PAROXYSMAL TACHYCARDIA IN INFANCY

BY

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The occurrence of paroxysmal tachycardia in childhood has been noted in case reports for over twenty years. Cases were reported in the English press in 1924 and 1925, but after this interest seems to have lapsed until 1941 when a review of the subject, with special reference to infancy, was made by Hubbard (1941) with a report of nine cases. Full references to the literature up to that year are to be found in his paper and in a paper by Hobbs (1941) reporting another case. Subsequently case reports have been published by Blackford and Hoppe (1943) and by Hubbard and Starback (1943).

Hubbard differentiates cases in infancy from those in older children, because of what he believes to be a characteristic syndrome in young infants, consisting of fever, leucocytosis, dyspnoea, pallor, vomiting, and dilated heart with signs of congestive failure. Those of his cases which were cardiographed showed either auricular or nodal tachycardia, with ventricular rates varying from 220 to 305, but his 1943 case had a ventricular rate of 350. Although he believes the condition to be more common than has been recognized, it is sufficiently rare for it to be unusual for clinicians with many years of practical experience in children's work to have observed a case.

Clinical record

A girl baby born on August 13, 1944, the fifth child of apparently healthy parents, was admitted to the Royal Northern Infirmary on October 12, 1944. The pregnancy was full term and the mother was healthy throughout. The first child, born in 1939, a boy, is healthy. The second died in hospital in Dublin 'of a heart attack' at the age of six weeks. It has unfortunately not been possible to trace the records of this child. The third, born in 1941, a girl, is healthy. The fourth child, a boy, now aged 1½ years, had been admitted to the Royal Northern Infirmary for a few weeks in August, 1944, for observation. There had been feeding difficulties with this boy (D.O'R.) when he was a few weeks old, which had necessitated his admission to hospital in Ireland. Subsequently he made excellent progress, and weighed 22 lb. at ten months when he had also learned to stand up, when he began to vomit and in a few weeks had lost 4 or 5 lb. in weight. He was reported as peculiar in behaviour, burying his face in the pillow, very fretful, and reluctant to

feed. This state continued until he was examined at hospital on August 8, 1944, aged thirteen months. He was apathetic, hypotonic and disliked being handled. There were no signs of meningitis, nor any focal central nervous system signs. Tendon jerks were present: there was no photophobia, and no rash except on the buttocks. He was afebrile. His heart rate was 140-160 at this examination, but subsequently in the ward 100-120. Blood pressure 75/50. Blood sedimentation rate (capillary method) 13 millimetres at one hour. R.B.C. 4.79 million per c.mm., Hb. 85 per cent., W.B.C. 6,000 per c.mm. He gained weight, was trained to a mixed diet, resumed his progress and a tentative diagnosis of neurosis was made. He was readmitted after the death of his infant sister because of a recurrence of symptoms on November 14, 1944, aged eighteen months, weighing 20 lb. No fresh signs were discovered. His mother described his behaviour as suggesting an aversion to her.

Two children born to the father's brother are reported to have died rather suddenly in infancy.

The baby who is the subject of this case record was bottle fed and made steady progress until October 10, 1944, when she was discovered by her mother in her cot looking 'suffocated.' She was red in the face, sweating and gasping for breath. This condition obtained for about two hours when she improved, was given a feed and vomited. Subsequently she appeared to have spasms of pain, turning her head from side to side and pulling up her legs. She was restless all night and the following day breathing was laboured; she was fretful, was reluctant to take feeds, and vomited several times. The stools, which had previously been normal, now appeared to be undigested, and an umbilical hernia not previously noticed became evident when she was crying.

Examination. Shortly after admission the following note was made of her condition. General state of nutrition good. Eyes are sunken. Slightly cyanosed. Fontanelle normal. Respiration quick but not distressed. Temperature 105° F. Respirations 38. No physical signs of pneumonia. Buttocks raw. Offensive intestinal gas and undigested stool. Heart rate 240 with regular rhythm. No extrasystoles and no murmurs. General muscle tone normal. No head retraction nor other evidence of focal disease in nervous system. Total white cell count 20,000 per c.mm. The cardiogram (fig. 1) showed auricular tachycardia, rate 240, with right axis deviation. The heart rate was not

FIG. 1

influenced by compression of carotid or eyeball, or by alterations of posture.

The question of digitalis or quinidine therapy was debated, but it was decided to withhold both overnight until further experience of the child's condition had been gained, since there were no present signs of congestive failure. Three grains of chloral hydrate were prescribed followed by 1 grain every four hours and, because of the evidence suggesting an acute infection, sulphadiazine 0.5 gm. followed by 0.25 gm. every four hours.

On October 13, 1944, the infant appeared much better, was ready to take feeds (saline with corn syrup only), did not vomit, and the heart rate was slower. This improvement continued until the evening of October 14, 1944, the heart rate then being 160, when without any resumption of the tachycardia, a deterioration in the baby's condition set in. Feeds were again refused, and the infant appeared hypersensitive, starting at the slightest noise, and appearing distressed when touched or disturbed in any way. Head retraction with a tendency to opisthotonos appeared and intermittent squint. The spinal fluid by lumbar puncture was diffusely blood-stained and from cisternal puncture likewise. There was no excess of white cells: the Wassermann reaction was negative. The general condition now rapidly deteriorated, and the baby died.

Post-mortem examination. The autopsy showed an apparently normal heart, with no enlargement or signs of infection. No evidence of congestive failure in liver, spleen, kidneys or lung. There was slight atelectasis of the right lower lobe (an area which had been questioned on clinical examination as showing early signs of bronchopneumonia). Brain and meninges were apparently normal. There was no evidence of scurvy or rickets. The general report on the post-mortem examination was (Dr. A. Dick) — 'Nothing from the post-mortem examination to account for the tachycardia. No evidence of any infective process or encephalitis found.'

HISTOLOGICAL EXAMINATION (Dr. H. J. Kirkpatrick). Heart (tissue from neighbourhood of interventricular septum). There is cloudy swelling and oedema. Congestion of vessels, but no inflammatory infiltration.

Brain. Vessels are congested and there is some oedema. In one or two places there is a little capillary extravasation of blood—probably not significant. No evidence of inflammatory process.

Comment

This infant did not have the signs of heart failure either in life or at the autopsy, nor did the mode of

death suggest that the heart was the organ the function of which primarily failed. On the contrary, the whole syndrome in its latest stages was one of an acute encephalopathy. Hubbard suggests that the leucocytosis and fever in his cases were to be explained not on a toxic or infective basis, but as a direct result of heart failure and due to congestion of the lungs. It is impossible to accept such an explanation in this case. It is noteworthy that in one of his subjects, aged two weeks, the spinal fluid was examined and found to be xanthochromic and to contain red cells; this infant was readmitted to hospital at the age of four weeks with a recurrence of tachycardia, and again found to have a blood-stained spinal fluid. That this was not the only patient in whom symptoms suggestive of encephalopathy occurred, is shown by the fact that in another case meningitis had been diagnosed by the practitioner before admission to hospital, the baby being described as 'comatose.' It is, of course, possible that the haemorrhage into the spinal fluid in both Hubbard's case and the present one was traumatic, but it seems unlikely. The experienced operator recognizes fairly easily the difference between the haemorrhagic fluid which is the result of a disease process and one which has been caused by himself. In the present case, the blood cells were uniformly mixed with fluid which appeared haemorrhagic from the first, and cisternal puncture was done immediately, the child's head being a little raised throughout, the appearance of the fluid from the cistern being exactly the same as that obtained from the lumbar region. In Hubbard's case the spinal fluid was haemorrhagic on both occasions and was xanthochromic on the first occasion. The post-mortem appearances failed to disclose any gross meningeal haemorrhage, and it must be assumed that the blood cells had reached the spinal fluid by process of diapedesis. In so far as any positive conclusions could be drawn from the post-mortem examination, the picture resembled one of a toxic encephalopathy, not of any infective process.

In a paper entitled 'Human-Milk Intoxication,' Dr. Lydia Fehily (1944) describes a syndrome, often fatal in Chinese infants, which she considers due to intoxication by methylglyoxal and other products of incomplete carbohydrate metabolism in the breast milk of mothers suffering from beriberi.

The acute syndrome she describes as one of 'vomiting, abdominal pain, diarrhoea, abdominal distension, stiffness of the neck and extremities, and convulsions. In the most acute form the attack consists of dyspnoea, cyanosis and running pulse. The attacks often end fatally, but in case of survival the infants pass into the chronic stage with symptoms of oedema, oliguria, aphonia, constipation, meteorism, neck retraction, enlargement of liver and right side of heart, loss of weight, retarded growth and marasmus.'

She suggests that the syndrome she describes is due to acute milk intoxication, but her reasons do

not amount to proof with any experimental work, e.g. the production of symptoms demonstrated by the giving of milk from a beriberi mother to the baby of an apparently healthy mother. She suggests that this syndrome is a possible explanation of cases of sudden dyspnoea and cyanosis, or of sudden death in breast-fed infants in the western hemisphere.

I have seen at least one attack of this kind in a breast-fed baby whose temperature reached 107° F., and in whom no definite physical signs were observed. This baby was already weaned when I saw it at the age of six days, but had been breast fed at the time that the attack occurred. There is, of course, no question of the whole series of cases of paroxysmal tachycardia which has been reported in the press from time to time and now numbers twenty or

thirty, being accounted for by such an explanation as that offered by Fehily for her cases, nor does she specifically mention rapid heart rates as characteristic of the syndrome she is describing. The resemblance nevertheless between the two types of syndrome: (a) Dr. Fehily's infantile 'intoxication,' and (b) the syndrome of tachycardia with fever, leucocytosis, and symptoms suggestive of encephalopathy, is close enough to be worthy of remark.

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