ACUTE EFFECT OF PERCUTANEOUS TRANSLUMINAL BALLOON VALVULOPLASTY ON QT INTERVAL EVIDENCE FOR CONTRACTION-EXCITATION FEEDBACK IN HUMANS

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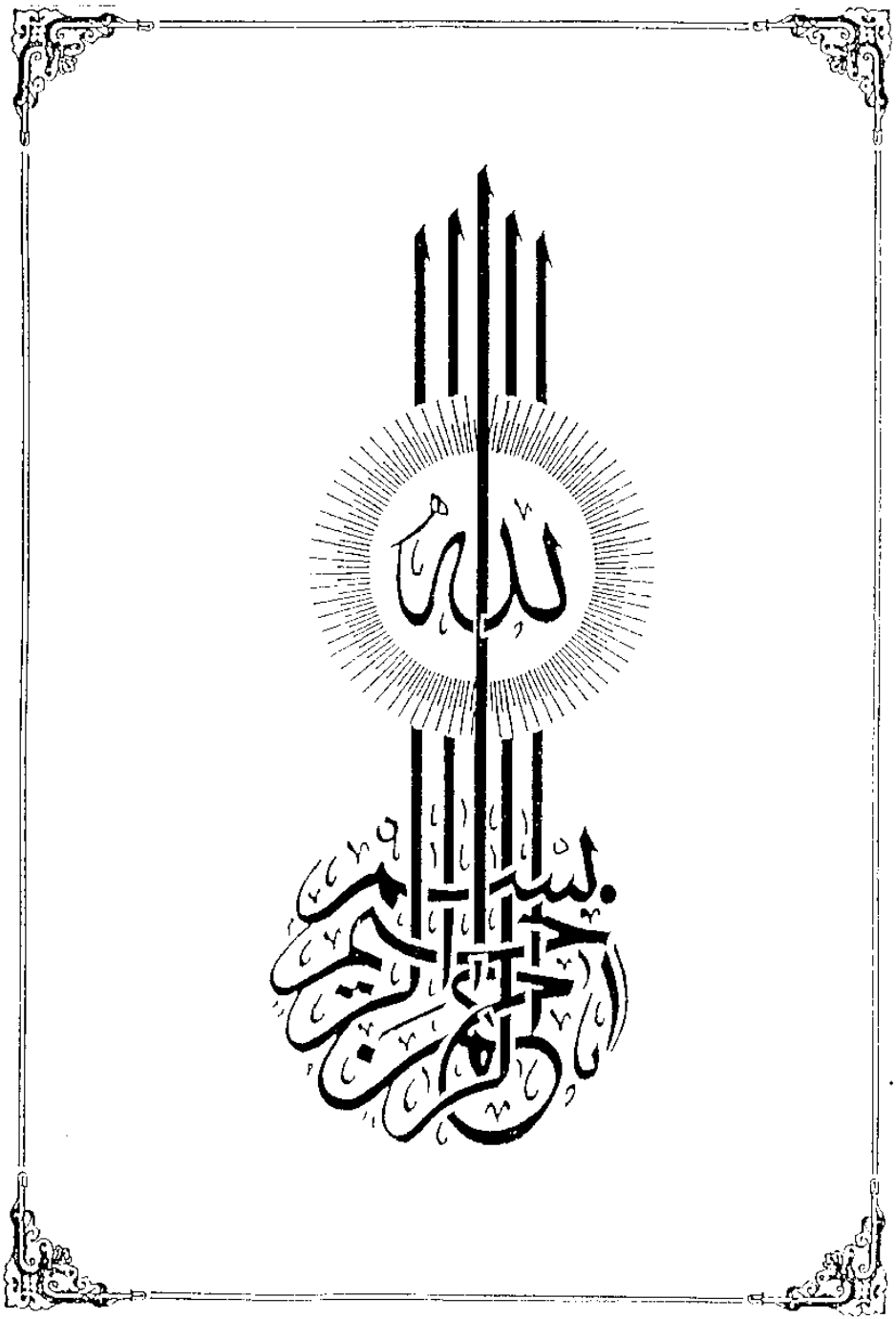

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INTRODUCTION

* Percutaneous transluminal balloon angioplasty is a technique that has safely applied as treatment for peripheral vascular **(Dotter et al 1964-Tegtmeyer et al 1980)** and coronary artery disease **(Gruntzig et al 1979)**, hypoplastic and stenotic pulmonary arteries **(Lock et al 1983)**, aortic coarctation **(Lock et al 1983)**, venous obstruction **(Lock et al 1984)**, pulmonic stenosis **(Kan et al 1982)** and mitral and congenital aortic stenosis in childern and young adults **(Lock et al 1985).**

* The QT interval in the surface electrocardiogram is an indirect measure of cardiac action potential duration. **(Yamashita et al 1991).**

* The QT interval is modified by the preceding R-R interval , by autonomic nervous tone **(Lecoeq et al 1989)** and by mechanical load on cardiac muscle **(Lab, 1982)**. Percutaneous transluminal balloon valvuloplasty alters mechanical load on cardiac muscle **(Yamashita et al 1991)**,thus alters Qt interval according to contraction-excitation feedback theory which has been primairly investigated in isolated muscle preparations or nonmammalian hearts **(Lerman et al 1985).**

In this study we investigated contraction-excitation feedback theory in mammalian hearts by studing the acute effect of transluminal balloon valvuloplasty of mitral, pulmonary and aortic stenosis on QT interval.

AIM OF THE WORK

Our aim is to aprove a certain about mechanism of ventricular arrhythmia that may follow Percutancous transluminal balloon valvuloplasty of mitral , pulmonary and aortic stenosis .

REVIEW OF LITERATURE

CELLULAR ELECTROPHYSIOLOGY OF THE HEART

* Recent advances in electrophysiologic techniques has greatly increased our insight into the nature of the ionic translocations underlying the transcription of action potential in different myocardial fibers, healthy as well as diseased .

* Basic knowledge of the normal electrical events of the single fiber is crucial in understanding the formation and propagation of the cardiac impulse **(Bellet, 1971)**

The resting membrane potential :

* The cell membrance has openings called the channels that serve as conduits through which ions move **(Miura et al 1977)**

* Ions are positively (cations) or negatively (anions) charged atoms such as Na+, K+, Ca+, or Cl^- and other molecules whose movement inside the cell or across the cell membrane constitutes a flow of current **(Miura et al, 1977).**

* It is the flow of current that generates signals in excitable membranes . The intracellular potassium concentration $(K)^{+}_{i}$ is about thirty times greater than the extracellular concentration $(K)^{+}_{o}$. In purkinje fibers, for example, $(K)^{+}_{i}$ and $(K)^{+}_{o}$ are 140 to 150 mM amd 4 to 5 mM respectivly **(Miura et al 1977).**

* On the contrary the intracellular Sodium cocentration $(Na)^{+}_{i}$ is much smaller than the exracellular concentration $(Na)^{+}_{o}$, $(Na)^{+}_{i}$ and $(Na)^{+}_{o}$ are usually about 10 mM and 150 mM respectivly in pukinje fibers **(Ellis 1977).**

* There are forces governing the movement of K^{+} across the cell memberane at rest. Because of the concentration gradient, K^{+} ions tend to diffuse out of the cell building up a negative charge in the interior of the cell , electoneutrality cannot be maintained by an outward movement of cellular anions because they are mainly large ions and often associated with protein to which the cell memberane is impermeable **(Carmeliet, 1978).**

* When the interior of the cell membrane becomes sufficiently negative so that the inwardly directed electrostatic attraction for K^{+} is equal the outwardly

directed force due to concentrarion gradient, the net movement of K^+ ceases **(Carmeliet, 1978)**.

* The intracellular potenial at which the net positive flux of potassium ions is zero, is called "equilibrium potential" for potassium ions, (EK) and its value is expressed by the " Nernest equation" :-

$$Ek = RT/F \quad \text{in } (K)^+_o /(K)^+_i$$

Where :- R is the gas constant.

T is the absolute tempreture.

F is Faraday's constant.

$(K)^+_o$ is extracellular potassium concentration.

$(K)^+_i$ is the intracellular potassium concentration.

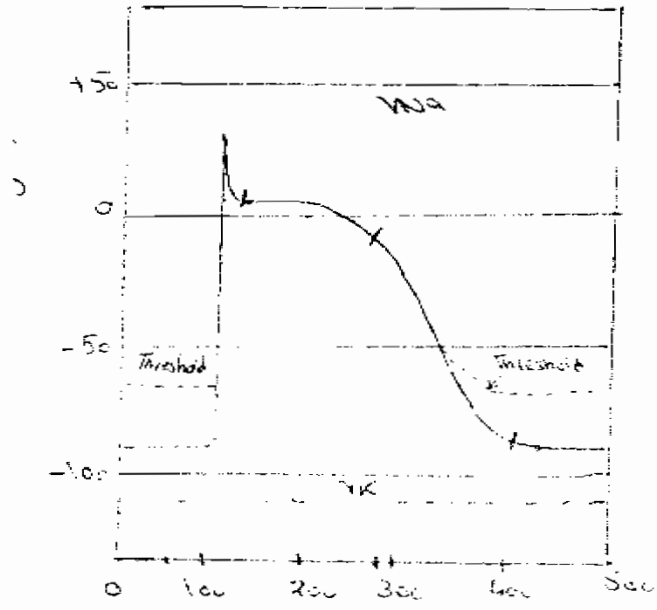
The equilibrium potential for potassium ions (EK) equal 90 mv which is nearly equal the resting membrane potential actually recorded.This finding suggests that the potassium equilibrium potential is the major factor responsible for resting transmemberane potential **(Hoffman and Cranefield 1960).**

* The intracellular potential during electrical quiescence in diastole is -50 to -95 mv, depending on the

cell type this means that the inside of the cell is 50 to 95 mv negative relative to the outside of the cell owing to the distribution of ions such as K^+, Na^+, Cl^- and Ca^{++} across the cell membrane **(Fozzard, 1977)**

* During diastole the cell membrane is quite permeable to K^+ and relatively impermeable to Na^+, because of the Na-K pump, which pumps Na^+ out of the cell against its electrochemical gradient and simultaneously pumps K^+ into the cell against its chemical gradient **(Zipes 1984)**. This pump fueled by an Na-K ATP ase enzyme that hydrolyzes ATP for energy, is bound to the membrane and it requires both Na^+ and K^+ to function and can transport three Na+ ions outward for two K^+ ions inward **(Zipes 1984)**. ATPase acts as a carrier that transports potassium inward at the same time **(Gyton, 1982).**

*** The Electrical Basis Of Cardiac Action Potenial :-**



(Fig 1) Aschematic diagram of the cardiac action potenial and the sequence of events that determine the time change in voltage.VNa = soduim equilibrium potential , VK = potassium equilibrium potential. The five phases of the action potential are noted. **(Gyton, 1982)**

* On excitation, there is a sequence of ions traslocation resulting in the appearance of the so-called action potential two types of action potential are observed in the heart. **(Wit et al 1974).**

One type, the so-called fast response, occurs in the normal myocardial fibers in the atria and ventricles and purkinje fibers. The other type of action potential, the so-called slow response is found in the SA node and AV node **(Wit et al 1974).**

* In normal atrial and ventricular muscle and in the fibers of the His-purkinje system action potential have very rapid upstroke with a large Vmax (the maximum rate of change of voltage over time) and are called the fast response. Action potential in the normal sinus and atrio-ventricular node have very slow upstrokes with reduced Vmax and are called slow response **(Glimour et al 1982).**

The upstroke of slow response are mediated by a slow inward, predominantly Ca^{++} current rather than the fast inward Na^{+}current. These potentials recieved the name "slow response" because the time required for activation (10-20 msec) and inactivation (50-500 msec) of the slow inward current (Isi) is considerbly longer than that for the fast inward Na current (INa^{+}). **(Glimour et al 1982).**