PAROXYSMAL TACHYCARDIA IN INFANCY AND CHILDHOOD

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Occasionally in pædiatric wards and more often in private practice, one learns of cases of sudden death in infants and children. The clinical signs have not been characteristic for a diagnosis, and the autopsy has failed to determine the cause of death. Among these there are a number whose sudden death is due to paroxysmal tachycardia. Its occurrence has been considered rare in infancy and childhood, but is more frequent among children than is commonly thought. Of recent years, the number of case reports has been increasing, due to the greater interest taken in the cardiovascular diseases in childhood.

Apart from reports of isolated cases, there are only three papers dealing with more than one case. Koplik (1917) reported three cases: the first was a baby of 22 months, who had tonsillitis and bronchitis two weeks before the onset of paroxysmal tachycardia; the second was a child three years of age, and the third a child ten years old; in the last case the paroxysmal tachycardia started after a tonsillar sepsis and the attacks were observed for two years. Shookhoff *et al.* (1932) reported four cases: the paroxysms occurred in a child of eight with septic teeth; in a child of three after pertussis; in a child aged ten with an abscess of the right lower premolar and pharyngitis; and in another case three years of age. Campbell (1937) added three cases in the *Guy's Hospital Reports*: the first was a baby one month old with a temperature 101° F., but with no other definite pathological findings; the second a baby of 20 months with vomiting and liver enlargement; and in the third paroxysmal tachycardia manifested itself in a child of three years with bronchitis of two weeks standing and with otitis media. Hubbard (1941) called attention to the fact that paroxysmal tachycardia shows itself as a distinctive clinical entity in young infants, responding satisfactorily to proper treatment; he reported nine cases in infants less than one year of age.

Considering the paucity of reports on this subject it seems justifiable to give an account of the cases observed in this hospital from 1941 to 1945. It comprises eleven cases; a short clinical report, cardiographic findings, pathological, anatomical, and histological reports are given below.

Clinical signs. The onset of paroxysmal tachycardia is sudden. The attack lasts for minutes, hours, or days. The child appears acutely ill on physical examination. It is restless at the outset, but apathetic once the attack is developed, and looks pale and often cyanosed. There is respiratory distress with rapid and shallow respirations. During the attack, most commonly the heart rate is 160–200 a minute, but rates of 270 and 300 have been observed (Campbell, Langley, Lewis, Werley). Between and following attacks premature beats are often found. Signs of congestive heart failure, e.g. râles in the lungs, engorged jugular veins, and liver enlargement may develop. The pulse is often imperceptible. The temperature is usually raised. The X-ray shows pulmonary congestion and an enlarged heart in some instances.

A heart rate exceeding 190–200 in a small infant should always rouse suspicion. Too little attention is paid to a rapid heart action in babies, and that is the reason for paroxysmal tachycardia being sometimes overlooked.

CASE REPORTS

Case 1. Three years old, admitted 13/12/1941. No previous diseases. Clinical diagnosis: nasopharyngeal diphtheria of gravis type. 60,000 units antitoxin given intraperitoneally. Heart on admission: cardiac dullness enlarged, embryocardia. The next day there was restlessness and cyanosis, and the paroxysm started (Fig. 1). The patient died the same day.

FIG. 1.—Case 1. 14/12/41. Ventricular rate 155 a minute; duration of QRS, 0.07 sec.; P-R, 0.05 sec. in lead III. This supraventricular tachycardia is considered to be of upper nodal origin.

Case 2. Four years old. Admitted 27/12/1941. Previous diseases: pertussis, chickenpox, mastoid operation. Diphtheria two weeks ago. Clinical diagnosis: diphtheritic myocarditis. Heart on admission: enlarged cardiac dullness, systolic apical murmur. Second apical sound accentuated, pulse 108 a minute.

29/12/41. Onset of heart paroxysm (Fig. 2A), cyanosis, liver enlargement, albuminuria.

31/12/41. Heart action irregular due to premature beats.

1/1/42. Pulse 138. Extrasystoles. B.P. 76–48 (see Fig. 2B).

8/1/42. Heart action regular. Colour fair. Liver not enlarged, albuminuria subsiding. No murmur audible.

FIG. 2.—Case 2. (A) 29/12/41. Heart action regular, ventricular rate 150 a minute, duration of QRS 0.08 sec. Supraventricular paroxysmal tachycardia. (B) 2/1/42. Sinus rhythm, ventricular rate 130 a minute; P-R, 0.17 sec. Premature beats. T flat in leads I and II. Ventricular deflections notched, voltage less than 1.5 mV. in all three leads.

Case 3. Six years old. Admitted 4/5/1942. No previous diseases. Clinical diagnosis: nasopharyngeal diphtheria of gravis type. 60,000 units antitoxin intraperitoneally. Heart normal on admission.

6/5/42. Pale and apathetic, with vomiting and an imperceptible pulse (see Fig. 3A).

7/5/42. Cardiac dullness enlarged, vomiting persistent. Attack persisted but was thought to be paroxysmal ventricular tachycardia (see Fig. 3B). The patient died two days later.

Case 4. Five years old. Admitted 14/4/1943. Previous diseases: measles. Clinical diagnosis: hæmorrhagic nasopharyngeal diphtheria of gravis type. 60,000 units antitoxin given intravenously. Blood transfusion. Heart normal on admission. B.P. 80/53.

FIG. 3.—Case 3. (A) 6/5/42. Action irregular. Alternately every second and third P wave hidden within the T wave. Auricular tachycardia with 3:1 and 2:1 A-V block; A, 300; V, 130. In this and in other figures dots designate P waves. (B) 7/5/42. Action regular. Ventricular rate 140 a minute. Duration of QRS 0.10 sec. Atypical configuration of the ventricular deflection. Ventricular paroxysmal tachycardia.

18/4/42. Pale; cardiac dullness enlarged. Pulse 100. B.P. 66/42.

21/4/42. Apathetic, vomiting. Pulse 100–90 a minute.

24/4/42. Cyanosis, restlessness. Heart rate 88. Premature beats.

24/4/42. Onset of heart paroxysm, pulse imperceptible. B.P. 38/20 (see Fig. 4). The patient died two days later.

Case 5. Four years old. Admitted 11/4/1944. Previous diseases: chickenpox. Clinical

FIG. 4.—Case 4. 24/4/42. Heart action regular. Ventricular rate 160 a minute. Duration of QRS 0.08 sec. Since no P waves are visible, this supra-ventricular tachycardia is considered to be of nodal origin.

FIG. 5.—Case 5. 15/5/44. Heart action regular. Ventricular rate 120. Ventricular complexes bizarre; duration 0.23 sec. Ventricular paroxysmal tachycardia.

diagnosis: nasopharyngeal diphtheria of gravis type. 80,000 units antitoxin given intravenously. Heart normal on admission.

15/5/44. Onset of heart paroxysm (Fig. 5). Vomiting; pulse imperceptible; respiratory rate 20; albuminuria. Died.

Case 6. Five years old. Admitted 5/5/1944. Previous diseases: ?diphtheria, measles, otorrhœa. Clinical diagnosis: nasopharyngeal diphtheria of intermedius type. 40,000 units antitoxin given intramuscularly. Pulse rate 130–140. First and second sounds equal; B.P. 110/80.

11/5/44. Second apical sound greater than first. Pulse 100–108. Albuminuria.

12/5/44. Pallor, vomiting, temperature normal. Onset of heart paroxysm (Fig. 6). Died.

Case 7. Six months old. Admitted 20/6/1944. Previous diseases: bronchitis, eczema, measles a week ago. Clinical diagnosis: pertussis. Heart normal on admission. W.B.C., 56,000; 68 per cent lymphocytes.

19/7/1944. Syncope, cyanosis. Pulse imperceptible (Fig. 7). No clinical signs of pneumonia (Fig. 7).

20/7/1944. Extreme tachycardia and tachypnœa. Temperature raised. Signs of heart failure, râles in the lungs. Died.

FIG. 6.—Case 6. 12/5/44. Action regular in lead II and III. Ventricular rate 143. Duration of QRS 0.12 sec. Auricular waves thought to be visible in chest lead CF₂, auricular rate 272, every second P wave hidden in the following T wave. Auricular paroxysmal tachycardia with 2:1 A-V block.

FIG. 7.—Case 7. 19/7/44. Heart action regular. Auricular paroxysmal tachycardia with possible 2:1 conduction: A, 460; V, 230.

Post-mortem report. (From the Department of Pathology, University of Durham.)

Pericardium, smooth and glistening. Heart, 52.0 g. (Mean weight at this age 31.0 g.) Epicardium quite smooth. Right auricle dilated and filled with agonal clot; no thrombus in appendix. Foramen ovale completely closed. Tricuspid leaflets thin and delicate. Right ventricle slightly dilated; wall 0.3 cm. thick. Pulmonary valves thin and delicate. Pulmonary artery smooth and elastic. Left auricle empty; no evident dilatation; no thrombus in appendix or elsewhere. Mitral leaflets thin and delicate. On anterior leaflet 0.2 cm. from the free margin there is an "angioma"; about 0.2 cm. from it is another one which appears to have

healed. Left ventricle not dilated; very little clot; wall 0.7 cm. thick. Aortic leaflets thin and delicate. Orifices of coronary arteries patent. Ductus arteriosus closed. Aorta smooth and elastic. Myocardium firm but greyish and pink.

Histology. A topographical survey of the heart, including epicardium, right and left auricles, mitral and tricuspid valves, annulus fibrosus, auriculo-ventricular sulcus, right and left ventricles, was made and there was no trace of any form of myocarditis. Special staining showed a mild grade of diffuse fatty degeneration of the myocardium which could be due to many forms of toxæmia.

Case 8. Three years old. Admitted 18/10/1944. Previous diseases: rubella, pertussis. Clinical diagnosis; hæmorrhagic nasopharyngeal diphtheria and *S. hæmolyticus* infection. 50,000 units antitoxin intravenously. Blood transfusion. Heart normal on admission.

21/10/44. Runs of premature beats and short paroxysm (Fig. 8A). B.P. 85/38. Albuminuria.

24/10/44. Onset of longer paroxysm. Pulse too fast to count (Fig. 8B).

26/10/44. Extremities icy-cold. Liver enlargement. B.P. could not be measured. Patient died.

FIG. 8.—Case 8. (A) 21/10/44. Onset of supraventricular paroxysmal tachycardia in lead I. Ventricular rate 171. The paroxysm has subsided in lead II, the rate being 82. There are no P waves visible, the heart action is regular especially in lead III, in which the ventricular rate is 100. Duration of QRS 0.10 sec. The paroxysm in lead I and the rhythm in lead II and III are considered to be of nodal origin. (B) 24/10/44. Heart action regular. Ventricular rate 188, duration of QRS 0.11 sec. Left ventricular preponderance. Ventricular paroxysmal tachycardia.

Post-mortem report. (From the Department of Pathology, University of Durham.)

Heart. Weight 90 g. (fixed in formalin). Right auricle slightly dilated, containing a large amount of post-mortem thrombus. Auricular appendage contains adherent mural thrombus. Foramen ovale closed. Tricuspid valve admits two fingers: cusps thin and delicate. Right ventricle slightly dilated and contains post-mortem thrombus. Endocardium shows hæmoglobin staining; muscle, 0.3 cm. thick at the base. Pulmonary valve shows marked hæmoglobin staining of its cusps. Coronary artery normal apart from hæmoglobin staining. Left auricle healthy: no mural thrombi. Mitral valve admits one finger and cusps thin and delicate. Left ventricle dilated with some endothelial hæmorrhages on the interventricular septum: muscle at the base 0.7 cm. thick. Aortic valve healthy. Coronary arteries patent. Myocardium, no obvious fatty change.

Histology. Heart: left auricle, mitral valve, left ventricle. Intense veno-capillary congestion in the myocardium together with a sparsely but widely distributed fatty degeneration. No evidence of myocarditis or valvulitis.

Right auricle, tricuspid valve, right ventricle. Recent mural ante-mortem thrombus in appendix, but apart from this changes similar to those in the left side.

Auriculo-ventricular node; similar changes but no inflammation.

The appearance here pointed to an acute and intense cardiac failure, the cause of which is not apparent from this material.

Case 9. Four years old. Admitted 1/12/44. No previous diseases. Clinical diagnosis: meningo-encephalitis. Heart normal on admission, no consolidation in the lung. W.B.C., 16,800; 74 per cent polymorphs and 21 per cent lymphocytes.

7/12/44. Pulse 124. B.P. 105/65. Lung: no apparent disease.

9/12/44. Onset of heart paroxysm, pulse too fast to count (Fig. 9). Temperature 100° F., signs of consolidation in the lung. Died.

FIG. 9.—Case 9. 9/12/44. Heart action regular. Auricular rate 376, ventricular rate 188. Alternate P waves hidden within the S-T transition. Paroxysmal auricular tachycardia with 2:1 A-V block.

FIG. 10.—Case 10. 18/2/45. Action regular. Ventricular rate 176. Auricular rate 352. Both auricular waves are hidden within the S-T-T complex. Paroxysmal auricular tachycardia or flutter with 2:1 A-V block.

Post-mortem report. (From the Department of Pathology, University of Durham.)

Anatomical diagnosis: Acute suppurative meningitis. Acute bronchopneumonia. Accidental involution (thymus).

Pericardial sac, smooth and glistening.

Thoracic aorta, quite smooth and elastic.

Heart. Weight 75 g. (fixed in formalin). Pericardium smooth and glistening: no gross abnormality. Right auricle empty. Foramen ovale completely closed. Tricuspid leaflets thin and delicate. Right ventricle a small amount of post-mortem clot: wall 0.3 cm. thick. Pulmonary valves thin and delicate. Pulmonary artery smooth and elastic. Left auricle empty. Mitral leaflets thin and delicate. Left auricle empty, but dilated: wall 0.9 cm. thick. Pulmonary leaflets thin and delicate. Orifices of coronary arteries patent. Myocardium distinctly pale and turbid in appearance.

Histology. Heart (left auricle, myocardium, left ventricle): no evident abnormality.

Case 10. Four months old. Admitted 13/2/45. Previous diseases: otorrhœa. Clinical diagnosis: epidemic cerebrospinal meningitis. Heart on admission: tachycardia. Pulse volume poor.

17/2/45. Slight vomiting. B.P. 98/40. W.B.C.: 24,000; 61 per cent polymorphs and 25 per cent lymphocytes.

18/2/45. Onset of heart paroxysm (Fig. 10).

20/2/45. Heart action regular, rate 150 a minute.

22/2/45. Cardiac dullness enlarged, pulse volume good.

1/3/35. Heart: no apparent disease.

Case 11. Five months old. Admitted 15/2/45. No previous diseases. Has been ill for the last five days, but no clinical signs could be found. The baby's mother had a sore throat a few weeks ago, and a child in the same family got scarlet fever two weeks ago; because of this the baby was given scarlet antitoxin and sulphonamide before admission. Heart on admis-

FIG. 11.—Case 11. 15/2/45. Heart action regular. Ventricular rate 222, auricular rate 444. Paroxysmal auricular tachycardia with 2:1 A-V block.

sion: action too fast to count. Lung: no apparent disease. Clinical examination did not reveal any signs of a *S. hæmolyticus* infection.

16/2/45. Colour grey, eyes sunken, hands cold. Lung: no consolidation: X-ray negative. Bacteriological findings negative. Patient died.

DISCUSSION

Eleven cases of paroxysmal tachycardia are reported in the present series. The age of the patients was under one year in 3 cases, and from three to six years in 8 cases.

The heart condition was associated with various infections: a diphtheritic infection of gravis or intermedius type was present in seven cases, pertussis was found in another, meningitis in two more, but no infectious focus could be traced in the last case, a baby five months old. These findings leave little doubt that paroxysmal tachycardia in childhood occurs more often in the presence of an infection than in its absence. Koplik assumed that the first attack of influenza in one of his cases was the starting point of the paroxysm, and that the cardiac collapse of the first paroxysm was the forerunner of the subsequent paroxysms. The infective origin is obvious in Shookhoff's cases, e.g. in one child while convalescing from pertussis. Hauser (1921) referred to pertussis as a cause of paroxysmal tachycardia. Hubbard admits

that paroxysmal tachycardia may be associated with some other illness, but he believes it may start with no other evident cause. One child in Campbell's series probably had a diphtheria prior to the onset of the paroxysmal tachycardia.

The attack sets in suddenly. There are runs of premature beats lasting for a few seconds, heralding the onset of the paroxysm in some instances (see Fig. 8A). Frequent premature beats may also occur when the attack terminates (see Fig. 2B).

The main clinical signs enumerated in order of their frequency in the present series are the following: imperceptible pulse (6 cases), lowered systolic and diastolic blood pressure (5), vomiting (4), cyanosis (4), albuminuria (4), enlarged cardiac dullness (4), pallor (3), liver enlargement (2), apathy (2), raised temperature (2), syncope (1), embryocardia (1), systolic apical murmur (1), râles in the lung (1), and restlessness (1).

There are two cases in the present series which are of the type described by Hubbard (Cases 7 and 11), occurring in babies of six months and of five months. Pallor, restlessness, raised temperature, and the paroxysm being the signs of the onset, followed by signs of a failing heart the next day. No consolidation in the lung is found on clinical or X-ray examination. Attacks similar to those described might be mistaken for pneumonia; tachypnoea, cyanosis, and a raised temperature readily suggest a diagnosis of pneumonia.

Cardiographic findings. Auricular paroxysmal tachycardia and auricular flutter are closely related. Campbell found paroxysmal flutter to be more frequent in infants than in adults, and he regarded it as more common in infants than all varieties of simple paroxysmal tachycardia combined. Koplik's Case 3 was an example of auricular flutter. Three out of four cases of paroxysmal tachycardia reported by Shookhoff were auricular flutter. He gives a review of 40 cases of paroxysmal tachycardia occurring in children and published by different authors up to 1932. These cases presented themselves as ventricular paroxysmal tachycardia, 1 instance; auricular paroxysmal tachycardia, 5 instances; nodal paroxysmal tachycardia, 2 instances; paroxysmal auricular flutter, 12 instances; undiagnosed paroxysmal tachycardia with a rate of 200 or more, 18 instances.

Campbell regarded his Case 3 as probably flutter with 1:1 response. Up to-day it has been held that paroxysmal tachycardia is found with an auricular rate varying between 160 to 240, while auricular flutter is associated with higher auricular rates of about 300 a minute. Furthermore it was thought that auriculo-ventricular block is characteristic of auricular flutter, but was considered uncommon in paroxysmal tachycardia. This no longer holds good as A-V block is found in auricular tachycardia as well (Barker *et al.*, 1943; Decherd *et al.*, 1943; and Evans, 1944). It was found in two instances (Cases 3 and 6) in the present series. Chest leads proved to be more reliable in demonstrating auricular activity than the limb leads. In our experience chest leads are more useful in the examination of adults than of babies and infants. The comparatively small size of the heart and chest wall in this early age of life seems to be the reason for the difficulty in applying an electrode to a selected area of the heart. Fig. 6 shows that a chest lead might reveal auricular activity while there are no certain signs of auricular activity in the limb leads. In most cases, however, the limb leads in babies and infants show the auricular waves as well or in some instances even better than the chest leads. Lewis (1912) and Carr (1932) found that paroxysmal tachycardia may change into auricular flutter, which suggests some relationship.

Ventricular paroxysmal tachycardia. There were three records in the present series. The duration of QRS is prolonged, and QRS is not accompanied by P waves. The configuration of the ventricular deflections is such that it may be difficult to distinguish the QRS from the S-T-T.

Nodal paroxysmal tachycardia. Three records were found in the present series. The ventricular rate varies between 150 to 160 a minute.

Sinus tachycardia is readily distinguished from paroxysmal tachycardia by the presence of P waves and a normal P-R interval which may normally be of 0.08 sec. duration in babies and infants.

Pathological anatomy and histology. It is remarkable that distinctive pathological changes either in the myocardium or in the conductive tissue have not been found in three cases of the present series, where an autopsy was performed. Two were cases of paroxysmal auricular

tachycardia or flutter with 2:1 block, one of paroxysmal ventricular tachycardia. The pathological findings in these three showed no evident abnormality, no trace of myocarditis or valvulitis, though the appearances pointed to an acute and intense cardiac failure in one instance. This is of great practical importance; it is obvious that paroxysmal tachycardia has to be diagnosed during life or it will not be diagnosed at all, the post-mortem examination showing no pathological changes.

The prognosis is grave in infants and very grave in cases in which the underlying infection is severe, e.g. in diphtheria of gravis type. In milder cases, the attack may stop spontaneously.

Treatment. Digitalis given promptly may have an effect in some instances, and save the child's life. Koplik had this impression in one of his cases. Hubbard reports very good results. The treatment with digitalis had no chance in the diphtheria cases of the present series. In only one (Case 2) was the result satisfactory. In a case of meningitis (Case 10), flutter was changed into fibrillation and this came under control by digitalis treatment. The results with other drugs recommended for treatment of paroxysmal tachycardia, e.g. quinidine and magnesium sulphate intravenously are also not always satisfactory. Further, it was found that pressing on the carotid sinus or the eyeball, which may stop an attack in an adult, were not effective in infants.

This report gives a rather poor prognosis for paroxysmal tachycardia in infancy and childhood. It must not be forgotten, however, these investigations were carried out on children admitted with or suspected of severe acute infective diseases. Where a mild infection may have been the causative factor the response to treatment, and thus the prognosis is probably much better.

SUMMARY

Paroxysmal tachycardia occurs more often in infancy and childhood than is commonly thought, and it accounts for some instances of sudden and unexplained death in infancy.

Clinical and cardiographic signs of eleven cases and pathological anatomical and histological findings of three autopsies are reported.

Prognosis and treatment are discussed.

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