Postoperative Use of Inotropic Drugs in Patients Undergoing Open Heart Surgery

Essay
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By

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List of abbreviations

ADP	Adenosine diphosphate
ATP	Adenosine triphosphate
A-V	Atrioventricular
CABG	Coronary artery bypasses grafting
CAD	Coronary artery disease
Ca	Calcium
CHF	Congestive heart failure
CPB	Cardiopulmonary bypass
CK-MB	myocardial creatine kinase isoenzyme
CHD	Congenital heart disease
CPP	Coronary perfusion pressure
EP	Epinephrine
EF	Ejection fraction
H	Hydrogen
K	Potassium
LAD	Left anterior descending coronary artery
LCX	left circumflex coronary artery
LMCA	Left main stem coronaryartery
LCOS	Low cardiac output syndrome
LV	Left ventricle
LVF	Left ventricular failure
Mg	Magnesium
MVO2	myocardial oxygen demand
Na	Sodium
NE	Norepinephrine
PVR	Pulmonary vascular resistance
RCA	Right coronary artery
RV	Right ventricle
RVF	Right ventricular failure
SVR	Systemic venous resistance
SWMA	Segmental wall motion abnormality
TEE	Transesophageal echocardiography

Chapter 1

Physiology of the heart

Physiology of the Cardiac muscle

The heart is composed of three major types of cardiac muscles: atrial, ventricular, and specialized excitatory and conductive muscle fibres (Figure 1). The atrial and ventricular types of muscle contract in much the same way as skeletal muscle, except that the duration of contraction is much longer. Conversely, the specialized excitatory and conductive fibers contract only feebly because they contain few contractile fibrils; instead they exhibit either automatic rhythmical electrical discharge in the form of action potentials or conduction of the action potentials through the heart, providing an excitatory system that controls the rhythmical beating of the heart. **(Guyton, 2006)**

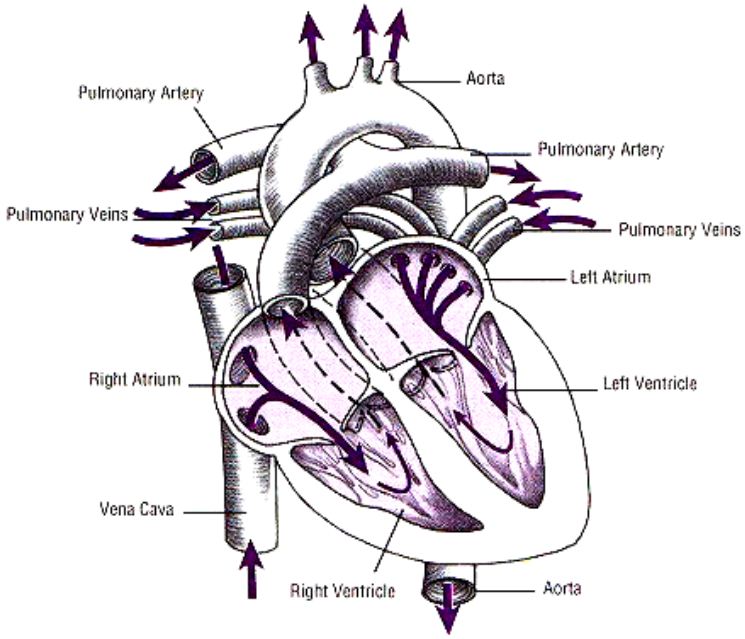

Figure 1: Structure of the heart and course of blood flow through the heart chamber and heart valves. (Guyton, 2006)

Physiological anatomy of cardiac muscle

Cardiac muscle fibers are arranged in latticework, with the fibers dividing, recombining, and then spreading again. The cardiac muscle is striated in the same manner as in typical skeletal muscle. It has typical myofibrils that contain actin and myosin filaments almost identical to those found in skeletal muscle; these filaments lie side by side along one another during contraction in the same manner as occurs in skeletal muscle.

Cardiac muscle as a syncytium

The intercalated discs are actually cell membranes that separate individual cardiac muscle cells from one another. That is, cardiac muscle fibers are made up of many individual cells connected in series and in parallel with one another (Figure 2) **(Guyton, 2006**).

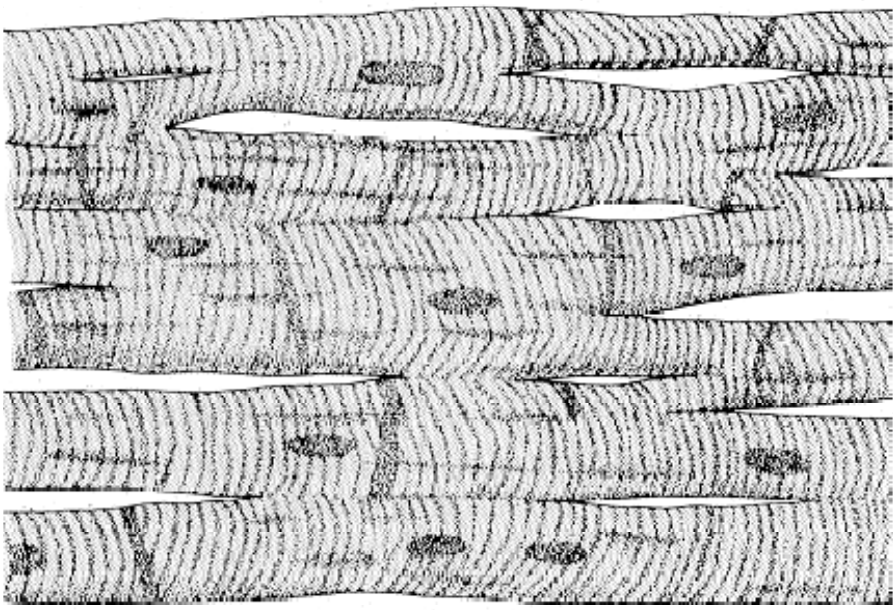

Figure 2: "Syncital." Interconnecting nature of cardiac muscle **(Guyton, 2006).**

At each intercalated disc the cell membranes fuse with one another in such a way that they form permeable "communicating" junctions (gap junctions) that allow most totally free diffusion of ions. Therefore, from a functional point of view, ions move with ease in the intercellular fluid along the longitudinal axes of the cardiac muscle cell to the next, past the intercalated discs. Thus, cardiac muscle is a syncytium of many heart muscle cells in which the cardiac cells are so interconnected that when one of these cells becomes excited, the action potential spreads to all of them, spreading from cell to cell throughout the latticework interconnections. The heart actually is composed of two syncytiums: atrial syncytium that constitutes the walls of the two atria, and the ventricular syncytium that constitutes the walls of the two ventricles. The atria are separated from the ventricles by fibrous tissue that surrounds the atrioventricular (A-V) valvular openings between the atria and the ventricles. Normally, potentials are not conducted from the atrial syncytium directly through the fibrous tissue. Instead, they are conducted only by way of specialized conductive fibers several millimeters in diameter. These divisions of the muscle of the heart into two functional syncytiums allow the atria to contract a short time ahead of ventricular contraction, which is important for effectiveness of heart pumping. **(Guyton, 2006)**

Action Potentials in Cardiac Muscle

The action potential recorded in a ventricular muscle fiber averages about 105 millivolts, which means the intercellular potential rises from a very negative value, about –85 millivolts between beats, to a slightly positive value, about +20 millivolts, during each beat. After the initial spike, the membrane remains depolarized for about 0.2 second, exhibiting a plateau, following at the end of the plateau by abrupt repolarization. The presence of this plateau in the action potential causes ventricular contraction to last as much as 15 times as long in cardiac muscle as in skeletal muscle **(Figure 3)**. **(Brette F. et al, 2003)**

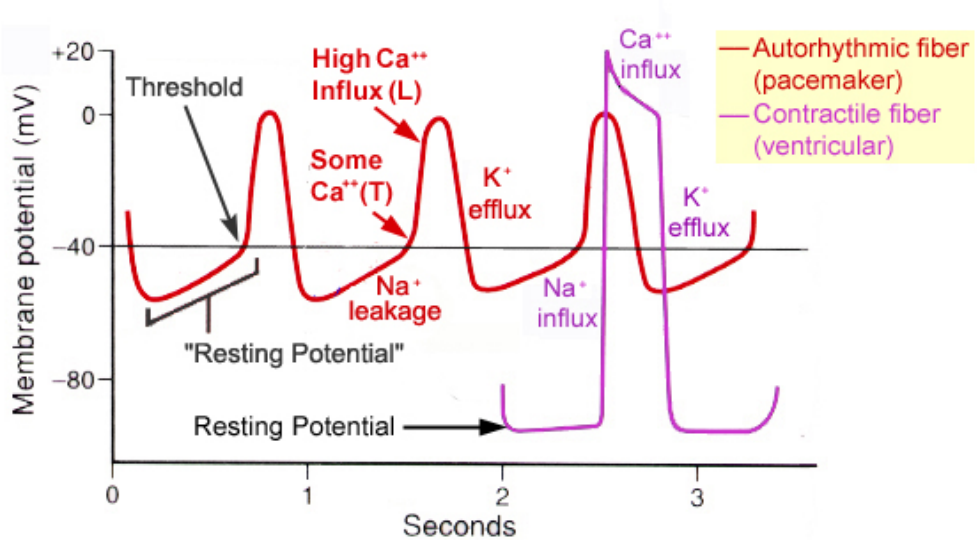

Figure 3: Action Potential in Cardiac Muscle **(Brette F. et al, 2003)**

The action potential is caused by opening of two types of channels; the fast sodium channels and slow calcium channels, which are also called calcium-sodium channels. This second population of channels differs from the fast sodium channels in that they are slower to open and, even more important, remain open for several tenths of a second. During this time, a large quantity of both calcium and sodium ions flow through these channels to the interior of the cardiac muscle fiber, and this maintains a prolonged period of depolarization, causing the plateau in the action potential. Further, the calcium ions that enter during this plateau phase activate the muscle contractile process **(Brutsaert DL, 2003).**

Immediately after the onset of the action potential, the permeability of the cardiac muscle membrane for potassium ions decreases about five fold. This decreased potassium permeability may result from the excess calcium influx through the calcium channels just noted. Regardless of the cause the decreased potassium permeability greatly decreases the outflux of positively

charged potassium ions during the action potential plateau and thereby prevents early return of the action potential voltage to its resting level. When the slow calcium-sodium channels close at the end of 0.2 to 0.3 second and the influx of calcium and sodium ceases, the membrane permeability for potassium ions also increases rapidly; this rapid loss of potassium from the fiber immediately returns the membrane potential to its resting level, thus ending the action potential **(Brette F. et al, 2003).**

Velocity of signal conduction in Cardiac muscle.

The velocity of conduction of the excitatory action potential signal along both atrial and ventricular muscle fibers is about 0.3 to 0.5 m/sec or about 1/250 of the velocity in very large nerve fibers and about 1/10 the velocity in skeletal muscle fibers. The velocity of conduction in the specialized heart conductive system- in the purkinje fibers- is as great as 4m/sec in most parts of the system, which allows reasonably rapid conduction of the excitatory signal to the different parts of the heart. **(Brette F. et al, 2006)**

Refractory period of Cardiac muscle.

Cardiac muscle, like all excitable tissue, is refractory to restimulation during the action potential. Therefore, the refractory period of the heart is the interval of time during which a normal cardiac impulse cannot re-excite an already excited area of cardiac muscle. The normal refractory period of the ventricle is 0.25 to 0.3 second, which is about the duration of the prolonged plateau action potential. There is an additional relative refractory period of about 0.05 second during which the muscle is more difficult than normal to excite but nevertheless can be excited by a very strong excitatory signal (premature contraction). The

refractory period of atrial muscle is much shorter than for the ventricles, about 0.15 second for the atria compared with 0.25 to 0.30 second for the ventricles. **(Guyton, 2006)**

Excitation – Contraction Coupling – Function of Calcium ions and the transverse tubules

The term "excitation- contraction coupling" refers to the mechanism by which the action potential causes the myofibrils of muscle to contract. There are differences in the mechanism in cardiac muscle that has important effects on the characteristics of cardiac muscle contraction. When an action potential passes over the cardiac muscle membrane, the action potential spreads to the interior of the cardiac muscle fiber along the membranes of the transverse (T) tubules. The T tubule action potentials in turn act on the membranes of the longitudinal sarcoplasmic tubules to cause release of calcium ions into the muscle sarcoplasm from the sarcoplasmic reticulum. In another few thousands of a second, these calcium ions diffuse into the myofibrils and catalyze the chemical reactions that promote sliding of the actin and myosin filaments along one another; this produces the muscle contraction. (Figure 4) **(Bers, 2002)**

In addition to the calcium ions that are released into the sarcoplasm from the catenae of the sarcoplasmic reticulum, a large quantity of extra calcium ions also diffuse into the sarcoplasm from the T tubules themselves at the time of the action potential, without this extra calcium from the T tubules, the strength of cardiac muscle contraction would be reduced considerably because the sarcoplasmic reticulum of cardiac muscle is less well developed than that of skeletal muscle and does not store enough calcium to provide full contraction **(Bers, 2002)** .Conversely, the T tubules of cardiac muscle have a diameter 5 times as great as that of the skeletal muscle tubules,

which means a volume 25 times as great. Also, inside the T tubules is a large quantity of mucopolysaccharides that are electronegatively charged and bind an abundant store of calcium ions, keeping these always available for diffusion to the interior of the cardiac muscle fiber when a T tubule action potential appears **(Brette F, et al, 2003).**

The strength of contraction of cardiac muscle depends to a great extent on the concentration of calcium ions in the extracellular fluid. The reason for this is that the openings of the T tubules pass directly through the cardiac muscle cell membrane into the extracellular spaces surrounding the cells, allowing the same extracellular fluid that is in the cardiac muscle interstitium to percolate through the T tubules as well. Consequently, the quantity of calcium ions in the T tubule system—that is, the availability of calcium ions to cause cardiac muscle contraction—depends to a great extent on the extracellular fluid calcium ion concentration **(Brette F, et al, 2003).**

At the end of the plateau of the cardiac action potential, the influx of calcium ions to the interior of the muscle fiber is suddenly cut off, and the calcium ions in the sarcoplasm are rapidly pumped back out of the muscle fibers into both the sarcoplasmic reticulum and the T tubule—extracellular fluid space. As a result, the contraction ceases until a new action potential comes along (**Brette F, et al, 2003).**

Cardiac muscle begins to contract a few milliseconds after the action potential begins and continues to contract until a few milliseconds after the action potential ends. Therefore, the duration of contraction of cardiac muscle is mainly a function of the duration of the action potential, including the plateau about 0.2 second in atrial muscle and 0.3 second in ventricular muscle (**Gyuton, 2006).**

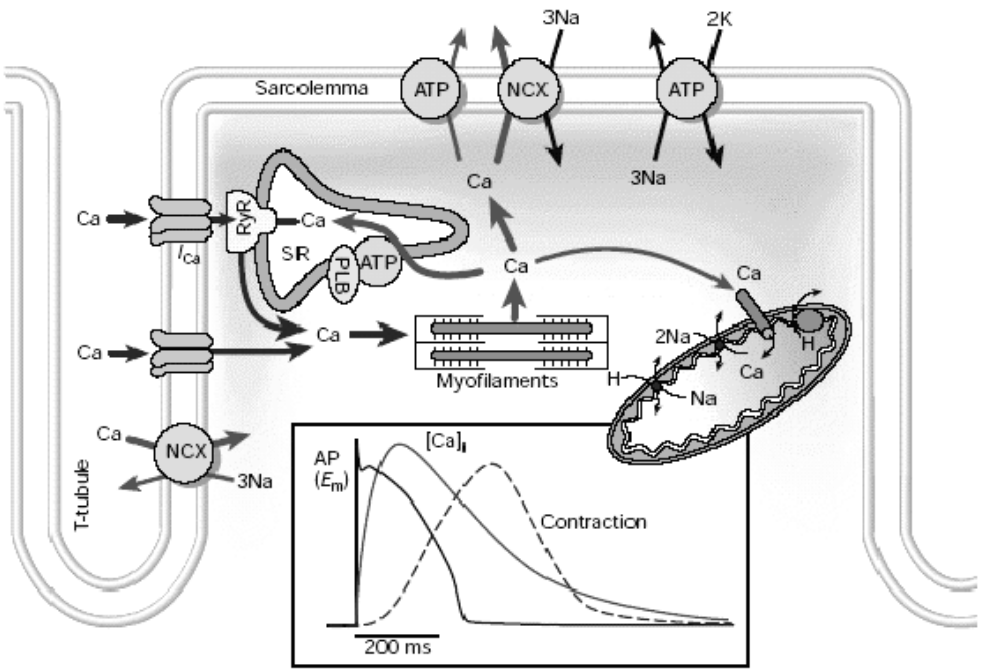

Figure 4: Scheme of cardiac excitation-contraction-coupling events in a ventricular myocyte **(Bers 2002).**

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. This node is located in the superior lateral wall of the right atrium near the opening of the superior vena cava, and the action potential travels from here rapidly through both atria and then through the A-V bundle into the ventricles. Because of this special arrangement of the conducting system from the atria into the ventricles, there is a delay of more than 0.1 second during passage of the cardiac impulse from the atria into the ventricles. This allows the atria to contract ahead of

ventricular conduction, thereby pumping blood into the ventricles before the strong ventricular contraction begins. Thus, the atria act as primer pumps for the ventricles, and the ventricles in turn provide the major source of power for moving blood through the body's vascular system **(Ganong, 2005).**

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 (Figure 5) **(Ganong, 2005).**

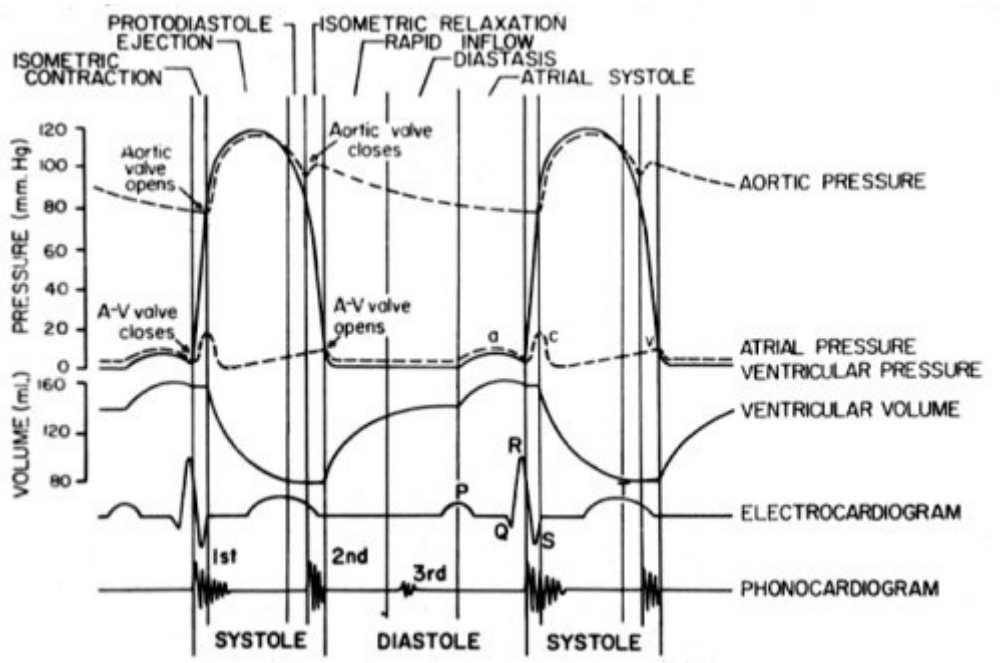

Figure 5: The figure shows the different events during the cardiac cycle for the left side of the heart. The top three curves show the pressure changes in the aorta, left ventricle, and the left ventricular volume, the fifth the electrocardiogram, and the sixth a phonocardiogram, which is a recording of the sounds produced by the heart- mainly by the heart valves- as it pumps **(Ganong, 2005).**