Cardiac and/or circulatory assist has been widely implemented as a means for reversing the process of left ventricular failure and its attendant sequela. The myocardial impairment is secondary to myocardial ischemia and/or myocardial infarction. Throughout the treatment of heart disease many divergent medical and mechanical techniques of heart assist have been utilized for the treatment of the depressed state of the myocardium. Presently one of the most widely used devices in the clinical setting for cardiac assist is the intra-aortic balloon pump. At the present time there are many types of intra aortic balloon pumps on the market. However, it is not the purpose of this presentation to compare and contrast the specific operational aspects of each of these machines. My purpose is to present to you the fundamental concepts that apply to any intra aortic balloon pump presently available. Let us establish a foundation of working knowledge by defining an intra aortic balloon. An intra aortic balloon is an internal volume displacement device made out of synthetic material that transiently changes the relative fluid volume in the central arterial vascular space. The balloon itself is integrally attached to a catheter which allows the balloon to be connected to a power console or a pump and this power console or pump provides the vehicle for the interfacing of the balloon with the patient and with the balloon pump operator.

In the clinical situation the population of patients that undergo treatment with the intra aortic balloon pump can be placed into three general categories. The first category are patients who present with cardiogenic shock solely resulting from a myocardial infarction or post cardiopulmonary bypass left ventricular failure. The second category of patients are those who demonstrate through history of angina and ekg changes, ongoing unstable myocardial ischemia of an acute or chronic nature. These patients may also present with various cardiac arrhythmias or may show a stable cardiac pattern. In the third category are patients who present with cardiogenic shock but also present with evidence for either a post infarction ventricular septal rupture or those patients who show evidence for acute mitral regurgitation which is a result of papillary muscle dysfunction or frank rupture of the chordiac tendineae or subcordial mechanism.

Cardiogenic shock has been defined by a number of criteria. Four criteria are most important. 1) A arterial blood pressure which is less than 90 mm of mercury systolic. 2) A pulmonary capillary wedge pressure which is greater than 18 mm of
mercury. 3) The cardiac index is less than 2.2 liters per minute per meter square. 4) There is evidence of secondary organ failure such as low urinary output (less than 20 mls per hour) or complete anuria, azotemia, and/or cerebral dysfunction.

The result of this CGS state is a vicious cycle of events. There is an increased work load on the myocardium with an ongoing decreasing oxygen supply to it, as a result of the impaired coronary circulation. In order to stem the tide of events, this cycle must be interrupted. The intra aortic balloon provides the needed intervention.

Once the patient has been selected for intra aortic balloon therapy the balloon is most often inserted through the femoral artery and is positioned in the descending thoracic aorta so that the tip of the balloon catheter rests just below the left subclavian artery.

It is then possible by setting the various controls on the pump console for the balloon to be electronically timed so that it operates in a synchronized manner with the heart on the principle of counter-pulsation.

Counterpulsation is defined as an artificial process of creating pressure changes in the arterial vascular space out of phase with the action of the left ventricle. In general the event that initiates the balloon timing sequence can be one of two events. It can be the R wave of the ekg when using the ekg trigger or the upstroke of the systolic pressure wave form when utilizing the pressure trigger. Some balloons presently on the market come equipped with an internal triggering device which adapts the balloon pump for other applications such as pulsatile assist with an external pulsator or pulsatile use of the balloon inserted into the aorta during cardiopulmonary bypass while the heart is fibrillating. Balloon timing procedures vary from machine to machine, due to the nature of the differing operational characteristics inherent in each of the balloon pumps. These operational characteristics must be taken into account when timing the balloon.

No matter what balloon system is used one wants to accomplish the following in timing the balloon. First, inflation of the balloon in diastole at the moment of aortic valve closure. Second, maintenance of the balloon in the inflated state during the entire diastolic phase of the cardiac cycle, and lastly deflation of the balloon immediately prior to systole.

In terms of cardiac and circulatory assist, counterpulsation ideally achieves the following results: increase in the coronary blood flow, increase in the overall mean arterial pressure, increase in the stroke volume of the left ventricle, an increase in the patient’s cardiac output and a decrease on the work load of the left ventricle will result.

In order to understand the benefits of the balloon we must first briefly review the anatomy and physiology of the heart. The heart is a four-chambered organ which is divided into “two” sides. Each side has its function and is supplied with a system of one way valves which open and close in response to the pressures of the various chambers of the heart. The pressures result from the heart’s contraction. The right side of the heart pumps blood into the lungs via the pulmonary vasculator and the left side of the heart pumps blood throughout the body via the aorta and the arterial system. At the base of the left ventricle is a unidirectional valve which is responsible for the flow of blood out of the heart and into the aorta in such a man-
ner that the blood does not return into the heart. This is the aortic valve which is tricuspid in nature.

Blood flows through the circulatory system as a result of the pulsatile action of the heart. The heart is constantly being cycled through systole and diastole in response to an electrical stimulus. This stimulus can be natural or it can be artificially induced by the action of a pacemaker. In systole the ventricular musculature is contracting and as a result blood is driven out of the ventricles and into the lungs and the body. During this time the coronary arteries are literally being strangled by the squeezing muscle mass. As a result, little if any blood flows through them. In diastole the ventricles are relaxing and blood enters them through the contracting atria and at the same time the aortic valve closes in response to the back flow of blood against it. Unlike the systemic circulation, the coronary circulation receives the greatest supply of oxygen rich blood during diastole.

In reviewing the cardiac cycle and the volume changes in the heart during this cycle, the electrical event precedes the mechanical event in a short span of time. During dyastole the volume in the ventricles is increasing. Then during systole the blood is ejected out of the ventricles as the pressure in the left ventricle overcomes that in the aorta and the aortic valve opens. The volume in the ventricle decreases sharply until the pressure in the aorta is greater than that in the ventricles again during diastole. Then the aortic valve closes producing the dicrotic notch and the ventricular pressure falls while the systemic pressure decreases producing the diastolic run off. This is the pressure and the volume changes in the ventricle during the cardiac cycle in relation to the ekg.

The counter pulsatory action of the balloon provides augmentation of the diastolic pressure. Therefore, the increase in the systemic diastolic pressure is a response to the intra-aortic volume increase during diastole. This augmentation results in an increase in mean arterial pressure and increase in coronary blood flow.

Presently the balloons that are marketed are either of a unidirectional or bi-directional type. With a uni-directional balloon the blood volume in the central aorta is displaced in one direction that is proximal to the balloon. This results in an increase in blood flow back towards the coronary arteries and the aortic arch vessels. With a bi-directional balloon, the blood is displaced both proximally and distally from the balloon. In addition, balloons come in various sizes and shapes and necessarily need to be matched to the patient’s size of aorta.

Having discussed two of the four ideal results of balloon pulsation, those are the increase in coronary blood flow and the increase in arterial blood pressure, we will now turn our discussion to the other two results of balloon assist. They are the increase in stroke volume of the left ventricle and the decrease in the workload of the left ventricle. Both of these results are ultimately linked to optimal timing of the balloon’s deflation. In timing the balloon, one wants to inflate the balloon at the moment of aortic valve closure, keep the balloon inflated during diastole and deflate the balloon just prior to systole. Essentially what occurs with optimal balloon timing of deflation is a decrease in the pressure in the aorta during systole. This is due to the decrease in the intra-aortic volume. As a result, the left ventricle does not have to push against as high a resistance with the ejection of its stroke volume during systole. Thus the workload is decreased on the left ventricle.
From the equation, pressure equals flow times resistance; we derive the equation, flow is the result of pressure divided by the resistance. Therefore, since the mean arterial pressure is increased and at the same time the resistance in the aorta is decreased then the flow or stroke volume is increased. Coincidentally you will recall that the cardiac output is the result of heart rate times the stroke volume and, assuming the patient’s heart rate stays the same, we can make the assumption that the cardiac output increases due to the increase in stroke volume.

Earlier in this paper it was stated that the balloon is used in patients with mitral regurgitation and post infarction ventricular septal rupture. The balloon is utilized in these patients since it is felt that the amount of shunting or mitral regurgitation is decreased. By decreasing the resistance in the aorta which is created by the balloon deflation, blood selectively flows into the aorta instead of through the vsd or mitral valve. In conclusion, it can be summarized that the net results of the balloons effects are: first, increased coronary blood flow; second, increased mean arterial pressure; third, increased stroke volume and cardiac output; and fourth decreased workload on the left ventricle which have all resulted from the balloons counterpulsation. Therefore, the heart is allowed to work less or rest while at the same time it can receive an abundance of blood flow through to the coronary arteries during diastole, all of which theoretically should allow the heart to recover.

DISCUSSION

Q: What’s azotemia? And what are the contraindications for using intra aortic balloon pumping?

A: Azotemia is one of the secondary signs for organ failure. And that’s brought about through the lack of good perfusion of the blood through the kidneys which allows the kidneys not to function optimally. This allows the build up of the products of urea and urea bound nitrogen in the body as evidenced by BUN, also all of these patients will show an increase in creatinine.

Contraindications of balloon pumping: I did not go into contraindications of balloon pumping. However, there are a few that are noted, the most glaring one being frank aortic regurgitation. As you can all understand, knowing the disease process, that as a result of aortic regurgitation intra aortic balloon counterpulsation would only aggravate this condition. And another contraindication to balloon pumping is the fact that you can’t get the balloon inserted as I described. The majority of the patients have the balloon put in through the femoral artery, but in some cases where the patient shows marked arteriosclerotic disease as evidenced through the fact that you can’t insert it into the femoral artery, some people put it in from above.

Q: The Datscope balloon is smaller?

A: Correct

Q: Do you use a one directional assist balloon from the top?

A: Theoretically, that’s not right. However, I have asked this question of the Datascope people and they say that if the balloon is put in from above, the occlusive property of that second small balloon in that position will not function. So, in essence, if put in from above the Datascope balloon becomes a bi-directional balloon.
Q: How do they explain that to you? We have tried that out and that is not what we have found at all.

A: We haven’t tried it from above and we don’t do it from above with the dual chamber balloon. If you remember the schematic in the aorta, they feel that the aorta necessarily increases in size going from the base of the heart through the body. They feel that if the balloon was put in from above then the size of the aorta at that point is larger than volume displaced by that secondary balloon. That is their theory. If your practice shows it can’t work, your clinical situation may show a great deal of divergence of what has been presented in this paper as basic facts.

Q: Is it really that advantageous to use a balloon pump considering the medical regimen one can now use?

A: I in no way want to leave you with impression that at my institution we have the grand and glorious results that initially were published. We do not. But, by practice, we found that through intra balloon counterpulsation some patients are discharged from the hospital and are able to go home to lead relatively good lives. The majority of our patients do not. Just because of the fact that it ideally allows their hearts to recover, even if you intervene surgically, which is not often possible since their hearts just aren’t good enough to recover from the damage that has been done. I think a lot of this is due to the fact that the medical practitioners do not intervene in a patient who they feel they can get along with medical management. They wait, maybe they don’t wait for the patient to be blue, but they wait just one step more before they decide to use the aortic balloon. And so I think that is part of the reason for the high mortality or low success rates.

Q: Why don’t we get the results (better success rate) even though we see what we are supposed to see?

A: That’s a question which can only be answered through a lot of post mortem research, maybe there was something biochemically going on with the heart. The balloon just can’t change the biochemical structure. I don’t know I just can’t answer that question.