Tachycardia induced myocardial dysfunction

A reversible phenomenon?

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SUMMARY Four patients with myocardial dysfunction related to tachycardia underwent electrophysiological studies, which showed a re-entrant supraventricular tachycardia using an accessory atrioventricular connexion. Serial assessment of left ventricular function by echocardiography before and after control of the tachycardia indicated a variable degree of reversibility. Endomyocardial biopsy in two patients detected non-specific histological changes. Because of the possible role of ischaemia in this condition effective control of prolonged tachycardia is needed to prevent deterioration of myocardial function.

Paroxysmal supraventricular tachycardia is usually well tolerated in the absence of underlying impairment of cardiac function.¹⁻³ There are, however, published reports of patients with chronic or frequent episodes of supraventricular tachycardia who present with symptoms and with clinical and radiological signs of congestive cardiac failure.⁴⁻⁹ The patients in these reported cases were in general young, often in the paediatric age group. Many were without underlying cardiac disease, and with control of the tachyarrhythmia the symptoms and signs of congestive heart failure resolved. Detailed assessment of the mechanism of the arrhythmia and accurate serial determination of left ventricular function before and after the resolution of congestive heart failure, as a result of successful antiarrhythmic treatment, has only recently been reported.^{10 11} We report four patients with chronic or frequent episodes of supraventricular tachycardia, resulting in severe impairment of left ventricular function. The mechanism of the arrhythmia was determined in each case by electrophysiological study, and endomyocardial biopsy was performed in two cases. Serial assessments of left ventricular function were made in each case by echocardiography, radionuclide scintigraphy, or both after control of the arrhythmia as well as clinical assessment of symp-

tomatic and functional status.

Patients and methods

CASE 1

A 16 year old girl had an episode of syncope which caused no personal injury. Supraventricular tachycardia (rate 160 beats/minute) was noted at the local hospital, and a chest x ray film showed cardiomegaly. She had been asymptomatic up to the time of the accident, and there was no history suggesting a recent viral illness or excessive alcohol consumption. Four weeks later she was referred to the Mayo Clinic with persistent tachycardia. She had remained asymptomatic. Chest x ray films showed cardiomegaly without pulmonary congestion, and an electrocardiogram showed a narrow complex supraventricular tachycardia (rate 150 beats/minute) with a long ventriculoatrial interval (Fig. 1). The P waves were negative in leads II, III, and aVF, and the ratio of the interval between the R wave and the retrograde P wave to the cycle length was >0.5, suggesting the permanent type of reciprocating junctional tachycardia.¹² The left ventricular ejection fraction measured during tachycardia by echocardiography (Fig. 2) and radionuclide ventriculography was 29% and 33% respectively. The left ventricle was globally hypokinetic, and an echorefractile apical thrombus was noted. There were no other major structural abnormalities of the heart detected by clinical examination or echocardiography. At cardiac catheterisation there were no shunts and the mean pulmonary capillary wedge pressure was 13 mm Hg. A left ventricular biopsy showed mild hyper-

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Fig. 1 Twelve lead electrocardiogram during tachycardia (case 1) showing negative inferior P waves and an RP interval greater than 50% of the RR interval.

trophy and mild focal interstitial fibrosis.

An electrophysiological study detected a re-entrant supraventricular tachycardia, using a concealed accessory pathway, with earliest atrial activation at the os of the coronary sinus, as has been previously described in this arrhythmia.¹² The supraventricular tachycardia could be easily terminated by ventricular extrasystoles but would spontaneously return after one or two sinus complexes without prolongation of the AH interval. Procainamide terminated the supraventricu-

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lar tachycardia by abolishing retrograde conduction via the accessory pathway. Subsequent oral treatment with digoxin and quinidine allowed sinus rhythm to return, and a repeat echocardiogram nine days later showed that left ventricular function was unchanged. She was discharged taking digoxin and quinidine and returned for follow up two months later. She had remained in sinus rhythm, and both an echocardiogram (Fig. 3) and radionuclide angiogram showed normal left ventricular function (ejection fraction 60% at rest, 70% at peak exercise). A repeat study one year later in sinus rhythm showed ejection fractions of 53% at rest and of 76% at peak exercise, but the cardiomegaly persisted. The most recent echocardiogram recorded about two and a half years after admission showed normal left ventricular dimensions and function with an ejection fraction of 63%.

CASE 2

A 9 day old boy, born after a normal pregnancy and with a normal delivery, presented with a two day history of poor feeding and irritability. On examination he was in cardiac failure and had a regular narrow complex tachycardia (280 beats/minute). During the following week he had recurrent episodes of supraventricular tachycardia with haemodynamic deterioration. During sinus rhythm, anterograde pre-excitation was noted. An echocardiogram performed in sinus rhythm and with a normal serum bicarbonate concentration showed an ejection fraction of 30%. No other structural cardiac abnormalities were noted clinically or during echocardiography. An electrophysiological study confirmed supraventricular tachycardia using a macro-reentrant circuit with a left lateral accessory pathway as the retrograde limb. The

Fig. 2 Case 1: cross sectional echocardiograms at diastole before tachycardia control in the (a) long and (b) short axis views. End diastolic dimension 60 mm. LV, left ventricle; LA, left atrium; RV, right ventricle; PW, posterior wall; VS, interventricular septum; A, anterior; P, posterior; I, inferior; S, superior; R, right; L, left.

Fig. 3 Case 1: Cross sectional echocardiograms at diastole three months after tachycardia control in the (a) long axis and (b) short axis views. End diastolic dimension 50 mm. Abbreviations as for Fig. 1.

arrhythmia was eventually controlled, and six days later a repeat echocardiogram showed a left ventricular ejection fraction of 50%. The child was discharged with treatment and has remained well for four and a half months.

CASE 3

A 3 week old boy, born after a normal pregnancy and with a normal delivery, presented with a two day history of poor feeding. On examination he had a regular tachycardia (280 beats/minute) and was in circulatory shock. He was acidotic, and a chest x ray film showed cardiomegaly and alveolar oedema. A narrow complex supraventricular tachycardia was noted on electrocardiography. After cardioversion, sinus rhythm returned (no anterograde pre-excitation) followed by clinical improvement. There was no detectable structural cardiac abnormality on clinical or echocardiographic examination, apart from a global reduction in left ventricular function with an ejection fraction of 10% with a normal arterial pH. During an electrophysiological study a concealed left free wall accessory pathway was shown to participate in the supraventricular tachycardia. Sinus rhythm was maintained with digoxin and quinidine, and a repeat echocardiogram three days later showed normal left ventricular function (ejection fraction 63%). He was well with medication four months after discharge.

CASE 4

A 10 year old boy had had Ebstein's anomaly diagnosed by cardiac catheterisation at the age of 3 years. Since birth he had had infrequent episodes of supraventricular tachycardia, and an electrocardiogram showed anterograde pre-excitation. Evaluation in

1981 by echocardiography and cardiac catheterisation confirmed the diagnosis, and left ventricular function was only mildly abnormal (ejection fraction 48%). He was mildly cyanotic at this time. An electrophysiological study showed two mechanisms for his supraventricular tachycardia: (a) macrore-entry using a right free wall accessory pathway retrogradely and (b) atrioventricular nodal re-entry using the fast pathway retrogradely. He remained well for two years, being able to play competitive sports. Regular assessments showed good functional status and a normal heart size radiologically. Over a five month period before his most recent admission the episodes of supraventricular tachycardia became more frequent and prolonged, and over the last six weeks his arrhythmia was virtually incessant. Drug treatment (digoxin, propranolol, verapamil, quinidine, encainide) proved ineffective, and he developed worsening cardiac failure and cardiomegaly. Evaluation showed moderate to severe cyanosis and gross cardiac failure. Severe impairment of left ventricular function was noted on echocardiography during sinus rhythm (ejection fraction 20%). Recurrent supraventricular tachycardia (rate 145 beats/minute) with a long VA interval was noted on monitoring. Arterial oxygen saturation was 68%, and left ventricular biopsy showed mild to moderate cellular hypertrophy with focal degenerative changes and mild to moderate interstitial oedema. There was no inflammatory infiltrate. Oral treatment with amiodarone was started, and in four days sinus rhythm was re-established and maintained with pronounced clinical improvement. A repeat echocardiogram three months later showed only a mildly improved ejection fraction of 30% which was unchanged at six months, but at the time of the most

recent follow up six months later the ejection fraction had improved further to 40–45%.

Discussion

The effects of chronic or frequent episodes of tachycardia on cardiac function are not well understood. Tachycardia results in an increase in myocardial oxygen demand while causing relative ischaemia due to reduced time spent in diastole. This effect is most prominent in the subendocardium, and, at least in the setting of congestive cardiomyopathy, may possibly result in the observed histological changes of subendocardial fibrosis and cellular vacuolisation as well as depletion of myocardial energy stores.¹³ Coleman *et al* induced chronic ventricular tachycardia in dogs with pacing and noted significant reductions in left ventricular function after 2–4 weeks of continuous pacing.¹⁴ This effect persisted after the cessation of pacing. Total myocardial energy stores were also reduced, and this was considered to be related to the impaired myocardial function. Myocardial blood flow was not determined during tachycardia, and the authors remained uncertain as to the cause of the loss of energy stores and impaired myocardial function.

Measurement of coronary blood flow in dogs during atrial tachycardia at rates above 180 beats/minute has shown a 34% reduction in flow.¹⁵ The investigators concluded that this was related to a reduction in aortic pressure and a shortening of diastole. An illustration accompanying the report shows the haemodynamic changes at the onset of tachycardia, accompanied by an electrocardiogram with progressive ST segment depression, suggesting ischaemia.

Direct extrapolation of the data from these animal studies to humans can only be made with caution and reservation. None the less the studies appear to have identified a plausible but unconfirmed explanation for our findings—namely, relative ischaemia despite normal coronary artery anatomy.

If a reduction in coronary blood flow with resultant myocardial ischaemia contributes appreciably to the development of cardiac failure in patients with tachycardia, then such factors as chronicity of tachycardia, rate of tachycardia, anatomical lesions causing reduced myocardial blood flow (for example, coronary artery disease) or increased myocardial oxygen demand (for example, aortic stenosis) and lesions causing arterial desaturation (for example, right to left shunt) might be expected to augment the myocardial depressive effect of tachycardia by accentuating the ischaemic process.

Our first case illustrates the effects of chronic tachycardia. The arrhythmia had been noted for four weeks and, in all likelihood, had been present for

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longer. Pronounced impairment of left ventricular function both during and soon after resolution of the tachycardia, with subsequent return to normal after two months, suggests a reversible derangement of myocardial cellular function related to the tachycardia. It would seem unlikely that digoxin would account for such a dramatic improvement.¹⁶ Although resting and exercise cardiac function were normal, there was evidence of permanent myocardial functional impairment, with a mild increase in diastolic dimension, and evidence of myocardial fibrosis, without inflammation, on biopsy. None the less at the time of the most recent echocardiogram left ventricular dimensions and overall function had reverted to normal.

In the fourth case frequent prolonged episodes of tachycardia had been occurring for five months, associated with pronounced systemic arterial oxygen desaturation and worsening cardiac failure. Previous evaluations showing near normal left ventricular function and the coincident clinical deterioration imply a detrimental effect of the tachycardia on myocardial function. After resolution of the tachycardia there was only mild improvement in left ventricular function over a nine month period, and an endomyocardial biopsy showed focal severe myocardial damage. Systemic arterial oxygen desaturation in association with tachycardia may have resulted in ischaemia, which led to the observed histological changes, and permanent impairment of myocardial function. Despite the histological appearance there was a further improvement in overall left ventricular function with an increase in ejection fraction to approximately 40–45%, which could reflect a compensatory response from other undamaged areas of the myocardium.

Cases 2 and 3 illustrate the consequences of extremely rapid rates, as are commonly found in infants with tachycardia associated with an accessory atrioventricular connexion. Left ventricular function during sinus rhythm, and in the absence of acidosis, was considerably impaired in both but showed a rapid improvement within one week of controlling the arrhythmia. Whether the immature infant left ventricle is particularly susceptible to the demands of a rapid tachycardia or whether these infants have an underlying left ventricular disorder is speculative, but are intriguing issues which cannot be answered from our observations.

There are several implications of this study. Firstly, chronic or frequent episodes of supraventricular tachycardia can have major deleterious effects on left ventricular function, which may be asymptomatic and transient. Secondly, the effects on left ventricular function are not necessarily completely reversible after control of the arrhythmia and may result in chronic ventricular dysfunction. Thirdly, patients

with systemic arterial oxygen desaturation and tachyarrhythmias should be treated promptly to avoid permanent severe myocardial damage. Finally, current knowledge is insufficient to explain the mechanisms of tachycardia induced impairment of left ventricular function and the relation between the biopsy findings and the arrhythmias.

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