Chronic recurrent ventricular tachycardia

Chronic recurrent ventricular tachycardia is always a serious arrhythmia since it may deteriorate into fatal ventricular fibrillation. Chronic recurrent ventricular tachycardia at a rate of 250 beats/minute may cause syncope, but at a rate of 100-150 may not cause any symptoms. Often, but not always, a signal to serious underlying heart disease, the arrhythmia may recur or may disappear as mysteriously as it came. Treatment usually demands long-term drugs, and control may prove difficult.

Chronic recurrent ventricular tachycardia may complicate ischaemic heart disease and almost any heart-muscle disease. It is more likely when the QT interval is prolonged, when both recurrence and conversion to ventricular fibrillation may be encouraged by quinidine-like antiarrhythmic drugs that further prolong repolarisation. Chronic recurrent ventricular tachycardia seems to have become more common—possibly as a result of increased survival of patients after myocardial infarction.

Chronic recurrent ventricular tachycardia may be the first sign of heart disease. The symptoms depend on the ventricular rate and the underlying cardiac disorder, patients complaining of blackouts, angina, or palpitations, but they are often unaware of any change in heart rate. Repeated Holter ECG tape recording may help to detect the arrhythmia in patients with fainting attacks or other poorly defined symptoms; monitoring is also valuable in assessing the response to treatment. When chronic recurrent ventricular tachycardia is associated with ischaemia it tends to be provoked by tachycardia, so that exercise or emotion may then precipitate both the arrhythmia and angina. In other cases the arrhythmia may occur at any time. Sudden, unexpected death is a feature of hypertrophic cardiomyopathy, but may be prevented if an occult ventricular arrhythmia is detected and treated. Chronic recurrent ventricular tachycardia is also common in dilated (congestive) cardiomyopathy, when it may bring on heart failure. If tachycardia is prolonged cardiac failure may develop even in subjects without organic heart disease.

The appearance of ventricular tachycardia on the electrocardiogram varies. Typically the arrhythmia starts with a premature ventricular beat occurring on, or close to, the T wave of the preceding sinus beat. The premature beat initiates a self-sustaining tachycardia with uniformly wide and bizarre QRS complexes, whose initial inscription is slow, with a mean axis different from that of the normal complexes.

In other cases the initiating ventricular beat occurs after full inscription of the T wave of the preceding sinus beat, and the complexes vary in form from beat to beat. One type is known as Torsades de Pointe because of progressive and cyclical twisting in the electrical axis of each complex. This form of ventricular tachycardia is often self-terminating, but also particularly likely to deteriorate into ventricular fibrillation. It may occur as a complication of drugs that prolong repolarisation, or may signify a metabolic abnormality, particularly potassium and magnesium deficiency.[1] A prolonged QT interval is familial in the cardioauditory syndrome of Jervell and Lange-Nielson in which ventricular tachycardia or ventricular fibrillation may occur.[2] This condition may be perplexing because the QT prolongation is not always present on the ECG, though it may often be recorded after exercise.

Emergency treatment of ventricular tachycardia is usually by intravenous lignocaine, but DC counter shock is often required. Lignocaine should be avoided when the arrhythmia complicates a prolonged QT interval. In such cases the heart rate should be accelerated by isoprenaline or temporary pacing. The aim, often impossible, is to identify and correct any underlying cardiac fault. Serum potassium concentrations should be measured and then kept as close as possible to 5 mmol(mEq)/l. Treatment must be continued with antiarrhythmic drugs, whose initial selection is usually empirical. These drugs are often only partially effective, and, reflecting this, the prognosis of chronic recurrent ventricular tachycardia is poor. Careful trials of antiarrhythmic drugs are time consuming, and the patient must be in hospital for his safety and to measure the response. Because of the deficiencies of drug treatment, purpose-built implanted pacemakers,[3] and surgical excision or exclusion of arrhythmogenic foci have been tried.[4] Classically chronic recurrent ventricular tachycardia complicating a ventricular aneurysm has been an indication for excising this, but any ventricular scar, either visible or theoretical, may be equally culpable. These scars can be located by ECG mapping at operation. Whether excision, encirclement, or cryotherapy is used, however, a further scar results and so recurrences of chronic recurrent ventricular tachycardia are possible.

These difficulties have led to an aggressive and invasive approach, where ventricular tachycardia is induced by electrode stimulation of the heart during catheterisation and the effect of drugs can then be studied.[5] These studies may take

several days.[6-9] Induction of supraventricular arrhythmia has been employed for some years to assess drug effects, but electrophysiological studies of ventricular tachycardia have hitherto been less informative. Moreover, the deliberate induction of a potentially fatal cardiac arrhythmia is a frightening procedure. To induce these arrhythmias multiple multipolar electrode catheters are positioned in the heart (usually through the femoral veins) for the delivery, by programmed electrical stimulation, of single or multiple extra stimuli to chosen points at predetermined phases of the cardiac cycle. It is usually possible to induce ventricular tachycardia similar in morphology and rate to the spontaneously occurring arrhythmia, and then to terminate it by similar means. Techniques for inducing ventricular tachycardia include rapid atrial or ventricular pacing, bursts of extra stimuli, and combinations of these. The arrhythmia is usually terminated by a single or short burst of extra stimuli, or by ventricular or atrial overdrive at rapid rates. Thus the mechanism and origin of ventricular tachycardia can be characterised, and serial electrophysiological studies performed after giving drugs. Patients selected for the prolonged studies have usually been refractory to medical treatment.

Though induction of ventricular arrhythmias is still contentious, and possible only in specialist units, the speedier formulation of an optimum drug regimen for the individual by these means would represent an important advance.

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Changes in treating soft-tissue sarcomas

Progress in treating soft-tissue sarcomas is difficult to assess, since these tumours are rare (1% of cancers), with multiple subtypes.[1 2] Though specialist institutions have been able to organise controlled trials of treatment for other rare tumours, the many different subtypes of sarcoma and the importance of histological grade[3] have made formal randomised trials virtually impossible.

Simple excision of the primary tumour is followed by local recurrence in 40-80% of cases. Radical surgical excision (including amputation for many limb tumours) has been widely accepted as the standard treatment,[4] and has cut local recurrences by half. Nevertheless, distant metastases occur in up to half of cases[5]—often the same patients who relapse locally.[4] Since even radical surgery has failed to control the disease, more recently conservative surgery has been combined with radiotherapy or chemotherapy or both.

Several studies attempted to avoid amputation by combining aggressive local surgery with radiotherapy.[3 6] Encouraging preliminary results have been reported, though this combination of treatments seemed not to influence distant metastases. Furthermore, it seemed less effective in preventing local recurrences in proximal than in distal limb lesions.[5] The next step was to combine local surgery and high-dose radiotherapy with adjuvant chemotherapy, and preliminary results are now available. Rosenberg et al[7] have reported on a study in which patients with soft-tissue sarcomas of the arms or legs were randomised to receive either amputation or a limb-sparing operation and postoperative radiotherapy. All patients received adjuvant chemotherapy (adriamycin, cyclophosphamide, and methotrexate). So far local recurrence has been reported in only one of the 16 patients treated by limb-sparing surgery and radiotherapy, and there have been no local recurrences in the group treated with amputation (10 patients). Comparison of the results in all 49 patients in the study (with limb, trunk, and head and neck lesions) with historical controls treated with radical surgery alone shows improvements in both disease-free survival and overall survival. So far too few patients have relapsed to distinguish between the two randomised subgroups.

Morton et al[5] used a regimen which included radiotherapy and intra-arterial adriamycin (without proximal vascular compression) with local resection. Postoperatively some patients also received chemotherapy, with or without immunotherapy. Of the 17 patients treated with surgery alone, 11 have had local recurrence and required amputation. None of the five who had postoperative radiotherapy have had local recurrences. Of the eight patients who received preoperative intra-arterial adriamycin, five had shrinkage of tumour, but the limb was salvaged in only one patient. Thirteen of 14 patients treated with intra-arterial adriamycin and radiotherapy had sufficient shrinkage of the tumour to allow local resection. All patients treated with this combined approach have remained free of local or distant recurrence for four to 34 months. When these preliminary results are compared with those achieved in the unrandomised group of 17 patients managed by surgery alone, the combined modality approach seems superior.

Karakousis et al[8] have recently reported the results of concomitant radiotherapy and chemotherapy. Intra-arterial adriamycin with a tourniquet applied proximally was used in two patients and produced definite necrosis of the tumour, though at the expense of increased local toxicity.[8] Giving adriamycin by this technique may result in tissue concentrations up to 40 times greater than that achieved by bolus injection.[9 10] In seven other patients chemotherapy[11] was started within two to three days of surgery and followed within a week by radiotherapy. No relapses have been seen in these patients, but the follow-up is very short and the numbers small.

Preliminary results of combined modality treatment suggest that it may improve survival, reduce the frequency of relapse, and preserve limb function. Sadly, however, none of the published studies have been randomised: controlled trials are particularly important in tumours with so many histological subtypes, grades, and sites of disease. At the National Cancer Institute in the United States Rosenberg[7] has begun a prospective randomised study of adjuvant chemotherapy, and will continue to report on the protocol, in which amputation is also being compared with local surgery together with radiotherapy. The soft-tissue sarcoma co-operative group of the European Organisation for Research on the Treatment of Cancer is also running a randomised study of adjuvant chemotherapy after primary surgery.

The results of chemotherapy for metastatic soft-tissue sarcomas have been less encouraging. Even with regimens which produce remissions in up to half of patients the clinical responses have been relatively short and many are incomplete.[4] Immunotherapy has been used for advanced disease and as an adjuvant, but there are no reports of convincing benefit, though trials are continuing.[4 6 7]