British Heart Journal, 1975, 37, 339–344.

Bidirectional tachycardia in a child
A study using His bundle electrography

D. S. Reid, Michael Tynan, L. Braidwood, and G. R. Fitzgerald
From the Department of Cardiology, Newcastle General Hospital, Westgate Road, Newcastle upon Tyne

This is a report of a case of bidirectional tachycardia in a 6-year-old girl with no evidence of any structural abnormality of the heart. The patient had never received digitalis. The arrhythmia appeared to be precipitated by effort and emotional stress, and could be induced by increasing the heart rate by atrial pacing or isoprenaline administration. His bundle electrography showed that the arrhythmia was ventricular in origin. This emphasizes the importance of recording an effort electrocardiogram in all children with unexplained syncopal episodes, even when the resting electrocardiogram is normal.

Bidirectional tachycardia is an uncommon rhythm disturbance, the origin and mechanism of which are still disputed. In the majority of cases it occurs as a manifestation of digitalis toxicity or in the presence of severe myocardial disease. This report describes the electrophysiological and haemodynamic findings in a 6-year-old girl with bidirectional tachycardia not caused by digitalis, but precipitated by effort and emotion.

Case report

A 6-year-old girl was initially seen at another hospital in June 1973 after sudden loss of consciousness which had lasted for 10 minutes. Examination at that time was normal. A further episode occurred in August 1973 while she was climbing in a children's playground, and she became pale, limp, and remained unconscious for 10 to 15 minutes. Examination after this episode was again normal but an electrocardiogram recorded after effort showed a rapid arrhythmia. She was admitted to this hospital in November 1973. Examination once more was normal apart from a short grade 2/6 ejection systolic murmur localized to the base of the heart.

An electrocardiogram (Fig. 1) showed sinus rhythm, a PR interval of 0·11 sec, and a normal QRS complex suggesting pre-excitation of the Lown-Ganong-Levine type (Lown, Ganong, and Levine, 1952). The QT_c was 0·42 sec which is on the 75th percentile for a girl of this age (McCammon, 1961), though the observed QT_c

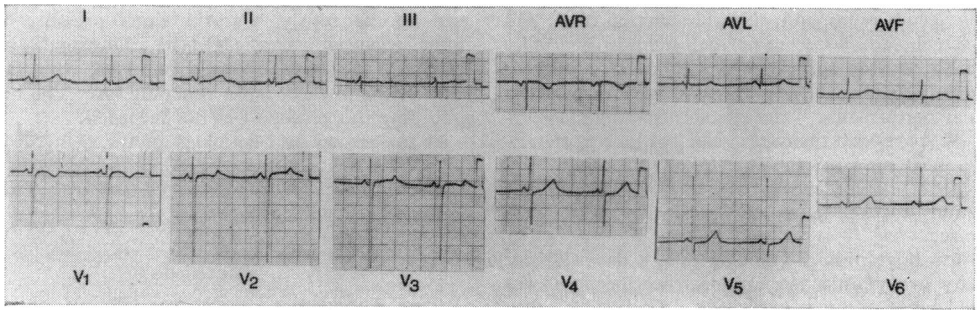

FIG. 1 *Twelve lead electrocardiogram.*

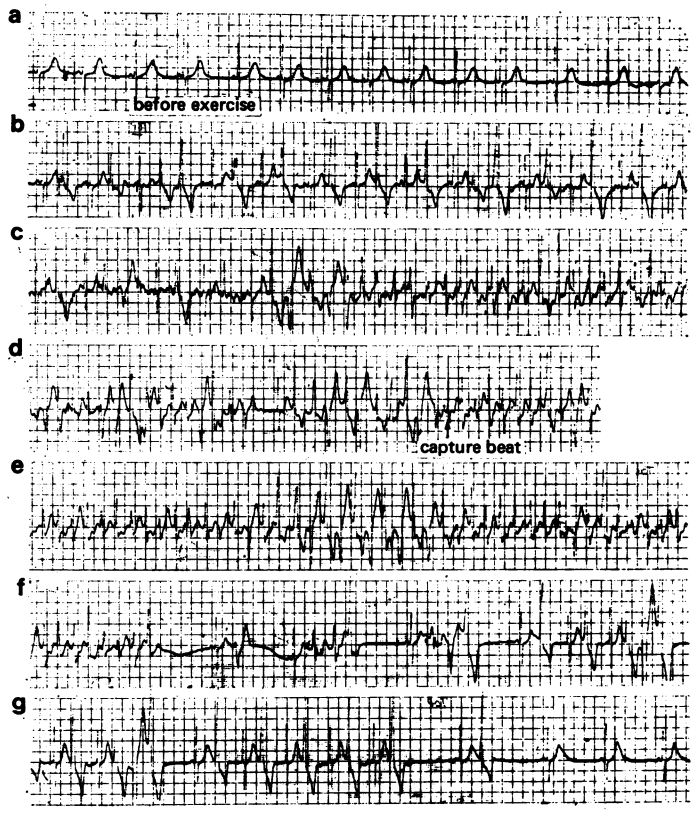

FIG. 2 *Electrocardiogram recorded during exercise: a) immediately before exercise sinus rhythm, b) during exercise frequent ventricular extrasystoles, c) onset of the tachycardia, d and e) during the tachycardia there is considerable variation in the QRS morphology from beat to beat, but in e) there is an episode of bidirectional tachycardia, f) termination of the tachycardia, and g) ventricular extrasystoles persist after termination of the tachycardia, then sinus rhythm without extrasystoles.*

exceeded the QT_c calculated from the formula of Fraser, Froggatt, and James (1964) by 0·05 second. Chest *x*-ray showed a normal cardiothoracic ratio of 52 per cent with normal lung fields. Laboratory findings, including serum potassium, thyroid function tests, and urinary catecholamines, were normal. Echocardiography showed no structural abnormality of the heart and from the echocardiogram of the left ventricle, two indices of left ventricular performance – the ejection fraction (Feigenbaum *et al.*, 1972) and the mean velocity of circumferential fibre shortening (mean VCF) (Fortuin, Hood, and Craige, 1972) – were calculated. These were normal, the ejection fraction being 0·76 and the mean VCF 1·10 circ/sec.

Electrocardiographic monitoring over 5 days did not reveal any arrhythmias so an electrocardiogram was performed while she was exercising on a bicycle ergometer. During this exercise, when the heart rate was 107 beats/min. ventricular extrasystoles occurred followed by a tachycardia with considerable beat-to-beat variation in the QRS morphology, including at times a clearly defined bidirectional tachycardia (Fig. 2). During this the patient did not lose consciousness and the tachycardia terminated spontaneously. Carotid sinus pressure did not influence the tachycardia. On the following day a further brief episode of tachycardia occurred when a doctor approached to carry out a venepuncture. The heart rate preceding this episode was not recorded.

As the nature and aetiology of the tachycardia were not clearly defined, and as the electrocardiogram at rest suggested pre-excitation, it was felt that further investigation was indicated.

Methods

Cardiac catheterization was carried out under basal anaesthesia using gamma-hydroxybutyric acid. After the haemodynamic measurements were recorded, cine-

angiography was performed in the right anterior oblique projection, with injection into the pulmonary artery. From the cineangiogram left ventricular volumes, ejection fraction, and mean velocity of circumferential fibre shortening were calculated by the methods previously described (Tynan *et al.*, 1974).

A quadripolar catheter was positioned in the right atrium, with the distal poles situated in the right atrial-superior vena caval junction. The two distal poles were used for pacing and the proximal two poles for recording the atrial electrogram. A 5 F bipolar (10 mm) pacing catheter was then positioned across the tricuspid valve to record the His bundle electrogram. The atrial and His bundle electrograms and leads I and II were recorded simultaneously on an Elema Schönander ink jet recorder at various paper speeds.

After recording at the resting heart rate, recordings were made during atrial pacing at twice diastolic threshold at gradually increasing rates from 100 to 200 beats/min. Sinus node recovery time was determined after pacing at rates of 100 and 120 beats/min for 2 minutes. Isoprenaline was then given in single injections of 1 μg, 2 μg, and 3 μg.

Results

The haemodynamic measurements, left ventricular morphology, and left ventricular function were normal (Table 1). The components of the His bundle electrogram during sinus rhythm and during atrial pacing are summarized in Table 2. The AH time was shorter than the normal of 60 to 125 msec reported by Roberts and Olley (1972) for children under the age of 7 years, but was within the range of 50 to 105 msec reported by Bekheit and her colleagues (1973) for children of less than 12 years. During atrial pacing the AH interval lengthened, which excludes atrioventricular nodal bypass, but not to the duration normally seen in adults. However, as there are no figures available for children it is difficult to be certain whether this response to pacing is normal or whether it suggests partial AV nodal bypass. The 2:1 post-H block during pacing at 200/min, a response that is not normally seen during atrial pacing in adults, showed that the refractory period of the atrioventricular nodal pathway was shorter than the refractory period of the specialized ventricular conduction system. Sinus node recovery times were normal.

TABLE 1 *Results of cardiac catheterization*

	Pressures		*Oxygen saturation*
	Dynamic	*Mean*	%
Superior vena cava			75
Right atrium	a=6		
	v=3		74
	x=1		72
	y=1	3	71
Right ventricle	20/0–3		73
Main pulmonary artery	14/7	11	74
Pulmonary artery wedge		7	—
Right femoral artery	104/50	70	95

LVEDV = 78 ml/m²
LVESV = 30 ml/m²
Ejection fraction = 0·62
Mean VCF = 1·17 circumferences/sec

TABLE 2 *Measured electrogram intervals during atrial pacing and sinus node recovery time*

Heart rate (beats/min)	*PA (msec)*	*AH (msec)*	*HV (msec)*
79	30	50	35
100	50	50	35
120	50	60	35
130	50	60	35
140	50	70	35
150	50	75	35
160	50	85	35
170	50	85	35
180	50	90	35
200	50	100	35
	and 2:1 post H block		

Sinus node recovery time 120/min = 860 msec
100/min = 790 msec

The tachycardia could be initiated by atrial pacing at rates of 130 to 150 beats/min and by isoprenaline when the rate increased to 117/min. Atrial pacing at rates below 130 beats/min and above 150 beats/min did not initiate the tachycardia. Each episode of tachycardia was preceded by ventricular extrasystoles, and the initiating beat was either a ventricular extrasystole or a fusion beat with a coupling interval of 330 to 370 msec (Fig. 3).

The rate of the tachycardia varied from 162 to 207/min. A His potential was never seen preceding the ventricular depolarization during the tachycardia, unless capture beats or fusion beats were observed, confirming the ventricular origin of the tachycardia (Fig. 3). The alternating morphology of the QRS in the two standard leads was characteristic of bidirectional tachycardia. However, during some of the paroxysms the morphology of the QRS changed though the bidirectional nature of the tachycardia persisted.

Retrograde atrial activation was observed on occasions during the tachycardia, sometimes with Wenckebach periodicity, but complete AV dissociation also occurred.

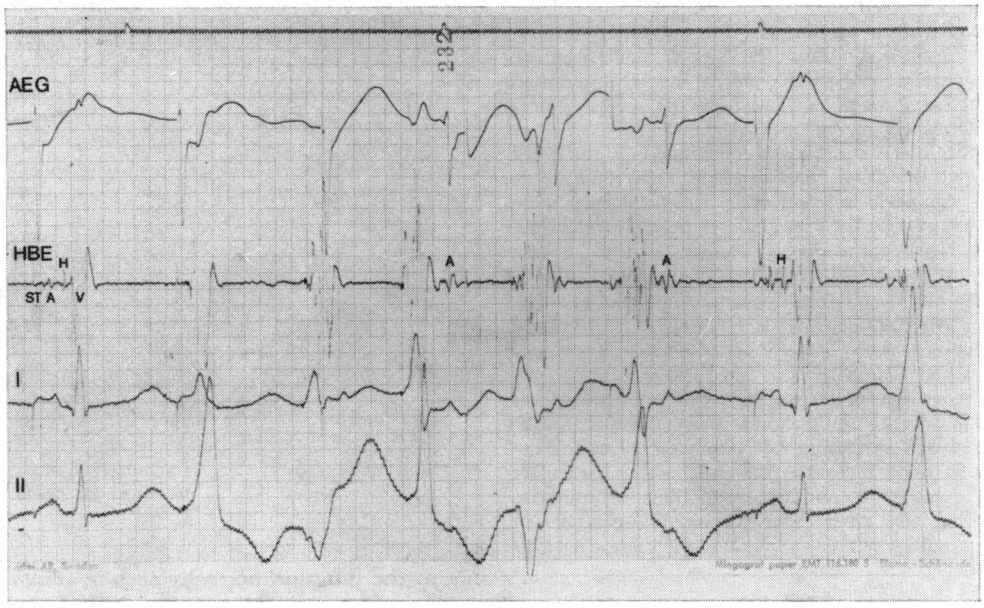

FIG. 3 *A brief episode of bidirectional tachycardia initiated by atrial pacing with recordings of His bundle electrograms (HBE), atrial electrogram (AEG), and simultaneously recorded leads I and II at 100 mm/sec. The first beat is an atrially paced beat, the second beat is a ventricular extrasystole which initiates the tachycardia. The tachycardia terminates spontaneously and is followed by an atrially paced beat. The His potential is clearly seen in the two paced beats but not during the tachycardia. The atrial electrogram shows that the first three beats are atrially paced beats, but the next three are caused by retrograde atrial activation (ST – stimulus, A – atrial depolarization, V – ventricular depolarization, H – His depolarization).*

Carotid sinus stimulation had no effect on the tachycardia. Termination occurred spontaneously, usually without alteration in the cycle length of the tachycardia, but on one occasion two spontaneous ventricular extrasystoles terminated the tachycardia.

Progress

After catheterization practolol was given in a dose of 50 mg twice daily, increasing to 100 mg twice daily. Exercise was again carried out, as described above. The heart rate increased to 120/min and apart from isolated extrasystoles sinus rhythm persisted. During the follow-up period of 3 months, she has remained well without any further syncopal episodes.

Discussion

The mechanism and origin of bidirectional tachycardia have been disputed since it was first described by Schwensen in 1922. The main reason for the dispute is that, with the standard electrocardiogram, the distinction of ventricular from supraventricular tachycardia can be difficult, even with the additional information obtained using atrial and oesophageal leads.

Rosenbaum, Elizari, and Lazzari (1969) have recently reviewed the mechanism of bidirectional tachycardia and have proposed that this arrhythmia is supraventricular in origin with permanent aberration in the right bundle-branch and alternating aberration in the two divisions of the left bundle-branch. Indirect support for its supraventricular origin is the association of this arrhythmia with nodal and junctional tachycardia (Zimdahl and Kramer, 1947; Zimdahl and Townsend, 1954; Castellanos, Azan, and Calvino, 1960; Sepaha, Jain, and Bhandari, 1962). In this case the presence of a short PR interval raised the possibility of pre-excitation and of a re-entry tachycardia involving the atrioventricular junction. However, His bundle electrography failed to confirm this and demonstrated in this case, as in the three others studied

using this technique (Morris and Zipes, 1973; Kastor and Goldreyer, 1973; Cohen, Deisseroth, and Hecht, 1973), that the tachycardia was ventricular in origin.

In a recent review of the published cases of bidirectional tachycardia it was shown that the majority occurred in association with severe myocardial disease and in 82 per cent digitalis had been administered and was thought to be a contributory factor in the production of this arrhythmia (Cohen *et al.*, 1973). In this case neither digitalis nor myocardial disease played a part in the production of the arrhythmia, in fact increase in the heart rate on effort or with emotional stress appeared to be the important precipitating factor. It is interesting that the arrhythmia could be reproduced in the catheter laboratory by atrial pacing or isoprenaline.

The appearance of bidirectional tachycardia in this case is unusual for several reasons. The patient was very young, had normal heart function demonstrated by angiography and echocardiography, and was not receiving digitalis, and the arrhythmia appeared to be precipitated by increasing the heart rate.

Although ventricular arrhythmias have been described during and immediately after effort in people with clinically normal hearts, these arrhythmias tend to be transient, lasting for 3 to 5 beats (Gooch and McConnel, 1970). Emotion and effort are characteristic precipitating factors of the ventricular arrhythmias associated with the syndrome described by Jervell and Lange-Nielson (1957), and in the variant without deafness, the Romano-Ward syndrome (Romano, Gemme, and Pongiglione, 1963; Ward, 1964). However, prolongation of the QT interval which is an integral part of these syndromes, was not present in this case. There are, however, isolated reports of children without organic heart disease and with normal QT intervals who have arrhythmias and syncope produced by effort and emotion (Wennevold, Melchior, and Sandøe, 1965; Horan and Venables, 1962). In the case described by Wennevold and his colleagues (1965), the arrhythmia (their Fig. 3) showed considerable variation in the QRS morphology and closely resembled that seen in this case. This was also seen in the case described by Horan and Venables (1962) in which there were runs resembling a bidirectional tachycardia. In both these cases, as in this case, the arrhythmia was only demonstrated by recording an electrocardiogram during or after effort. It, therefore, seems advisable to carry out routine exercise electrocardiograms in children who present with unexplained syncopal episodes, especially if these are related to effort or emotion.

This case supports the recent evidence that bidirectional tachycardia is ventricular in origin. However, the origin of bidirectional tachycardia may vary from patient to patient and further studies are required in more patients to define the mechanism of the tachycardia and to confirm that bidirectional tachycardia really is a 'ventricular' tachycardia.

References

Bekheit, S., Morton, P., Murtagh, J. G., and Fletcher, E. (1973). Comparison of sinoventricular conduction in children and adults using bundle of His electrograms. *British Heart Journal*, **35**, 507.

Castellanos, A., Jr., Azan, L., and Calvino, J. M. (1960). Simultaneous tachycardia. *American Heart Journal*, **59**, 358.

Cohen, S. I., Deisseroth, A., and Hecht, H. S. (1973). Infra-His bundle origin of bidirectional tachycardia. *Circulation*, **47**, 1260.

Feigenbaum, H., Popp, R. L., Wolfe, S. B., Troy, B. L., Pombo, J. F., Haine, C. L., and Dodge, H. T. (1972). Ultrasound measurements of the left ventricle. A correlative study with angiocardiography. *Archives of Internal Medicine*, **129**, 461.

Fortuin, N. J., Hood, W. P., and Craige, E. (1972). Evaluation of left ventricular function by echocardiography. *Circulation*, **46**, 26.

Fraser, G. R., Froggatt, P., and James, T. N. (1964). Congenital deafness associated with electrocardiographic abnormalities, fainting attacks and sudden death. *Quarterly Journal of Medicine*, **33**, 361.

Gooch, A. S., and McConnel, D. (1970). Analysis of transient arrhythmias and conduction disturbances occurring during submaximal treadmill exercise testing. *Progress in Cardiovascular Diseases*, **13**, 293.

Horan, M., and Venables, A. W. (1962). Paroxysmal tachycardia with episodic unconsciousness. *Archives of Disease in Childhood*, **37**, 82.

Jervell, A., and Lange-Nielson, F. (1957). Congenital deaf-mutism, functional heart disease with prolongation of the Q–T interval, and sudden death. *American Heart Journal*, **54**, 59.

Kastor, J. A., and Goldreyer, B. N. (1973). Ventricular origin of bidirectional tachycardia. Case report of a patient not toxic from digitalis. *Circulation*, **48**, 897.

Lown, B., Ganong, W. F., and Levine, S. A. (1952). The syndrome of short P–R interval, normal QRS complex and paroxysmal rapid heart action. *Circulation*, **5**, 693.

McCammon, R. W. (1961). A longitudinal study of electrocardiographic intervals in healthy children. *Acta Paediatrica (Uppsala)*, **50**, Suppl. 126.

Morris, S. N., and Zipes, D. P. (1973). His bundle electrocardiography during bidirectional tachycardia. *Circulation*, **48**, 32.

Roberts, N., and Olley, P. (1972). His bundle electrogram in children. Statistical correlation of the atrioventricular conduction times in children with their age and heart rate. *British Heart Journal*, **34**, 1099.

Romano, C., Gemme, G., and Pongiglione, R. (1963). Aritmie cardiache rare dell'eta pediatrica. *Clinica Pediatrica*, **45**, 656.

Rosenbaum, M. B., Elizari, M. V., and Lazzari, J. O. (1969). The mechanism of bidirectional tachycardia. *American Heart Journal*, **78**, 4.

Schwensen, C. (1922). Ventricular tachycardia as the result of administration of digitalis. *Heart*, **9**, 199.

Sepaha, G. C., Jain, S. R., and Bhandari, C. R. (1962). Bidirectional tachycardia. *British Heart Journal*, **24**, 61.

Tynan, M., Kaye, H. H., Reid, D. S., Ozme, F., and Hunter, A. S. (1974). Assessment of left ventricular performance in infants, children and adults using single plane cine angiocardiography (abstract). *British Heart Journal*, **36**, 399.

Ward, O. C., Jr. (1964). A new familial cardiac syndrome in children. *Journal of the Irish Medical Association*, **54**, 103.

Wennevold, A., Melchior, J. C., and Sandøe, E. (1965). Adams-Stokes syndrome in children without organic heart disease. Electrocardiogram after exercise as a diagnostic tool. *Acta Medica Scandinavica*, **177**, 557.

Zimdahl, W. T., and Kramer, L. I. (1947). On the mechanism of paroxysmal tachycardia with rhythmic alternation in the direction of the ventricular complexes. *American Heart Journal*, **33**, 218.

Zimdahl, W. T., and Townsend, C. E. (1954). Bidirectional ventricular tachycardia due to digitalis poisoning. *American Heart Journal*, **47**, 304.

Requests for reprints to Dr. D. S. Reid, Department of Cardiology, Newcastle General Hospital, Westgate Road, Newcastle upon Tyne NE4 6BE.