meanwhile there are as many men as opinions about what constitutes rational antibiotic treatment. Thus guidelines will be mainly derived from personal predilections and preferences.

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Supraventricular tachycardias

The term “supraventricular tachycardia” includes all those arrhythmias arising from the atrial myocardium and atrioventricular junctional tissue. Arrhythmias such as atrial fibrillation, atrial flutter, and atrial tachycardia are due to electrical instability of the atrial myocardium, are most often associated with organic heart disease, and are more commonly seen in older patients. In contrast, tachycardias occurring as a result of abnormalities of atrioventricular conduction, either functional (for example, multiple atrioventricular nodal pathways) or anatomical (for example, accessory bundles), are usually seen in patients without organic heart disease and occur at all ages.

Over the past decade intracardiac studies have shown many mechanisms for these tachycardias, most of which are caused by continuous circulation of depolarising activity and are known as “re-entrant.” A characteristic of re-entrant tachycardias is that they may be started or stopped by electrical stimulation; this correlates with the clinical observation of sudden onset and cessation in most patients. In a few tachycardia may be incessant. To some extent the mechanism of the tachycardia may determine the frequency and duration of attacks.

We still know little about the clinical course of the different types of arrhythmia, but what evidence there is suggests that in individual patients the frequency and severity of untreated episodes of tachycardia remain unchanged over many years. Many patients with infrequent or brief episodes do not need treatment. A substantial proportion, however, are troubled by recurrent attacks and require long term treatment. This most often takes the form of oral antiarrhythmic drugs to suppress events which may trigger tachycardia (for example, extrasystoles), to control the ventricular response to continuing tachycardia (as in atrial fibrillation), or to modify the circuit so that it can no longer sustain tachycardia despite the continued occurrence of triggering events. Though many patients may be treated successfully with conventional drugs, alternatives must be considered for the minority who are not controlled. Of the more recently available antiarrhythmic drugs, amiodarone is perhaps the most widely used. This drug is of great value in patients with tachycardias which fail to respond to other drugs, but a high incidence of side effects limits its value for long term treatment. If treatment with amiodarone is unsuccessful newer investigational drugs should be considered. One such group, with cellular properties similar to those of quinidine and disopyramide but with distinctly different clinical effects, is of particular interest. Flecaïnide is the most widely known of these drugs in Britain and offers considerable promise in the termination and prevention of supraventricular tachycardias of all types.

In selected patients with recurrent paroxysmal tachycardias the choice of an effective preventive drug may be helped by intracardiac stimulation studies—an approach already accepted as an essential part of the management of ventricular tachycardia. It allows the antiarrhythmic effect of several drugs to be tested individually over a short period of time, thus avoiding the laborious and often ineffective process of empirical drug trials. Suppression of a tachycardia induced electrically in the laboratory predicts a favourable long term response to oral treatment.

If antiarrhythmic drugs fail, are not appropriate (for example, in pregnancy), or are not tolerated several approaches may be considered. Surgical ablation of the bundle of His or accessory bundle will prevent rapid ventricular rates in response to atrial tachycardias or interrupt a re-entrant pathway. These procedures have been discussed in a previous leading article. More recently, several groups have successfully excised the focus of paroxysmal atrial tachycardia.

Surgery is usually considered as a last resort. Less drastic is the use of pacemakers, which can suppress tachycardia or terminate recurrent paroxysms. The former application is rarely successful in the long term, but tachycardia termination pacemakers have proved themselves valuable for patients with recurrent tachycardias. External versions of such pacemakers have been used for several years, and recently a fully implantable and automatic version has become available. These pacemakers “recognise” tachycardia as a fast rate or, in the more refined devices, as a sudden change to a rapid rate. They respond by rapid overdrive or premature stimulation designed to interrupt tachycardia. At present the termination algorithms are relatively crude and often allow the tachycardia to continue for some time before it is stopped. Physiological and technical problems are, however, likely to be overcome very shortly. Improvements in design and growing evidence of both the safety and efficacy of these devices seem likely to lead to their more liberal use as their cost and complexities are balanced against the side effects inevitable with drug treatments.

Treatment with pacemakers is not, however, appropriate in patients with frequently repetitive or continuous tachycardias—and some tachycardias (such as atrial fibrillation and some
types of atrial flutter) are not amenable to such treatment. In these circumstances an alternative to surgical ablation of atrioventricular conduction tissue is transvenous ablation of the His bundle or the atrioventricular node. This new technique, first applied to man by Scheinman et al after experiments on dogs, is designed to induce atrioventricular block, thus removing the ventricles from the influence of supraventricular rhythms. An electrode catheter is positioned in the region of the bundle of His and a shock of 200 to 375 watt seconds from a conventional cardioversion apparatus is delivered to the pole from which the largest His potential is recorded. In most patients the result is either complete atrioventricular block or a lesser degree of block. Most patients also develop a junctional escape rhythm, but in any case all need to be given a permanent pacemaker because the clinical course of the conduction defect and the escape rhythm is not known. This technique has been used successfully in many refractory supraventricular tachycardias. Its obvious advantage is that open heart surgery is avoided—and, indeed, the number of patients referred for surgery seems likely to decrease as transvenous ablation techniques become more widely accepted. Above all, no serious side effects or complications have been noted. Properly employed, this method is an important advance in the management of refractory supraventricular tachycardia, and with refinement it may even be possible to "cure" certain types of tachycardia. The technique does, however, require experience of intracardiac studies and should not be undertaken lightly.

These advances made recently in our understanding of supraventricular tachycardias and their treatment have been based largely on detailed information from intracardiac studies. With currently available drugs and therapeutic techniques there is no supraventricular tachycardia which could properly be described as "refractory."

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Microvascular free tissue transfer

The concept of free tissue transplantation facilitated by vascular anastomosis was introduced at the turn of the century but did not develop until recently, when technical innovation and an improved understanding of the cutaneous circulation combined to improve results. The successful anastomosis of small blood vessels was achieved in 1960 with an operating microscope. In the same decade surgeons began to explore the idea of vascular territories serviced by a single arteriovenous system and this led to the description of axial pattern skin flaps. The transfer of such a flap to a distant site with microvascular anastomosis of its supplying vessels began to be known as a "free flap."

The application of this technique and its extension to tissues other than skin have produced a revolution in reconstructive methods. The surgeon is no longer constrained by the teaching that a flap must not be longer than it is broad; vascularised tissue with a permanent blood supply may be introduced to a devitalised area; and a complex defect may be repaired in a single operation—no longer is there any need for the multiple staged procedures and the prolonged stays in hospital associated with earlier methods of tissue transfer.

Two surgical specialties that have benefited particularly from microvascular free tissue transfers are treatment of trauma and reconstruction after extirpation of malignant disease. So many cutaneous, myocutaneous, and fasciocutaneous flaps with differing characteristics of size and thickness have been devised that virtually any soft tissue defect may now be covered. In most cases the donor site may be closed directly, though a skin graft may be necessary. Alternatively, a muscle may be transferred alone and skin grafted in its new site, so leaving virtually no donor defect.

Composite flaps including skin, muscle, or bone may provide solutions to complex problems of reconstruction. In the head and neck, for example, defects of the mandible, the lining of the mouth, and covering skin may be repaired with a single flap from the foot, the iliac crest, the chest wall, the forearm, or the scalpula. An acceptable cosmetic result is achieved with an inconspicuous defect at the donor site. The introduction of