia with 2:1 AV conduction with an atrial rate of 214 beats a minute. The 9 patients with atrial flutter also generally had 2:1 AV conduction before treatment with digitalis was begun, but by definition their atrial rates were above 250 beats a minute.

Of the rarer arrhythmias, both the atrial and ventricular varieties of repetitive tachycardia (Gallavardin and Dumas, 1924; Parkinson and Papp, 1947) were seen in 2

patients. Three patients had ventricular extrasystolic beats with occasional runs of paroxysmal ventricular tachycardia in the absence of demonstrable heart disease. Four patients had recurrent paroxysmal ventricular tachycardia which was more sustained and which frequently could only be stopped with either intravenous procainamide or cardioversion.

Myocardial infarction Twelve patients had objective evidence of an old myocardial infarction electrocardiographically and are of special note. Two of the 3 patients with anterior wall infarction had paroxysmal ventricular tachycardia whereas all 9 patients with inferior wall infarction had recurrent supraventricular arrhythmias. Furthermore, 4 of these latter 9 patients also had involvement of the strictly posterior wall.

Urina spastica A remarkable renal response to paroxysmal tachycardia is polyuria, termed 'urina spastica' by Wenckebach and Winterberg (1927). Twenty-three of the 120 patients had urina spastica associated with their rapid heart action, an incidence of 19.2 per cent. No relation to a specific arrhythmia was evident (Table 4). Though urina spastica

TABLE 4 *Prevalence of polyuria in paroxysmal tachycardia*

<i>Paroxysmal arrhythmia</i>	<i>Patients</i>	<i>With polyuria</i>
Ectopic atrial tachycardia	8	2
Supraventricular tachycardia	37	11
Atrial reciprocating tachycardia	1	
Atrial flutter	9	
Atrial fibrillation	35	4
AV junctional tachycardia	2	
Ventricular tachycardia	7	1
Repetitive tachycardia: atrial	1	
ventricular	1	
Uncertain (no ECG)	19	5
Total	120	23

might appear before the onset of tachycardia or after an episode had terminated, it usually began within 10 or 15 minutes after the onset of rapid heart action. Large amounts of urine were then passed at frequent intervals for one to one and a half hours and then gradually declined, both in frequency and amount. Up to 1500 ml. urine have been passed in one hour by one of the present patients, and she has become well acquainted with the location of all available rest rooms contiguous to her

day-to-day work and shopping. Many expressed the feeling that 'there seems no end to it' and if they could only 'get it all out, it would stop.' Another described it as 'like taking a water pill.' One 57-year-old housewife developed angina pectoris with each episode of tachycardia and found it difficult to scurry back and forth to the bathroom. Her unique solution was to lie in bed on a bed pan for 60 to 70 minutes after the onset of rapid heart action while passing large amounts of urine.

Halting the tachycardia The majority of patients with paroxysmal atrial, junctional, or supraventricular tachycardia of undetermined origin soon found that their rapid heart action was limited in length and lay down or rested in a chair until it subsided. It was the rare patient in whom physical manoeuvres of vagal stimulation invariably aborted the attack, though often when one previously successful manoeuvre was not effective another was. In the present series, though carotid sinus pressure at times was effective in 17 patients, the Valsalva manoeuvre in 8 patients, gagging in 6 patients, and eyeball pressure in 5 patients, each patient, nevertheless, could not be entirely confident of abbreviating all episodes. At these times, medical advice was often sought. This usually consisted of pharmacological means of vagal stimulation via blood pressure rises such as phenylephrine in 2 patients or metaraminol in 2 patients. The effectiveness of the most commonly used medication, intravenous cedilanid (9 patients), may also be related to the production of a transient rise in blood pressure (Ross, Waldhausen, and Braunwald, 1960) and resulting vagal stimulation.

The above physical methods of vagal stimulation were usually learned from a physician. Of particular note were the experiments of patients on their own. Seven patients found that by leaning over with the head down, especially in a squatting position, but also from a sitting position, the attack of supraventricular tachycardia could be aborted (Fig. 2). Three of these patients felt that this might be too conspicuous when at work and had individually modified the position so that they appeared to be tying their shoes. It is notable that the term squatting was usually not used to describe this manoeuvre by patients from Appalachia, but instead a term from the eastern Kentucky patois, 'hunkering down', was employed. According to Webster's *Dictionary* (1961) 'hunker' is of Scandinavian origin and when combined with the word 'down' means to crouch or squat. The value

FIG. 2 'Hunkering down' for paroxysmal tachycardia.

of 'hunkering down' may be related to the low position of the head with resultant stimulation of the carotid sinus stretch receptors.

Another mechanism arrived at individually by 8 patients was sighing respiration or a deep breath. Scherf and Schott (1953) have termed this a pulmonocardiac reflex and Bamberger (1857) had mentioned this measure in 1857. Its mechanism is unknown.

An alternative method of aborting an attack, electric countershock, was used in 10 patients not receiving digitalis. Five patients with supraventricular tachycardia of undetermined origin who were in clinical shock and had hypotension without palpable brachial pulses were successfully treated. The method was also particularly effective in 5 patients with paroxysmal atrial fibrillation or flutter. These latter specific paroxysmal arrhythmias previously often lasted 24 hours or longer despite various drug treatments. Electric countershock was applied expeditiously with intravenous diazepam anaesthesia. Within 15 to 30 minutes the patients were fully awake and could be sent directly home when the procedure was performed in the emergency room.

Prophylaxis The difficulties inherent in evaluating the prophylactic treatment of the paroxysmal tachycardias are readily evident

when the history of one of the present patients is examined.

A 40-year-old housewife had 3 to 8 hour episodes of supraventricular tachycardia yearly. Following her third episode of rapid heart action, she was placed on maintenance digitalis therapy which was continued for one and a half years without the occurrence of rapid heart action. The drug was then stopped and for the past two years the patient has been asymptomatic.

Was digitalis effective in this patient? Obviously no reliable judgement can be made, but the vicissitudes of this paroxysmal disease are self evident. Twenty patients in the present series had episodes of rapid heart action of sufficient frequency – usually once per week but at least once monthly – so that the natural variation in the onset of tachycardia could be held to a minimum (Table 5).

TABLE 5 Prophylaxis of frequent recurrent paroxysmal tachycardia

Paroxysmal arrhythmia	Patients	Response* (+) (-)
Atrial fibrillation	8	
digitalis		8
quinidine 0.2 g. q.i.d.		3
quinidine 0.4 g. q.i.d.		1 3
Supraventricular tachycardia	12	
digitalis		9
quinidine 0.2 g. q.i.d.		5
quinidine 0.4 g. q.i.d.		1
procainamide 500 mg. q.i.d.		3

* Response to prophylactic drug treatment: (+) diminished frequency or elimination of symptoms of paroxysmal tachycardia; (-) no change.

None had ventricular tachycardia or rheumatic heart disease; one had pre-excitation; and 2 had evidence of inferior and strictly posterior wall myocardial infarction. Each patient was given each medication for at least 3 months, though some received pharmacological therapy for as long as 5 years. In 8 patients with paroxysmal atrial fibrillation there was no change in the frequency of tachycardia with prophylactic digitalis; quinidine was ineffective in 3 patients at 0.2 g. q.i.d. and in 3 other patients at 0.4 q.i.d.; however, no episodes of tachycardia occurred for one year in one patient when quinidine was increased to 0.4 g. q.i.d. In 12 patients with recurrent supraventricular tachycardia (of which 2 were discernibly ectopic atrial, one AV junctional, and 9 undetermined in origin) there was no change in the frequency of tachycardia with prophylactic

digitalis. Quinidine was ineffective in 5 of these patients at 0.2 g. q.i.d. and in one patient with pre-excitation it was ineffective at 0.4 g. q.i.d.; procainamide in a dose of 500 mg. every 6 hours was also ineffective in 3 of these patients. Though digitalis did not diminish the frequency of tachycardia in any patient, one patient with paroxysmal atrial fibrillation developed shorter episodes with the drug. Moreover, in 5 patients with paroxysmal supraventricular tachycardia, digitalis administration was accompanied by less intense subjective palpitations, greater effectiveness of physical means of aborting the tachycardia, as well as shortening of the periods of rapid heart action. Not included in this group of 18 patients are 2 patients, one with atrial and another with ventricular repetitive tachycardia. The former did not respond to digitalis or quinidine and the latter did not respond to quinidine. On the contrary, in 2 other patients, each with the centrally excitable type of paroxysmal tachycardia, episodes of rapid heart action were eliminated with propranolol 40 mg. q.i.d.

Discussion

The pale, clammy, intensely anxious patient with rapid heart action makes an indelible picture not soon forgotten. The present study was carried out prospectively in order to personally evaluate this clinical picture and several distinct clinical features have been considered.

Most patients diligently searched to relate the onset of their tachycardia to a specific event. However, only 7 of the 120 patients could consistently find some association for each of their episodes of tachycardia – the '*tachycardie paroxystique à centre excitable*' of Gallavardin (1922). Though the listing of precipitating factors is superficially impressive, their variability and lack of specificity suggest that their relation to the onset of tachycardia is questionable for the majority of patients, however significant they may appear in an individual episode of tachycardia. This has previously been exemplified by medical observations made in the Warsaw Ghetto (Tushnet, 1966), certainly at a time of physical exhaustion, infectious disease, and immense terror, wherein a lack of paroxysmal tachycardia was noted. This low incidence of specificity has also led to a consideration of the similarities of paroxysmal tachycardia and epileptic seizure disorders (Nothnagel, 1887; Parrow, 1966; Pasini *et al.*, 1967). The present small series of standard electroencephalograms is inconsistent with this latter supposition, as are the two cases wherein

leptazol was injected. Further information might have been obtained if a diencephalic lead system had been employed, but this is presently impractical.

An intricate relation between coronary artery disease and recurrent episodes of paroxysmal tachycardia existed in patients with angina pectoris and myocardial infarction. Since a pronounced tachycardia may conspicuously increase oxygen consumption of the heart, periods of rapid heart action may have represented a natural form of an exercise tolerance test, especially when symptoms typical of angina pectoris were produced in the present patients. Though Wolff (1945) has emphasized that an absence of angina pectoris in paroxysmal tachycardia does not preclude coronary artery disease, patients in the present study without a high probability of coronary artery disease did not develop angina pectoris at heart rates similar to those with pain. The role of myocardial infarction appeared aetiologic. The high incidence of inferior and also strictly posterior wall myocardial infarction may indicate significant disease of the nutrient vessels of the sinus node and its connecting conduction system, i.e. the right coronary artery.

Another relation was found between primary myocardial disease and paroxysmal tachycardia. The customary clinical presentation of patients with primary myocardial disease is either one of congestive heart failure, cardiac restriction, or left ventricular outflow tract obstruction (Goodwin *et al.*, 1961). The present findings suggest that a fourth chief presenting manifestation is recurrent paroxysmal tachycardia.

Urina spastica is a feature of paroxysmal tachycardia which has gained considerable attention as a result of the writings of Wood (1963). The water diuresis has been attributed to a mechanism involving left atrial distension and subsequent reflex antidiuretic hormone inhibition (Wood, 1963; Luria, Adelson, and Lochaya, 1966a). Cardiac output is not raised during the polyuria but creatinine clearance, total solute excretion, and free water clearance are increased (Luria *et al.*, 1966a). The cause of the large solute diuresis is unclear but a selective increase in renal blood flow has been postulated. Previous surveys have also indicated that urina spastica is not found in patients with valvular heart disease. If these latter patients are excluded, approximately one-half of patients with paroxysmal tachycardia are reported to have an associated polyuria (Wood, 1963; Luria *et al.*, 1966a). The lower incidence in the present study may reflect the effect of a prospective study.

Pharmacological therapy of the supra-ventricular paroxysmal tachycardias was generally ineffective. Thus, though digitalis, quinidine, or procainamide may appear effective in many patients with widely separated episodes of tachycardia, in a small group of selected individuals with a predictable frequency of paroxysmal tachycardia the results were discouraging. Of note is that though digitalis did not diminish the frequency of tachycardia, symptomatic improvement was observed in some patients and the attacks were of shorter duration. The apparent ineffectiveness of quinidine may also be more subtle; higher doses might be more effective, but gastrointestinal irritation then becomes a prominent limiting factor. Propranolol was given to only a few of the present patients and was thus not fully evaluated. However, its usefulness in these patients with the centrally excitable forms of paroxysmal tachycardias has been previously shown (Luria, Adelson, and Miller, 1966b; Gettes and Surawicz, 1967).

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