PAROXYSMAL TACHYCARDIA IN A CHILD SHOWING
STOKES-ADAM AND WOLFF-PARKINSON-WHITE
SYNDROMES

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Paroxysmal tachycardia in infancy may be 
accompanied, as in the adult, by some disturbance 
giving rise to pallor, restlessness, dyspnoea, weak-
ness and usually cardiac insufficiency and decomp-
sensation. A labile autonomic nervous system 
makes infants particularly vulnerable to paroxysmal 
forms of tachycardia. Exactly what it is that starts 
this disorder or rhythm is frequently obscure and 
consequently functional autonomic disturbances 
must often be assumed to be responsible.

The Stokes-Adam syndrome seen in adults in the 
course of the development of complete heart block 
is not often seen in children and the case described 
below, occurring in a child of 2 years during an 
attack of supra-ventricular paroxysmal tachycardia, 
is of unusual interest. The symptoms of cardiac 
insufficiency, more commonly seen in such cases, 
were conspicuous by their absence in this instance.

Case Report

A boy (P), aged 2 years and 3 months, was admitted to 
the Lady Ridgeway Hospital for Children, Colombo, at 
9 a.m. on January 14 with a history of 10 to 15 'fits' a day. 
He had had two fits in the out-patients' department while 
awaiting admission, each fit lasting one to two minutes. 
The previous history was that the child was healthy after 
a normal delivery. There was no illness of any impor-
tance before these fits. He had had similar fits when 
8 months old for four days, each fit lasting about a 
minute, and there were four to five fits each day. A 
similar attack of fits occurred three months later when 
the child was admitted to this hospital for a few days. 
He was discharged and had been admitted again subse-
quently to this hospital and to a branch hospital at 
Ragama. Each time he was discharged after a few days 
with a diagnosis of fits due to round worms and, on one 
occation, epilepsy. The mother stated that the fits were 
not associated with any fever. The child became semi-
conscious for a few seconds before the fit, and appeared 
to be quite normal afterwards.

On examination at about 9.30 a.m. the child appeared 
to be a healthy well-fed boy weighing 22 lb. (9·98 kg.). 
There were slight pulsations over the veins in the neck. 
The heart was within normal limits and there were no 
murmurs. The rate was very rapid—about 220 per 
minute—and the pulse was regular and very small in 
volume and tension. There was no cyanosis nor 
dyspnoea and the child was seated on the bed.

The skin turgor was normal and the fontanelles were 
closed. There was no spasticity of the limbs or neck. 
The cranial nerves were normal. The abdomen was soft 
and there were no masses palpable. The liver and spleen 
were not palpable. While this examination was going 
on the child quite suddenly became very pale and fell 
back on the bed in a faint. He remained so for about 
30 seconds. He next had a convulsive fit involving both 
lungs, lasting about one and a half minutes, and soon 
regained consciousness; he then appeared to be quite 
normal in colour and behaviour. It was noticed that 
during the faint there were missed beats and the heart rate 
was very slow, about 60 to 64 per minute. The child sat 
up in bed a few minutes later and seemed to be quite 
normal and unconcerned with what had happened a few 
minutes earlier. The heart rate was now very rapid and 
orose to 140 and later 200 per minute. An 
ecocardiogram was taken soon after and proved very interesting, 
as the child had a similar faint and fit while this record 
was being taken. In fact the record was interrupted 
during the fit for a minute or so. The recording proved 
an interesting study. Fig. 1 shows a paroxysmal 
tachycardia of the supra-ventricular type at a very rapid 
rate of 260-270 per minute. There are short periods of 
complete cardiac-standstill varying from 0·6 to 0·8 sec. 
It was noticed that the child fainted off during this 
period of asystole and when he recovered a few moments 
later the rhythm was slow and irregular at first but later 
was quite regular and rapid and soon reached a rate of 
240 per minute. While the E.C.G. was being examined 
the child had a similar faint and fit and appeared to be 
quite normal soon after the fit. He was treated with a 
sedative syrup of chloral (gr. 3 in 1 drachm) 6-hourly 
and Tab. Digoxin 0·25 mg. 4-hourly from 10 a.m. The 
pulse rate continued to be over 200 for 24 hours, but 
12 hours later it slowed down as shown in the table.

The child continued to have fainting attacks and short
fits about three or four times during this period but was generally comfortable and playing in bed.

On January 17, after discontinuing the Digoxin, another electrocardiogram was taken (Fig. 2). It is seen that Lead I starts off with eight normal beats but the ninth is absent while the PR interval of the tenth beat is 0.08 sec. This part of the record shows that every second or third beat has a short PR interval and that each of these beats is preceded by a dropped beat. The shortened PR interval varies from 0.02-0.08 sec. (the normal in infants being not less than 0.12 sec.). The QRS complex is widened 0.11 sec. with slurring of the upstroke of the R wave (the normal in infancy being 0.05 to 0.06 sec.). There are therefore on this record complexes of the Wolff-Parkinson-White syndrome type, each being preceded by a dropped beat. The slurring and widening of the QRS complex with short P waves indicates the presence of the Wolff-Parkinson-White syndrome without any doubt. The E.C.G. records on January 23 (Fig. 3) and February 17 (Fig. 4) showed a second or third beat has a short PR interval and that each of these beats is preceded by a dropped beat. The shortened PR interval varies from 0.02-0.08 sec. (the normal in infants being not less than 0.12 sec.). The QRS complex is widened 0.11 sec. with slurring of the upstroke of the R wave (the normal in infancy being 0.05 to 0.06 sec.). There are therefore on this record complexes of the Wolff-Parkinson-White syndrome type, each being preceded by a dropped beat. The slurring and widening of the QRS complex with short P waves indicates the presence of the Wolff-Parkinson-White syndrome without any doubt. The E.C.G. records on January 23 (Fig. 3) and February 17 (Fig. 4) showed a
normal picture with normal rhythm. On January 26 the child had two short fainting attacks without any convulsions and Digoxin was given again for four days. The child had another faint on February 2 and on February 17, after being quite well and happy, he left us. A follow-up two years later showed the child to be in good health, with a heart normal in rhythm and rate.

Comment

The case recorded is that of a 2-year-old child who had a paroxysmal tachycardia of the supraventricular type and showed fainting attacks followed by fits very similar to the Stokes-Adam syndrome seen in adults with heart block. A further interesting feature was that on recovering the child showed the presence of the Wolff-Parkinson-White syndrome in the electrocardiogram. This latter syndrome is an electrocardiographic entity and has been the subject of much discussion and study since it was originally described by Wolff, Parkinson and White (1930). Gleckler and Lay (1952) have described the Wolff-Parkinson-White syndrome in a 4-month-old infant with paroxysmal tachycardia in whom an electrocardiogram made on cessation of the arrhythmia showed the presence of the syndrome. The interest in this case is that, besides the Wolff-Parkinson-White syndrome, this child also showed the presence of Stokes-Adam while in hospital.

REFERENCES