CHAPTER 5

Successes and Limitations of Antiarrhythmic Drug Therapy

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A. Introduction

This book testifies to the growing fund of knowledge, both at the basic scientific and at the clinical level, concerning the electrophysiological effects of antiarrhythmic drugs. A large number of antiarrhythmic agents are available for clinical use. All are of proven efficacy. Yet the question remains: is a particular agent going to benefit a particular patient?

In this chapter indications for antiarrhythmic therapy are discussed together with the possible disadvantages and limitations of treatment.

B. Haemodynamic Effect of Arrhythmias

Disturbances of rhythm and conduction affect cardiac function in a number of different ways.

I. High Rate

Healthy hearts can tolerate heart rates of 150–200 beats/min for long periods without adverse consequences. However, tachycardia imposes added burdens on the heart. It is associated with increased contractility and a substantial increase in myocardial oxygen requirement. Because diastole is shortened, there is less time available for ventricular filling and for coronary blood flow. In the compromised heart, this can lead rapidly to cardiac failure and to myocardial ischaemia. Soon after the onset of tachycardia, the immediate fall in flow and pressure is reversed by reflex responses. This reflex adaptation is dependent on an intact sympathetic nervous system (Nakano and McGloy 1970) and is therefore attenuated or absent in patients on beta-blockers.

II. Loss of Atrial Transport Function

In the normal heart, some 20% of ventricular filling is achieved by atrial contraction, the exact figure depending on the PR interval (Leinbach et al. 1969). If atrial contraction is out of phase (as in atrioventricular dissociation) or ineffective (as in atrial fibrillation) there is a fall in cardiac output, especially on exercise.
III. Abnormal Sequence of Ventricular Activation

It has been found that stroke volume may be decreased by 10% as a consequence of an abnormal pattern of ventricular contraction, as may be seen in ventricular pacing (Soyeur 1986).

C. Symptoms and Complications of Arrhythmias

It is not surprising that haemodynamically important arrhythmias lead to dizziness, dyspnoea, chest pain and syncope, but many patients with arrhythmias never experience symptoms or do so only occasionally. Why this should be is by no means clear. It is remarkable that so few patients complain of symptoms which can be attributed to arrhythmias during the course of acute myocardial infarction, when such arrhythmias are almost invariable. Likewise, Holter monitoring has revealed that patients may have paroxysmal tachycardia at rates of up to 200/min without being aware of it. Some patients, on the other hand, may be uncomfortably aware of their heart when there is only a slight sinus tachycardia.

I. Palpitation

An uncomfortable awareness of the heart’s action is a common complaint and may take many different forms. With ectopic beats, the patient may be conscious of an occasional thump in the chest which is associated with the strong beat which follows a postectopic pause. The latter phenomenon may give rise to a feeling that “the heart has stopped”. Paroxysmal tachycardias give rise to a sensation of the heart racing, usually with a very abrupt onset, particularly if the episode is supraventricular in origin. Patients with atrial fibrillation may be aware of the fast and irregular action of the heart, especially when they first experience the arrhythmia, but they often cease to note this when the disorder becomes established.

II. Dyspnoea

Dyspnoea is likely to occur when a haemodynamically significant arrhythmia complicates heart disease, especially when the patient is close to or in heart failure already. Even in the absence of heart disease, prolonged episodes of paroxysmal tachycardia may give rise to breathlessness.

III. Dizziness and Syncope

If the arrhythmia causes a fall in cardiac output and blood pressure, dizziness and syncope may occur. This may happen virtually at the onset of the arrhythmia, particularly if the heart rate is very fast, but adaptations may take place which correct the blood pressure and restore cerebral blood flow. Syncope is especially prone to occur with ventricular tachycardia at high rates and in self-terminating episodes of torsade de pointes and ventricular fibrillation.