1. Introduction

Atrial arrhythmias, such as atrial fibrillation (AFIB), atrial flutter (AFL) and atrial tachycardia (AT) are relatively common following cardiac surgery [1-2]. Patients undergoing cardiac surgery may be particularly prone to develop atrial arrhythmias for a variety of reasons, including the type of surgery performed, the presence of underlying heart disease and other associated medical conditions such as hypertension, hemodynamic instability or congestive heart failure, pulmonary insufficiency, associated pericardial inflammation, and the increased sympathetic and vagal nervous system activity that accompanies such surgery [1-2]. Post-operative atrial arrhythmias may cause significant symptoms including palpitations, shortness of breath, chest pain and even syncope due to the hemodynamic instability that often exists in this setting. If associated with a rapid ventricular response, post-operative atrial arrhythmias may cause ischemia or congestive heart failure, and in the case of atrial fibrillation thromboembolic stroke may even occur. The treatment, and perhaps more importantly the prevention of atrial arrhythmias following cardiac surgery is therefore critical. Fortunately, through extensive experimental and clinical studies significant progress has been made towards delineating the mechanisms and possible treatments for most atrial arrhythmias, including those that occur in the post-operative period following cardiac surgery.

For example, clinical studies in man have demonstrated that reentry is the electrophysiologic mechanism underlying many atrial arrhythmias, including AFIB, AFL and most forms of AT. In AFIB multiple reentrant circuits propagate throughout
both the left and right atria [3-4], whereas in AFL a single reentrant circuit is confined
to the right atrium [5-6], and in AT reentry circuits may develop around surgical
incisions or prosthetic patch materials [7-8]. The delineation of these
electrophysiologic mechanisms has been made possible in part by the use of
percutaneous, catheter-based, multi-electrode mapping techniques [9-14], and in part
by the use of intra-operative multi-electrode mapping techniques [15-16]. However,
due to the invasive nature of the techniques required to study arrhythmias in man,
animal models have also been created to better understand arrhythmia mechanism(s)
and to develop safer and more effective treatments. These animal models, including
several anatomical and functional reentry models of AFL and AT [17-25] and the
pacing-induced models of AFIB [26-29], have electrophysiologic characteristics that
are similar to human atrial arrhythmias, including those that occur following open-heart
surgery for acquired or congenital heart disease.

This chapter will briefly review the relevant electrophysiologic characteristics
of AFIB, AFL and AT, atrial arrhythmias that commonly occur in humans following
cardiac surgery, and then describe in detail the experimental arrhythmia models that
have been developed to study these clinically occurring arrhythmias.

2. Electrophysiologic Characteristics of Cardiac Arrhythmias That Commonly
Occur Following Cardiac Surgery in Humans

Type 1AFL is a rapid, regular atrial tachycardia characterized by an inverted, saw­
tooth flutter (F) wave pattern on surface electrocardiogram (ECG), at a rate ranging
from 240-350 beats per minute. Type 1 AFL is due to a large reentry circuit in the
right atrium that may rotate either in a counterclockwise (common form) or
clockwise (uncommon form) direction [11-12]. The reentry circuit in type 1 AFL
had been shown to have a fully excitable gap, during which both overt and
concealed entrainment can be demonstrated [30-31]. Type 1 AFL can also be
terminated by rapid overdrive atrial pacing [6]. Furthermore, there is a critical zone
in the reentrant circuit where atrial flutter can be interrupted and cured by
radiofrequency catheter ablation [32-33]. This critical zone is comprised of an
isthmus of tissue between the inferior vena cava and tricuspid valve annulus (i.e. the
TV-IVC isthmus), which has been shown to be more slowly conducting than other
atrial tissue in the reentry circuit [34-35]. The TV-IVC isthmus has also been shown
to be prone to development of unidirectional block, leading to initiation of reentry
during induction of AFL by rapid atrial pacing [36-37]. Pharmacological termination
of AFL in humans has been shown to be due to conduction block in the TV-IVC
isthmus, which may occur either abruptly without cycle length oscillation, or
following premature eccentric activation due to failure of lateral boundaries or
reflected reentry in the reentry circuit [38]. During AFL double-potential
electrograms have been recorded along the Eustachian ridge and the crista
terminalis, suggesting that these anatomical structures form lines of block defining