VOLATILE ANAESTHETICS ANDCARDIAC PROTECTION

An Essay

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in Anesthesia

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Contents

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Moataz Mostafa Omran

LIST OF FIGURES

List of tables

Introduction

Volatile anesthetic agents are liquids at room temperature, but they can be easily evaporated for administration by inhalation. All of the volatile agents are widely used in all kinds of surgeries and they all share the property of being quite hydrophobic.

The ideal volatile anesthetic agent offers smooth and reliable induction and maintenance of general anesthesia with minimal effects on other organ systems. In addition it is odorless or pleasant to inhale; safe for all ages and in pregnancy; not metabolized; rapid in onset and offset; potent; and safe for exposure to operating room staff. It is also cheap to manufacture; easy to transport and store, with a long shelf life; easy to administer and monitor with existing equipment; stable to light, plastics, metals, rubber and soda lime; non-flammable and environmentally safe**.**

None of the agents currently in use are ideal, although many have some of the desirable characteristics. Inhalation anesthetics are the most common drugs used for the provision of general anesthesia and adding only a fraction of a volatile anesthetic to the inspired oxygen results in a state of unconsciousness and amnesia.

The most popular potent inhaled anesthetics used in adult surgical procedures are sevoflurane, desflurane, and isoflurane. In pediatric cases, sevoflurane is most commonly employed. Although there are many similarities in terms of the overall effects of the volatile anesthetics (e.g., they all have a dose-dependent effect to decrease blood pressure), there are some unique differences that might influence the clinician's selection process depending on the patient's health and the surgical procedure.

Aim of the Work

This work is intended to review the updated literature regarding the use of volatile anaesthetics as a cardiac protector in the field of general anaesthesia.

The first chapter will discuss the physiologic considerations regarding the cardiac muscle. The second chapter will deal with volatile anaesthetics. While the third chapter will discuss the updated literature about the role of volatile anaesthetics in cardiac protection.

Physiologic Considerations

The heart consists of two atria and two ventricles that provide two separate circulations in series. The pulmonary circulation, a low-resistance and high-capacitance vascular bed, receives output from the right side of the heart, and its chief function is bidirectional gas exchange. The left side of the heart provides output for the systemic circulation. It functions to deliver oxygen and nutrients and remove CO_2 and metabolites from various tissue beds ***(Berne and Levy, 2001).***

PHYSIOLOGY OF THE INTACT HEART

To understand the mechanical performance of the intact heart, it is important to have knowledge of the phases of the cardiac cycle and determinants of ventricular function.

Cardiac Cycle

The cardiac cycle is the sequence of electrical and mechanical events during the course of a single heartbeat. Figure-1 illustrates (1) the electrical events of a single cardiac cycle represented by the electrocardiogram (ECG) and (2) the mechanical events of a single cardiac cycle represented by left atrial and left ventricular pressure pulses correlated in time with aortic flow and ventricular volume ***(Berne and Levy, 2001).***

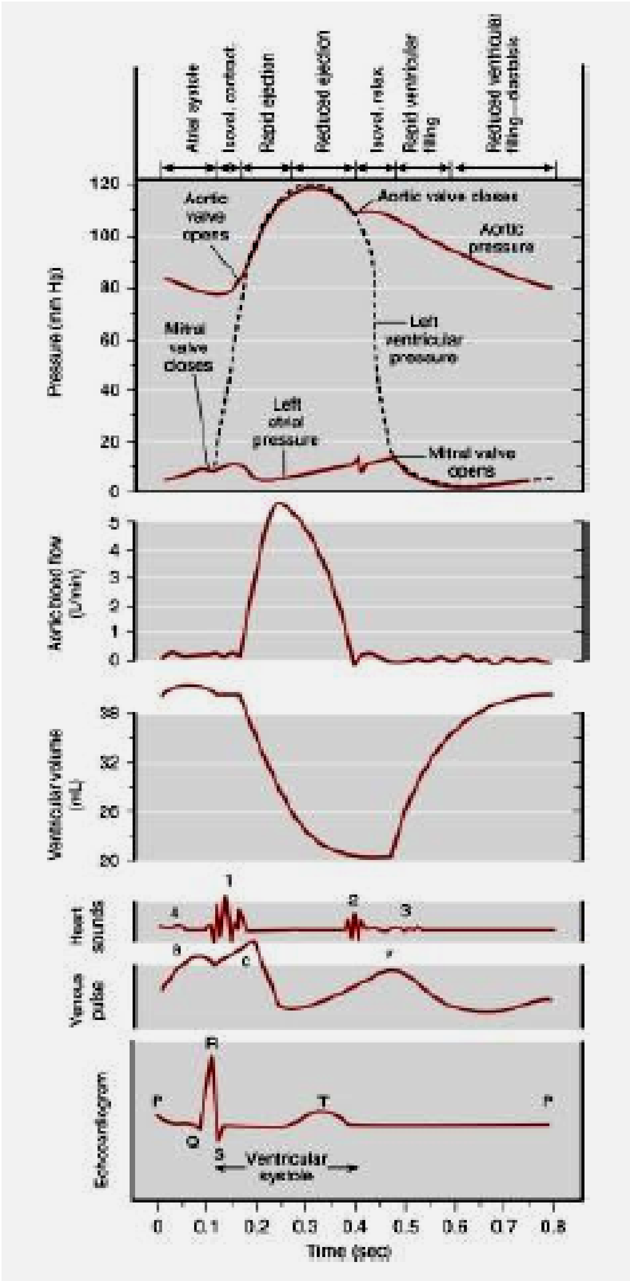

Figure-1: Electrical and mechanical events during a single cardiac cycle. Shown are the pressure curves of aortic blood flow, ventricular volume, venous pulse, and the electrocardiogram ***(Berne and Levy, 2001)***.

The cardiac cycle begins with initiation of the heartbeat. Intrinsic to the specialized cardiac pacemaker tissues is automaticity and rhythmicity. The sinoatrial (SA) node is usually the pacemaker; it can generate impulses at the greatest frequency and is the natural pacemaker ***(Berne and Levy, 2001).***

ELECTRICAL EVENTS AND THE ELECTROCARDIOGRAM

Electrical events of the pacemaker and the specialized conduction system are represented by the ECG at the body surface. It is the result of differences in electrical potential generated by the heart at sites of the surface recording. The action potential initiated at the SA node is propagated to both atria by specialized conduction tissue, and it leads to atrial systole (contraction) and the P wave of the ECG.

At the junction of the inter-atrial and inter-ventricular septa, specialized atrial conduction tissue converges at the atrio-ventricular (AV) node, which is connected distally to the His bundle. The AV node is an area of relatively slow conduction, and a delay between atrial and ventricular contraction normally occurs at this locus.

The PR interval can be used to measure the delay between atrial and ventricular contraction at the level of the AV node.

From the distal His bundle, an electrical impulse is propagated through large left and right bundle branches and finally to the Purkinje system fibers, which are the smallest branches of the specialized conduction system. Finally, electrical signals are transmitted from the Purkinje system to individual ventricular cardiomyocytes. The spread of depolarization to the ventricular myocardium is manifested as the QRS complex on the ECG. Depolarization is followed by ventricular repolarization and appearance of the T wave on the ECG ***(Berne and Levy, 2001).***

MECHANICAL EVENTS

The mechanical events of a cardiac cycle begin with return of blood to the right and left atria from the systemic and pulmonary circulation, respectively. As blood accumulates in the atria, atrial pressure increases until it exceeds the pressure within the ventricle, and the AV valve opens. Blood first flows passively into the ventricular chambers, and such flow accounts for approximately 75% of total ventricular filling.The remainder of the blood flow is mediated by active atrial contraction or systole, known as the atrial “kick’’ ***(Katz, 2001).***

The onset of atrial systole is coincident with depolarization of the sinus node and the P wave. While the ventricles fill, the AV valves are displaced upward and ventricular contraction (systole) begins with closure of the tricuspid and mitral valves, which corresponds to the end of the R wave on the ECG. The first part of ventricular systole is known as isovolumic or isometric contraction. The electrical impulse traverses the AV region and passes through the right and left bundle branches into the Purkinje fibers. This leads to contraction of ventricular myocardium and a progressive increase in intra-ventricular pressure. When intra-ventricular pressure exceeds pulmonary artery and aortic pressure, the pulmonic and aortic valves open and ventricular ejection occurs, which is the second part of ventricular systole ***(Katz, 2001).***

Ventricular ejection can be further separated into the rapid ejection phase and the reduced ejection phase. During the rapid ejection phase, forward flow is maximal, and pulmonary artery and aortic pressure is maximally developed. In the reduced ejection phase, flow and great artery pressure taper with progression of systole. Pressure in both ventricular chambers falls as blood is ejected from the heart, and ventricular diastole begins with closure of the pulmonic and aortic valves. The initial period of ventricular diastole consists of the isovolumic/isometric relaxation phase. This phase is concomitant with repolarization of the ventricular myocardium and corresponds to the end of the T wave on the ECG. The final portion of ventricular diastole involves a rapid decrease in intra-ventricular pressure until it falls below that of the right and left atria, at which point the AV valve reopens, ventricular filling occurs, and the cycle repeats itself ***(Katz, 2001).***

VENTRICULAR STRUCTURE AND FUNCTION

Ventricular Structure

The specific architectural order of the cardiac muscles provides the basis for the heart to function as a pump. The ellipsoid shape of the left ventricle (LV) is a result of the laminar layering of spiraling bundles of cardiac muscles (Fig-2). The orientation of the muscle bundle is longitudinal in the subepicardial myocardium and circumferential in the middle segment and again becomes longitudinal in the subendocardial myocardium. Because of the ellipsoid shape of the LV, there are regional differences in wall thickness that result in corresponding variations in the cross-sectional radius of the LV chamber. These regional differences may serve to accommodate the variable loading conditions of the LV***(Takayama et al., 2002).***

In addition, such anatomy allows the LV to eject blood in a corkscrew-type motion beginning from the base and ending at the apex. The architecturally complex structure of the LV thus allows maximal shortening of myocytes, which results in increased wall thickness and generation of force during systole. Moreover, release of the twisted LV may provide a suction mechanism for filling of the LV during diastole. The LV free wall and the septum have similar muscle bundle architecture. As a result, the septum moves inward during systole in a normal heart. Regional wall thickness is a commonly used index of myocardial performance that can be assessed clinically, such as by perioperative echocardiography or magnetic resonance imaging ***(Takayama et al., 2002).***

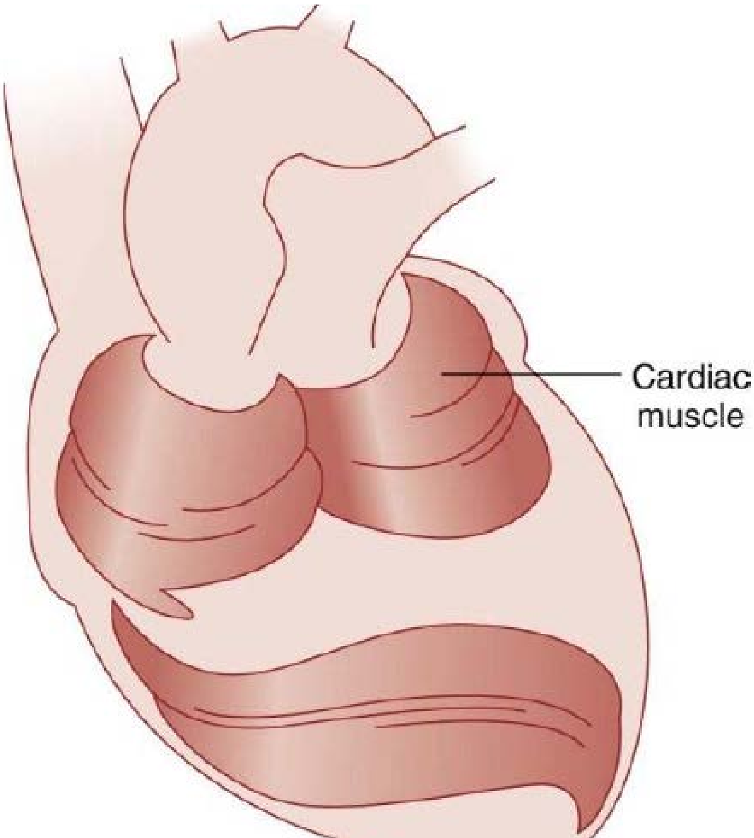

Figure-2: Muscle bundles ***(Marieb et al., 2001)***