PAROXYSMAL TACHYCARDIA IN INFANCY

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Since Bristowe (1887) first described a series of cases of 'recurrent palpitation of extreme rapidity in persons otherwise healthy', paroxysmal tachycardia in adults has become a well-known condition. In children the condition has been considered rare. Taran and Jennings (1937) in a review of the literature found only 52 cases under the age of 15 from 1892 to 1935, to which Johnson (1940) added 11. Wright (1938) was able to discover two cases out of 60,000 admissions to a children's hospital, and Landtman (1947) five in 5,600 children examined at his heart clinic. Neubauer (1945), one of the few British contributors to the subject, described 11 cases.

In infancy, sporadic cases have appeared from time to time, but the condition was regarded as an extreme rarity until Hubbard (1941) detailed nine cases seen by him in the period of one year and reviewed 19 others in the literature. Since then the condition has been recognized with increasing frequency, and has even been noted before birth (Garvin and Kline, 1947; Stadler, 1948).

A review of those cases that have been published reveals a uniform clinical picture. It is the purpose of this paper to describe this condition and to give a typical example.

Aetiology

Though in older children infections and digitalis poisoning are often associated with paroxysmal tachycardia, in infancy, in the majority of cases, no underlying cause can be found. A congenital defect of the heart, however, is not a rare finding as may be suspected on clinical grounds in the cases of Colegate and McCulloch (1926), Carr and McClure (1931) and as was found on necropsy in the cases of Schuster and Paterson (1924), Werley (1925) and Young (1944). The majority of post-mortem examinations reveal no cardiac abnormality though Shookoff, Litvak and Matusoff (1932) maintain that there is usually underlying myocardial disease.

That acute infections can be associated with paroxysmal tachycardia in infancy is illustrated by the case of Lyon (1937) when it accompanied

an attack of streptococcal meningitis, and in Neubauer's (1945) series where it occurred in meningococcal meningitis, pertussis and in an infant probably suffering from scarlet fever. Taran and Jennings (1937) emphasize the importance of measles and whooping cough.

Though the majority of attacks seem to start for no apparent reason, in individual cases certain specific stimuli or infections seem to have been of importance; diarrhoea (O'Flynn, 1925; Farr and Wegman, 1935), smiling or being tickled (Poynton and Wyllie, 1926), a cold (Van Cleve, 1930). Relapses following a first attack seem to be brought on by any stimulus which raises the pulse rate but in infancy particularly by feeding and defaecation.

The Nature of the Abnormal Rhythm

Campbell (1947) has pointed out that there are four varieties of paroxysmal tachycardia; supra-ventricular auricular and nodal, auricular fibrillation, auricular flutter and ventricular paroxysmal tachycardia. All these varieties have occurred in young children. The great majority of cases, however, are supraventricular in origin as is pointed out by Johnson (1940) in his review, and as is confirmed by those cases published since. Great difficulty occurs in distinguishing between paroxysmal tachycardia in infancy and paroxysmal flutter, since the infant's ventricle seems to be able to respond to a very high rate of stimulus without heart block appearing. A rate of 365 per minute has been recorded by Silverman and Race (1949). The rhythmical variation in the base line of the electrocardiogram is difficult to see. It is suggested by Frisell (1946) that in the newborn infant a rate over 300 per minute is more likely to be due to auricular flutter, and one below 300 to be paroxysmal tachycardia, but there are exceptions to this rule. The majority of cases are therefore designated as supraventricular paroxysmal tachycardia.

Clinical Picture

Whereas in adults and older children the complaint may be no more than a nuisance, in infants

the extreme rapidity of the heart rate leads to progressive heart failure. The infant usually presents as a case of sudden dyspnoea. The respiratory rate may rise to as much as 90 in the minute. Cyanosis, at first slight and easily missed in a poor light, becomes progressively more marked. The lungs are filled with moist râles, the liver and sometimes the spleen enlarge progressively. Associated with this dyspnoea is often some degree of fever, usually not above 102° F., and a leucocytosis of about 20,000 per c.mm. Radiological examination shows patchy opacities throughout the lung fields and usually an enlarged heart. It is not surprising, therefore, that a diagnosis of bronchopneumonia is made, and sufficient attention not paid to the tachycardia, either because the heart sounds may be inaudible due to the lung adventitia, or because the tachycardia is attributed to infection. This is the common picture in early infancy, and is seen in the cases described by Sargent and Gillespie (1949), Schieve (1949), Silverman and Race (1949), Moore (1948), Mannheimer (1946), Clarke (1935) and O'Flynn (1925). More rarely, the paroxysm appears to be associated with diarrhoea, vomiting and abdominal pain, and an alimentary infection may be suspected. When consciousness is lost, as is sometimes the case, disease of the central nervous system may be suspected, and finally, due to the large heart, congenital idiopathic hypertrophy of the heart may be suspected.

Such dramatic symptoms are not always seen in infancy, and in many of the cases described at least some of the attacks have caused slight or no symptoms at all. Russell and Ellison's (1927) patient had a tachycardia of 240 per minute in the second attack and had no symptoms, but he was aged 15 months. In early infancy in general symptoms appear to be marked.

Treatment

As opposed to experience with older children and adults, physical methods of bringing the attack to an end are very rarely successful. Drug therapy is usually required and the consensus of opinion favours digitalis. Thus, Gibson (1950) found the drug effective in 10 infants out of 12. Hubbard (1941) treated nine infants with digitalis with success. Campbell (1937), Hobbs (1941), Howard (1945), Hedberg (1945), Frisell (1946), Scott and Limper (1946) and Sargent and Gillespie (1949) have all reported success with the same drug. Garvin and Kline (1947) in a newborn infant found that digitalis gave incomplete control but the addition of quinidine was successful. A

contra-indication to digitalis in the opinion of Silverman and Werner (1950) is the Wolff-Parkinson-White syndrome, since the depression of the auriculo-ventricular node due to digitalis diverts the cardiac impulse through the aberrant pathways and aggravates the condition, whereas quinidine is successful in controlling the condition and rendering the electrocardiogram normal. Schieve (1949) considered digitalis to be effective in an infant with this syndrome, but since control was not obtained for three days it is possible that the remission was spontaneous. Bloom and Kendig (1946) expressed the opinion that if the physician has courage enough the attack can be left to terminate spontaneously. That this occurs is undoubted, but that it cannot be relied upon with safety is indicated by the fatal termination in some untreated cases (Werley, 1925; Edeiken, 1943; Silverman and Race, 1949). It is generally agreed that digitalis is contra-indicated in attacks of ventricular paroxysmal tachycardia in which quinidine is effective. The dosage of digitalis which has been found effective in infants has varied; Gibson (1950) recommends 50 mg. digitalis followed by 25 mg. four-hourly, and has given up to 300 mg. in an 8 lb. baby. For intravenous and intramuscular use, 'digifoline' (1 ml.=100 mg. of international standard digitalis powder) has commonly been used in a dose of from $\frac{1}{2}$ to 1 ml. repeated at four- to 12-hourly periods. Moore (1948) recommends 0.2 ml. of the tincture per kilogram body weight.

The value of quinidine is disputed. The experience of Garvin and Kline (1947) has already been quoted. Moore (1948) found this drug effective in an infant who had 31 attacks between the eighth day of life and the age of 5½ months after digitalis and mecholyl had become ineffective. A dose of 120 mg. two-hourly by mouth had to be given and eventually raised to 200 mg. Stadler (1948) in a newborn infant with auricular flutter succeeded in reducing the heart rate from 240 to 120 per minute by giving $\frac{1}{4}$ grain four-hourly, but had to abandon the drug because of vomiting. Digitalis was substituted with success. Sargent and Gillespie (1949) found the addition of quinidine, grains 2 three times a day, prevented the recurrence of tachycardia in an infant aged 6 weeks already under the influence of digitalis. Gibson (1950) recommends the drug for prophylactic use. As opposed to these opinions, Limper (1949) had one infant who had 42 attacks in the 68 days he was observed in hospital and in whom both digitalis and quinidine were of no use prophylactically, and Wright (1938), who followed an infant from the

age of 9 months to 8 years, found neither quinidine nor digitalis of any value in prevention.

More recently, certain parasympathomimetic drugs have been used with success. Mecholyl (acetyl- β -methylcholine) introduced by Starr (1933; 1936) was used by Hubbard (1941) in an infant aged 3 weeks, to whom he gave 5 mg. subcutaneously with alarming results. Moore (1948) gave 0.5 mg. subcutaneously to an infant of 45 days which successfully ended the paroxysm, but also led to such alarming side-effects that atropine had to be given. Following a relapse at the age of 12 weeks, 0.25 mg., 0.5 mg. and 1.5 mg. were given without effect at 30-minute intervals, but after 2 mg. were given the attack was controlled; the side-effects, however, were so marked that it was thought the baby might die. They were again brought to an end with atropine. Brownlee, Waters and McClendon (1950) found in an infant aged 10 weeks that 1 mg. repeated once subcutaneously was effective. Certainly, if this drug is to be used, atropine must be available to control its effects; it should never be given intravenously. Less violent in effect, but acting in a similar manner, is acetylcholine which Philipsborn and Gibson (1948) found effective when given intravenously in a dose varying from 1 to 4 mg. Cunningham and Schnitzker (1950) gave 0.2 mg. intravenously to a child of 9 months and successfully ended the paroxysm. Landtman (1947) found prostigmine $\frac{1}{8}$ mg. effective in five infants, and Mannheimer (1946) neostigmine $\frac{1}{8}$ mg. subcutaneously in an infant aged 3 weeks.

Recently, neosynephrine (laevo- α -hydroxy- β -methyl amino-3-hydroxy-ethylbenzene hydrochloride) has been used in adults (Youmans, Goodman and Gould, 1947), and in a child aged 27 months by Cunningham and Snitzker (1950) who recommend giving 0.1 mg. intravenously and increasing by 0.1 mg. each 30 minutes up to a dose of 0.5 mg. The drug acts by causing a sudden rise in blood pressure, but no reference to its use in infants has been found.

Prognosis

The Individual Attack. This is more serious in an infant than in an older child or adult, but as in the latter, depends mainly on the presence of underlying cardiac disease.

Future Attacks. Though many infants have recurrent attacks, there is in infants a greater likelihood of the attacks ceasing completely than in older children or adults.

Case Report

P.B., the fourth child of a healthy family, was born at home, weighing 8 lb. The delivery was normal and the mother had been well during her pregnancy save for pyelitis in the early months. For the first 24 hours the baby was noticed to be blue and no attempt was made at feeding. On the second day of life he seemed well, but from the third day he suffered from recurring attacks of blueness which appeared to be precipitated by feeding or straining at stool. These attacks lasted about five minutes and were associated with dyspnoea and gasping respirations. He was first seen on the eleventh day of

FIG. 1

FIG. 2

FIG. 3

life. Gross dyspnoea was present, the respiratory rate being 90 per minute, and there was slight cyanosis of the lips. He was afebrile, and the pulse rate counted with the stethoscope was 240 per minute. It was difficult to be certain of the latter as there were profuse moist sounds throughout the lung fields which tended to obscure the heart sounds. The heart was enlarged, the apex beat being in the anterior axillary line. The liver was enlarged down to the umbilicus, and there was pitting oedema of both legs. A provisional diagnosis of paroxysmal tachycardia was made, and he was admitted to hospital where an electrocardiogram was taken (Fig. 1). 'Digoxin', 0.125 mg., was given immediately by mouth. Six hours later the heart rate had fallen to 140 per minute and never rose above 150 per minute again. Digoxin was continued in a dose of 0.025 mg. eight-hourly for 48 hours, then 12-hourly for another 48 hours. An electrocardiogram taken 48 hours after the beginning of treatment (thirteenth day of life) showed the heart rate to be 120 per minute with evidence of the effect of digitalis (Fig. 2), and the record was essentially similar 48 hours later. Within 24 hours of the beginning of treatment the respiratory rate had fallen to 65 per minute, slight enlargement of the liver was still present and oedema of the legs still detectable. Within 48 hours no enlargement of the heart could be noted, and all abnormal signs had disappeared. The respiratory rate was elevated for five more days and then fell to a normal figure. On the second day of treatment, while the oedema was diminishing, looseness and frequency of stools occurred. At no time was a cardiac murmur noted, and on the fifty-eighth day of life an electrocardiogram revealed a normal record for his age (Fig. 3). At the age of 9 months he had developed normally and had had no further attacks of paroxysmal tachycardia, and no abnormalities could be detected clinically.

Summary

A case of paroxysmal tachycardia in infancy is described and the incidence, aetiology, treatment

and prognosis discussed with reference to previous cases described in the literature.

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REFERENCES

- Bloom, N. and Kendig, E. L. (1946). *J. Pediat.*, **28**, 474.
 Bristowe, J. S. (1887). *Brain*, **10**, 164.
 Brownlee, W. M., Waters, E. M. and McClendon, S. J. (1950). *Amer. J. Dis. Child.*, **79**, 838.
 Campbell, M. (1947). *Lancet*, **2**, 641.
 Carr, J. G. and McClure, W. B. (1931). *Amer. Heart J.*, **6**, 824.
 Clarke, T. W. (1935). *Arch. Pediat.*, **52**, 666.
 Colgate, C. E. and McCulloch, H. (1926). *Amer. Heart J.*, **2**, 160.
 Cunningham, G. C. and Schnitzker, W. F. (1950). *J. Pediat.*, **37**, 727.
 Edeiken, J. (1943). *Amer. J. med. Sci.*, **205**, 52.
 Farr, L. E. and Wegman, M. E. (1935). *Ibid.*, **190**, 22.
 Frisell, E. (1946). *Acta paediat., Uppsala*, **34**, 30.
 Garvin, J. A. and Kline, E. M. (1947). *Amer. Heart J.*, **33**, 362.
 Gibson, S. (1950). *Med. Clin. N. Amer.*, **34**, 217.
 Hedberg, G. T. (1945). *Acta obstet. gynec. scand.*, **25**, 392.
 Hobbs, L. F. (1941). *Amer. Heart J.*, **21**, 804.
 Howard, P. J. (1945). *J. Pediat.*, **26**, 273.
 Hubbard, J. P. (1941). *Amer. J. Dis. Child.*, **61**, 687.
 Johnson, G. D. (1940). *Ibid.*, **60**, 1137.
 Landtman, B. (1947). *Acta paediat., Uppsala*, **34**, Suppt. 1, p. 57.
 Limper, M. A. (1949). *Amer. J. Dis. Child.*, **77**, 753.
 Lyon, J. A. (1937). *J. Amer. med. Ass.*, **108**, 1393.
 Mannheimer, E. (1946). *Acta paediat., Uppsala*, **33**, 383.
 Moore, R. L. (1948). *Pediatrics*, **2**, 266.
 Neubauer, C. (1945). *Brit. Heart J.*, **7**, 107.
 O'Flynn, J. L. (1925). *Brit. med. J.*, **1**, 507.
 Philipsborn, H. F. and Gibson, S. (1948). *Pediatrics*, **1**, 205.
 Poynton, F. J. and Wyllie, W. G. (1926). *Lancet*, **2**, 371.
 Russell, H. B. and Ellison, J. B. (1927). *Ibid.*, **2**, 546.
 Sargent, F. K. and Gillespie, J. B. (1949). *Arch. Pediat.*, **66**, 437.
 Schieve, J. F. (1949). *Amer. J. Dis. Child.*, **77**, 474.
 Schuster, N. H. and Paterson, D. (1924). *Proc. roy. Soc. Med.*, **17**, (Sect. Stud. Dis. Child., p. 11).
 Scott, E. P. and Limper, M. A. (1946). *J. Pediat.*, **28**, 96.
 Shookhoff, C., Litvak, A. M. and Matusoff, I. (1932). *Amer. J. Dis. Child.*, **43**, 93.
 Silverman, J. J. and Race, O. M. (1949). *Amer. Heart J.*, **37**, 1139.
 — and Werner, M. (1950). *J. Pediat.*, **37**, 765.
 Stadler, H. E. (1948). *Ibid.*, **33**, 624.
 Starr, I. (1933). *Amer. J. med. Sci.*, **186**, 330.
 — (1936). *Ibid.*, **191**, 210.
 Taran, L. M. and Jennings, K. G. (1937). *Amer. J. Dis. Child.*, **54**, 557.
 Van Cleve, M. (1930). *J. Amer. med. Ass.*, **94**, 1758.
 Werley, G. (1925). *Arch. Pediat.*, **42**, 825.
 Wright, F. H. (1938). *Amer. J. Dis. Child.*, **56**, 1334.
 Youmans, W. B., Goodman, M. J. and Gould, J. (1947). *Proc. Soc. exp. Biol., N.Y.*, **64**, 380.
 Young, J. H. (1944). *Med. J. Aust.*, **1**, 538.