

INTRODUCTION

The term "hypothermia" has been used to describe the general reduction of body temperature. Hypothermia may result following prolonged accidental environmental exposure or may occasionally, by intention be induced as adjunctive therapy during some surgical procedures. Within the past 40 years, numerous reports have been published showing the usefulness of hypothermia in a variety of clinical circumstances such as intracardiac procedures, surgery associated with great blood loss, hypermetabolism and severe stress.

The most serious complication seen with induced hypothermia is the development of ventricular fibrillation, the usual form of cardiac failure preceding death in most mammals. This unpredictable complication is the major risk that severely limits the use of hypothermia under certain clinical conditions. Therefore, an analysis of the factors involved in the development of ventricular fibrillation during hypothermia is a matter of obvious interest but the electrophysiologic mechanisms underlying development of ventricular fibrillation in hypothermia are poorly understood.

Prevention of ventricular fibrillation is one component of an overall strategy for minimizing myocardial ischemia. If fibrillation ensues, the electrical cardioversion required to treat this condition may itself contribute to myocardial injury, particularly in an ischemic heart. Unfortunately, hypothermia induced ventricular fibrillation may be quite resistant to standard treatment. Electrical defibrillation often is unsuccessful at body temperature below 30°C. This possibility provides an additional reason for prevention of ventricular fibrillation during hypothermia in a variety of clinical treatments, especially in cardiac surgery.

The purpose of the present study was to evaluate and compare the efficacy of two chemicals, Lidocaine hydrochloride and Bretylium tosylate, in minimizing the incidence of ventricular fibrillation during hypothermia in anesthetized rats.

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