ANESTHETIC CONSIDERATIONS FOR PATIENTS WITH CARDIAC ASSIST DEVICES

Essay

Submitted for Partial Fulfillment of Master Degree in Anesthesiology

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INTRODUCTION

The conventional treatment for a patient with cardio-vascular disease is directed towards optimization of electrolytes, acid-base balance, oxygenation, ventilation, blood volume, heart rate and rhythm. Some patients remain haemodynamically unstable in spite of maximal pharmacological support *(Richenbacher et al., 2001).*

A variety of cardiac devices are available that provide temporary mechanical support of the failing heart. Most of these devices are used after cardiac surgery, where about 5% of patients require postoperative mechanical assistance to support the cardiac output *(Golding, 1991).*

However, the cardiac assist devices are capable of supplementing and replacing cardiac pump function for variable length of time. It is assumed that if the devices used correctly in appropriate patients; mechanical circulatory assistance devices are successful in prolonging life expectancy and improving the quality of that life *(Thomas and Kramer, 1993)*.

Cardiac pacemakers, Automatic Implantable cardioverter-defibrillators (AICDs), Ventricular assist devices (VADs), Intra-aortic balloon pumps (IABPs), are currently available cardiac and circulatory assist devices *(Dinardo, 1998).*

These devices are no longer confined to merely keeping the heart beating between a minimum rate (pacing function) and a maximum rate (ICD function), as they are now being used as therapy to improve the failing heart. Both the aging of population and our ability to care for a patient with increasingly complex disease suggest that we will be caring for many more patients with these devices, and we must be prepared for this situation. Safe and efficient clinical management of these patients depends upon our understanding of implantable systems, indications for their use, and the perioperative needs that they create ***(Moss et al., 2002).***

The complexity of cardiac generators limits generalizations that can be made about the perioperative care of cardiac patients. Population aging, continued technologic enhancements, and new indications for implantation of cardiac devices will lead to increased implantations. These issues led the American Colleague of Cardiology to publish perioperative care guidelines for the patient with a pulse generator ***(Eagle et al., 2002)***.

Not all generators implanted in the chest are cardiac devices, and devices resembling cadiac pulse generators are being implanted at increasing rates for pain control, thalamic stimulation to control Parkinson's disease, phrenic nerve stimulation to stimulate the diaphragm in paralyzed patients, and

vagus nerve stimulation to control epilepsy ***(Kazatsker et al., 2002).***

Finally, this essay is focused on cardiac devices with their cardiac physiological review, their development, recent advances, haemodynamics, pathophysiologicl changes associated with their use and the anesthetic management of patients with one of these cardiac devices including preoperative, intraoperative and postoperative management.

Cardiac action potential

The cardiac action potential is the electrical activity of the cardic muscle cells, Ventricular and Atria cells, and the individual cells of the electrical conduction system of the heart. The electrical conduction system of the heart has a special property of depolarizing without any external influence. This is known as automaticity *(Vander et al., 2001).*

The magnitude of the intracellular potentials depends on the relative permeability of the membrane to Na^+, Ca^{2+}, and K^+. When the membrane is much more permeable to K^+ than to Na^+ or Ca^{2+} (as occurs in the resting state), the measured potential is close to that which would exit if the membrane were permeable only to K^+ (potassium equilibrium potential). In contrast, when the membrane is more permeable to Na^+ than to other ions (as occurs at the peak of phase 0 of the action potential), the measured potential is closer to the potential that would exit if the membrane were permeable only to Na^+ (sodium equilibrium potential) (Fig. *1) (Katz, 1992).*

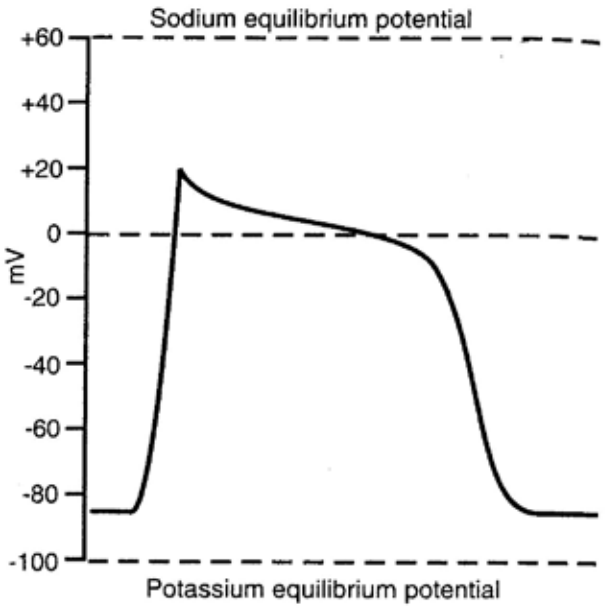

Fig. (1): *(Vander et al., 2001)*

Phases of the cardiac action Potential

Phase 4 (Resting Membrane Potential)

The resting (diastolic) membrane potential of ventricular cells is maintained by K^+ channels that are open at highly negative membrane potentials. They are called inward rectifier K^+ channels because when the membrane is depolarized they no longer permit outward movement of K^+. This phase of action potential is associated with diastole of the heart (Fig. 2) ***(Rhoades and Tanner, 1995).***

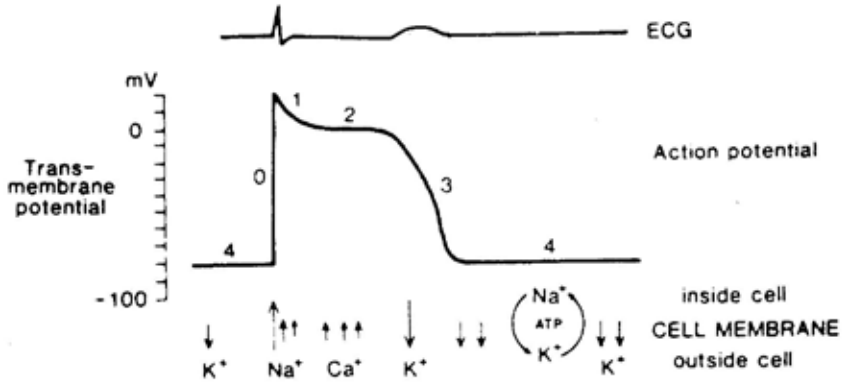

Fig. (2): Phasis of the cardiac action Potential ***(Kaplan et al., 1999)***

Phase 0

Phase 0 is known as the rapid depolarization phase. The slope of phase 0 is determined by the maximum rate of depolarization of the cell and is known as V-max This phase is due to opening of the fast Na^+ channels and the subsequent rapid increase in the membrane conductance to Na^+ and a rapid influx of ionic current in the form of Na^+ ions into the cell. Since permeability to Na^+ exceeds that to K^+ the membrane potential approaches the Na^+ equilibrium potential, so the inside of the cell becomes positively charged relative to the outside (Fig. 2) ***(Sperelakis, 1989).***

Phase 1

Phase 1 of the ventricular action potential is related to a decrease in the number of open Na^+channels and an opening of transient outward rectifier K^+channels. These changes tend to repolarize the membrane slightly. Phase 0 and 1 together

correspond to the R and S waves of the ECG (Fig. 2) ***(Rhoades and Tanner, 1995).***

Phase 2 (selective influx of calcium)

Phase 2 is often known as the plateau phase of the action potential, because of relatively small change in the membrane potential compared to phase 0. while the membrane potential does not change much during phase 2, and it results from a combination of low membrane permeability to K^+ due to closed K^+ channels and an increased permeability to Ca^{2+} due to opening of Ca^{2+} channels,causing the membrane potential to become more negative and approaches the K^+ equilibrium potential (Fig. 2) ***(Rhoades and Tanner, 1995).***

Phase 3 (Selective Efflux of Potassium)

The return of the membrane potential to the resting state during phase 3 reflects the closing of Ca^{2+} channels and the opening of delayed outward rectifier K^+ channels. This relative increase in permeability to K^+ drives the membrane potential toward the K^+ equilibrium potential. Phase 3 of the action potential corresponds to the T-wave on the ECG (Fig. 2) ***(Rhoades and Tanner, 1995).***

* Electrical conduction system of the heart

Pacemaker cells in the sinoatrial (SA) node spontaneously depolarize and a wave of depolarization spreads over the atria and into the atrioventricular

(AV) node through specialized pathways known as internodal tracts (Fig. 3) (*Chawhan et al., 2001).*

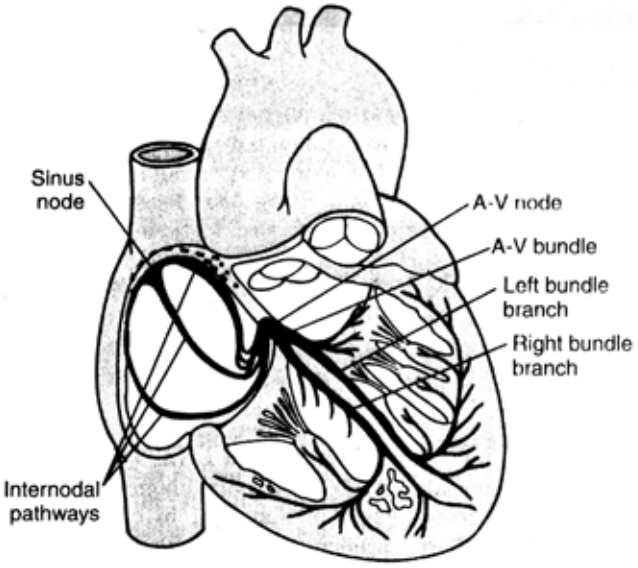

Fig. (3): Sinus node and the Purkinje system of the heart, showing also the A-V node, atrial internodal pathways, and ventricular bundle branches *(Gyuton and Hall, 2000)*.

The AV node functions as a critical delay in the conduction system. Without this delaly, the atria and ventricles will contract at the same time, and blood won't flow effectively from the atria to the ventricles. The delay in the AV node forms much of PR interval on the ECG. The distal portion of the AV node is known as the Bundle of His. The Bundle of His spilts into two branches in the interventricular septum, the left bundle branch and the right bundle branch. The left bundle branch activates the left ventricle, while the right bundle branch activates the right ventricle. The left bundle branch is short, splitting into the left

anterior fascicle and the left posterior fascicle. The left posterior fascicle is relatively short and broad, with dual blood supply, making it particulary resistant to ischemic damage. The two bundle branches taper out to produce numerous Purkinje fibers, which stimulate individual groups of myocardial cells to contract *(Des Jardins, 1998).*

**** Transformation of non-pacemaker into pacemaker cells***

It is important to note that non-pacemaker action potential cells can change into pacemaker cells under certain conditions. For example, if a cell becomes hypoxic, the membrane depolarizes, which closes the fast Na^+ channels. At a membrane potential of about -50 mV, all the fast Na^+ channels are inactivated. When this occurs, action potential can still be elicited; however, the inward current are carried by Ca^{2+} (slow inward channels). These action potentails resmble those found in pacemaker cells located in the SA node, and can display spontaneous depolarization and automaticity. This mechanism may serve as the elctrophysiogical mechanism behind certain types of ectopic beats and dysrhythmias, particularly in ischemic heart disease and following myocardial infarction *(Klabunde, 2004).*

The Cardiac Cycle:

The cardiac events that occur from the beginning of one heartbeat to the beginning of the next are called the cardiac cycle. Each cycle is initiated by spontaneous generation of an action potential in the sinus node. The cardiac cycle consists of a period of relaxation called diastole, during which the heart fills with blood, followed by a period of contraction called systole *(Guyton and Hall, 2000).*

Figure (4) shows the different events during the cardiac cycle for the left side of heart. The top three curves show the pressure changes in the aorta, left ventricle, and left atrium, respectively. The fourth curve depicts the changes in ventricular volume, the fifth the electrocardiogram, and the sixth a phonocardiogram, which is a recording of the sounds produced by the heart, mainly by the heart valves,as it pumps.

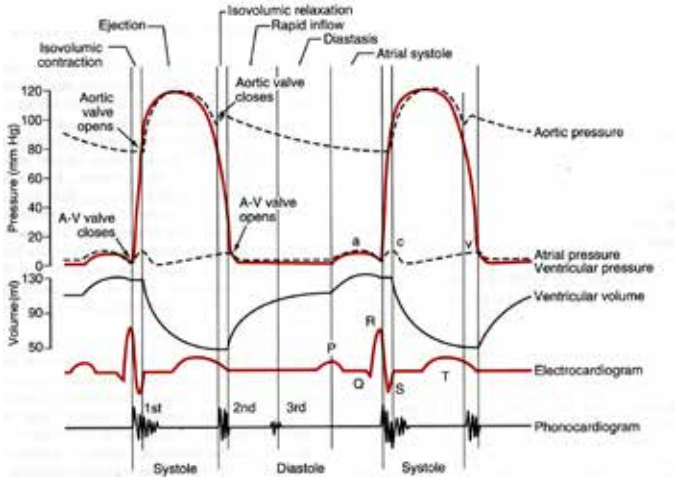

Fig. (4): Phases of the cardiac cycle *(Gyuton and Hall, 2000)*