The evolution of counterpulsation techniques

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Birtwell, W. C., R. H. Clauss, C. Dennis, D. E. Harken, and H. S. Soroff. The evolution of counterpulsation techniques. *Med. Instrum.* 10: 217-223, 1976.—Assisted circulation techniques are an outgrowth of extracorporeal pump oxygenator systems developed in the mid 1950s. But the differences in physiology of total perfusions and of partial perfusions for assisting circulation delayed the clinical application of the intra-aortic balloon. The problems and risks in providing circulation assistance and the approaches used to overcome them are discussed. The various counterpulsation techniques are described and data are presented to demonstrate the increased survival in patients with myocardial infarction and cardiogenic shock.

cardiogenic shock; circulation assist; counterpulsation; in-series assist; left ventricular pressure; myocardial infarction; perfusion; shunt assist

Introduction

IN MID 1950, the successful application of extracorporeal pump oxygenator systems to maintain the circulation of patients during open-heart surgical procedures stimulated the thought that similar techniques might be employed to assist the circulation of patients in circulatory shock. At that time no one would have predicted that more than a decade would pass before assisted circulation techniques would find clinical application. Discounting several preliminary clinical studies undertaken to test various assist modalities, it is only during the past few years that the intra-aortic balloon has found limited clinical use.

What caused the delay? Certainly not lack of effort. This subject attracted many investigators, stimulated the formation of the Artificial Heart Program, and received substantial financial support during the past 20 years. The need for an effective circulatory assist technique is dramatically demonstrated by the hundreds of thousands who have died for lack of a useful solution to the problem. Clearly, the difficulties associated with the development of effective methods for assisting the circulation were far greater than the early success with total perfusions would have suggested. The practical constraints imposed by the conditions under which most circulatory assist procedures must be undertaken are infinitely more difficult than elective cardiopulmonary bypass. The lack of control over the time and place, the emergency nature of most applications, the requirement for rapid response to the crises, and the limitations placed on surgical intervention are some of the problems that have had an important impact on the utility and effectiveness of circulatory assist techniques.

The Physiologic Problem

There is a significant difference between the physiology of total perfusions and that of partial perfusions for assisting circulation. During a total perfusion, the heart and lungs are isolated from the systemic circulation, and the perfusion can be controlled to meet or alter the demands of the systemic circulation. The isolated heart does not "see" the perfusion pressure that is being provided by the pump oxygenator. The risk of fatally overloading a failing or compromised left ventricle does not exist in a properly performed total bypass.

During assisted circulation procedures, the heart and lungs are still an integral part of the system. The systemic circulatory demands in cardiogenic shock cannot be provided without concern for the effects of externally provided, increased arterial pressure on the compromised left ventricle. If the left ventricle cannot eject its contents against the increased back pressure provided by the circulatory assist system, it may dilate and quickly be irreversibly damaged. These circumstances may exist after a long open-heart procedure on an enlarged sick heart when the surgeon attempts to put the patient back on his own heart. If the heart cannot maintain adequate perfusion pressure and flow, the pump oxygenator must again take over part of the load, and, in some cases, total perfusion must be

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reinstituted. Unfortunately, when this condition arises in a shunt or flow assist procedure, the effects on the left ventricle are not so obvious—but just as lethal.

Assisted circulation procedures strive to rest the heart and to increase systemic and coronary artery perfusion. When cardiogenic shock is being treated, both objectives—reduced heart work and increased flow—must be achieved. The flow and pressure in the systemic circulation must be maintained at a level compatible with survival of the organism at a tolerable energy cost to the heart.

Two basic approaches to assisting the circulation have been studied: the shunt or flow assist, and the in-series or pressure assist often referred to as counterpulsation. The veno-arterial assist (left atrium to femoral bypass) and the apico-aortic version of the artificial heart are examples of shunt or flow assists. In each, pumps are used to bypass the heart or ventricle with the intention of reducing the amount of blood that must be pumped by the heart.

The schematic diagram in Figure 1 illustrates shunt and series assist techniques as applied to the artificial heart. On the left, the artificial ventricle is installed to provide left atrium/aorta shunt assist. This principle cuts ventricular oxygen consumption nearly in half without lowering the mean aortic tension-time index pressure (9), or pressure time product per minute. This reduction in work is accomplished by shortening of the myocardial end-diastolic fiber length, i.e., by the law of LaPlace (6,11). On the right, the single-valved ventricle is providing an in-series pressure assist. The series assist effects a suction phase when the left ventricle ejects, providing a low pressure sink to accept the stroke volume of the ventricle. The distal valve in this prosthesis prevents blood from regurgitating from the distal aorta during the suction phase of the pump and permits the ultimate in counterpulsation pressure effects.

These assist versions of the artificial heart, designed to provide long-term maximally effective support to the critically damaged left ventricle, represent one end of the broad circulatory assist spectrum. The assist techniques which are discussed in this communication are designed for short-term assistance to a temporarily malfunctioning heart. The assist ventricles may achieve maximum potential hemodynamic benefits when operative trauma does not coexist.

The response of the complex regulating mechanisms of the circulatory system influences the hemodynamic effects produced by any circulatory assist procedure. The status of this regulating system depends on the patient's condition—his metabolic status and other flow-related physiologic parameters. For optimal effectiveness, the circulatory assist procedure should introduce tendencies that evoke appropriate responses from the heart, the regulating mechanisms of the circulatory system, and other perfusion-related physiologic functions.

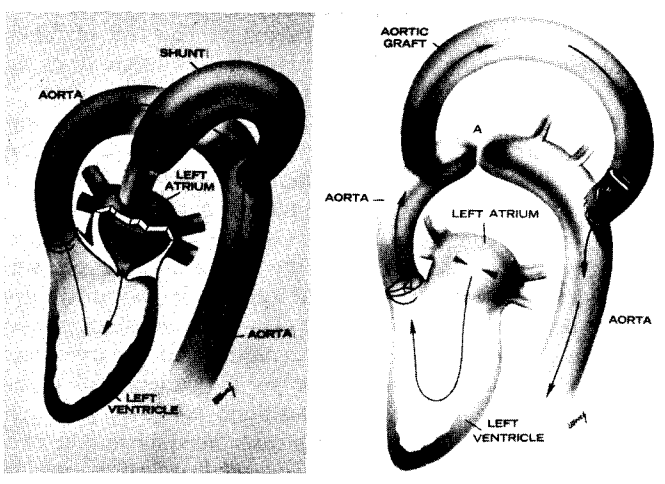

Fig. 1. Schematic diagram of shunt and series assist techniques applied to the artificial heart.

This communication is concerned only with the in-series pressure (counterpulsation) approach. No pumping action (unidirectional flow) is involved in this assist modality. It has been possible to evolve from conventional counterpulsation, which required the alternate withdrawal and return of blood to the arterial system, to the intra-aortic balloon, which achieves the same pressure response by displacing blood by means of a pulse of gas in a balloon. The same effects have been obtained with no invasion of the vascular bed by pulsating pressure externally on a portion of the body. The resulting volume change in the pressurized portion of the arterial system provides the counterpulsated pressure waveform in the aorta.

Although we have documented the optimum counterpulsation that can be achieved with the in-series pumping ventricle, our principal effort has been directed to the development of a counterpulsation system so simple, practical, and safe that it can be applied to the early treatment of the millions who are at an early phase of coronary insufficiency. When a coronary occlusion has occurred, time is a major determinant of outcome. Early treatment has the potential of minimizing irreversible damage to the ischemic myocardium, but requires simple and safe methodology.

Counterpulsation was based on the concept of a favorable response of the left ventricle to reduced arterial pressure during the systolic ejection period. Sarnoff showed good correlation between 0_2 consumption of the left ventricle and the tension-time index (TTI) which he calculated as the area under the left ventricular pressure curve, times the pulse rate (19). Later Levine more properly called this parameter pressure time per minute (PTM) (20). Dennis called it pressure time product (PTP) per minute. Both again demonstrated the relationship between pressure time and LVO_2 consumption. Clearly TTI and PTM are related through LaPlace's Law to the dimensions of the left ventricle.

The heart can be rested and its demand for oxygen reduced if the left ventricular pressure can be reduced. The requirement for increased flow to satisfy the metabolic needs of the organism dictates that the effective perfusion pressure must be increased. Diastolic pressure must be increased to offset the decreased systolic pressure. This requires a system that can be synchronized and phased with cardiac activity, a method called counterpulsation. Diastolic augmentation, post-systolic augmentation, and phase shift pumping are other names that have been used to describe synchronous pressure assist. No matter how the volume and pressure in the aorta are synchronously and phasically varied, the response of the cardiovascular system determines the hemodynamic results.

Studies of the hemodynamic effects of counterpulsation have revealed several components that recommend this modality of circulatory assist for the treatment of heart attacks and angina:

(*a*) The stroke volume per unit work of the left ventricle is increased. Either the left ventricular pressure and PTM are reduced or the cardiac output is increased, or both.

(*b*) The diastolic perfusion pressure and the ratio of the mean diastolic pressure to the mean systolic pressure are increased.

(*c*) The coronary flow increases preferentially with the diastolic pressure since coronary vascular resistance is minimal during cardiac diastole.

(*d*) The coronary collateral flow to ischemic regions of the myocardium is increased.

(*e*) The modification of the pulse pressure distribution in the aorta favors the increase of flow to the vital organs.

The dependence of the hemodynamic response to counterpulsation on the status of the regulating mechanisms of the cardiovascular system has produced some apparent inconsistencies in the literature. Clearly, if a study is performed to measure the effects of counterpulsation on the coronary flow, the metabolic status of the myocardium is a controlling factor. The coronary vascular resistance is not fixed. Therefore, unless an oxygen demand exists, the tendency for the coronary flow to increase with increased diastolic pressure will be offset in part by an increase in coronary vascular resistance. This effect is dramatically demonstrated in Figure 2. In the experiment, an in-series pumping ventricle provides a maximal counterpulsation effect. Note that when counterpulsation is instituted there is an immediate increase in cardiac output and coronary flow. During the next 30 sec, the regulatory mechanisms respond, and the cardiac output and coronary flow are returned to values that meet the flow requirements. The steady-state coronary flow actually is reduced to a level lower than control, reflecting the reduced oxygen demand of the ventricle at the very low left ventricular pressure.

Large pressure changes can be produced when the damping effect of the aorta is isolated by a distal valve. Ventricular prostheses are capable of reducing left ventricular pressure to atrial levels. Conventional counterpulsation cannot provide such profound pressure effects. Laboratory and clinical results have proved that modest pressure effects provided quickly can be effective in reversing circulatory failure and protecting ischemic myocardium.

Counterpulsation techniques have evolved in two important ways: The synchronous electronic control devices have been improved to provide more stable and accurate triggering of the counterpulsating actuator, and less traumatic and more practical methods for providing the displacement of blood to create the pressure changes have been developed. Both were essential to the successful clinical application of counterpulsation. The clinical environment demands simplicity, practicality, effectiveness and safety.

The Synchronous Electronic Control

Two physiologic signals have been utilized to synchronize counterpulsation systems: the ECG and the aortic pulse pressure. The R-wave of the ECG is by far the most common choice. Two characteristics have militated against the use of aortic pressure: (*a*) The pressure pulse has no sharp component to provide accurate triggering, and (*b*) counterpulsation introduces a diastolic pulse that must be gated out.

In spite of the above difficulties, there are good reasons for the use of the pressure pulse as a trigger signal. Although the normal ECG has a QRS complex that is ideal for triggering, the pathologic ECG of the typical patient is very often modified by myocardial ischemia. This signal is "noisier" in that the R-wave is more difficult to identify. Elevated T-waves and depressed R-waves with broader QRS complexes can make consistent identification of the R-wave difficult. Signal-

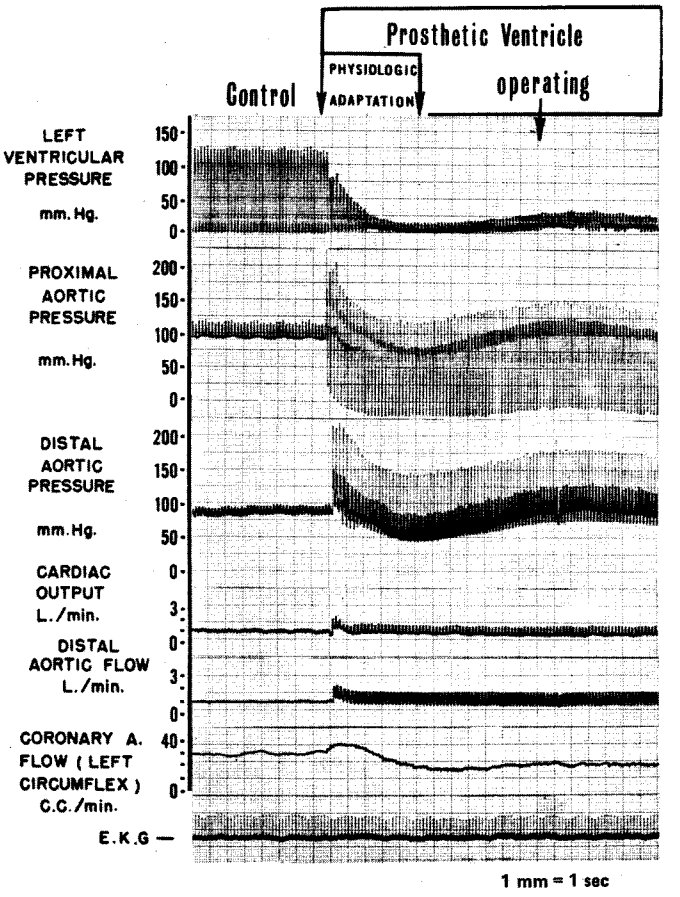

Fig. 2. Concurrent pressure, flow, and ECG recordings during counterpulsation.

conditioning systems that have been employed to permit more consistent R-wave triggering are polarity and lead selection, clamping, gating, and differentiation. These circuits can improve the signal-to-noise ratio and provide stable triggering under adverse conditions.

The lead and polarity selection capability permits the operator to select the lead with the least interference from elevated T-waves. The polarity selector makes it possible to trigger on either a positive or negative going signal. Clamping makes it possible to bias out unwanted portions of the signal.

Gating permits the electronic circuitry to be shut off for an adjustable period of time after the trigger signal. The typical gate is a monostable multivibrator circuit, triggered by the selected signal, that remains in that mode for the desired period of time. If the gate is adjusted to be closed for a period slightly less than the heart period, all ECG components except the R-wave are obliterated. If the gate period is increased to a value greater than the time between R-waves, the counterpulsation system will trigger on every other beat. This mode of operation is sometimes useful, particularly if the counterpulsation system cannot respond at the patient's heart rate.

When arrhythmias are present, some judgment must be exercised. If the gate is adjusted to the most stable condition so that it opens just before a normal R-wave, premature beats will be skipped. The gate period must be shortened if one wishes to trigger on premature beats. Again, if the gate is effective, it is possible with premature beats to shift from R-waves to T-waves, causing the gate to provide stable but incorrectly

phased operation. This condition must be avoided since improperly phased counterpulsation can be traumatic to the aortic valve and the left ventricle.

Differentiation permits fast-rising signals such as the R-wave to be identified from slower-rising T-waves of equal or greater amplitude. The differentiated QRS complex provides a signal that is a function of the rate of change or slope of the R-wave. Although differentiation is effective in eliminating T-wave triggering, extraneous high-frequency noise signals will still be troublesome. Careful design can provide stable function, but intelligent operation is still required.

Many synchronous electronic controls incorporating various combinations of the signal-conditioning circuits outlined above have been developed. Typically the output trigger signal from such circuitry is utilized to energize a monostable multivibrator, which provides a delay usually adjustable from about 50 to 500 msec. The differentiated output of the delay circuit provides the signal that triggers the duration circuit—another monostable multivibrator. This circuit provides the signal that controls the counterpulsation actuator—usually a hydraulic or pneumatic system. The duration signal suitably amplified usually controls a solenoid or servo valve that produces the positive pressure pulse.

Systems have also been devised that utilize the R-wave to trigger the suction or withdrawal phase of the actuator. Since the R-wave precedes the systolic pulse in the aorta by 20 to 80 msec, actuators can be designed to have a fast enough response to permit the withdrawal phase to be accomplished during cardiac systole. This mode of operation requires that the volume change in the aorta be made as close as possible to the aortic root. Propagation delays in the aorta militate against this mode when the volume pulse must traverse the aorta from the femoral arteries. The antegrade pulse delay to the femoral artery ranges from about 50 to 100 msec; the retrograde pulse requires a similar time to travel from the femoral arteries to the root. Clearly the withdrawal phase at the femoral level must anticipate ventricular systole sufficiently to allow for propagation delays.

Improvements made in the synchronous electronic control circuitry, simplifying the phasing adjustment and stabilizing the function of the systems, have made counterpulsation more practical. Clearly it is within the state of the electronic art to provide automatic phasing and control of the delay and duration periods as a function of heart rate. These developments undoubtedly will be made in the future to permit the earliest possible application of counterpulsation by emergency personnel who are first on the scene.

Counterpulsation Techniques

The development of new techniques for providing counterpulsation has had a major impact on the acceptance of this circulatory assist modality. The reduction of trauma and hemolysis, intra-aortic balloon substitution of external blood-handling actuators, and, finally, the elimination of operative procedures and invasion of the vascular bed have made counterpulsation a safe, practical, and effective treatment for myocardial infarctions and coronary insufficiency.

Conventional Counterpulsation

The original counterpulsation technique was developed by the Harvard Surgical Research Group under Harken in 1957. This approach utilized a synchronous extracorporeal actuating system which was developed by Birtwell. One or both femoral arteries were cannulated and attached to a valveless pneumatically operated actuator. A volume of blood (10-20 cc in the dog, 30-70 cc in human beings) was alternately withdrawn and returned to the arterial system. This action was phased with cardiac activity so that the response in the root of the aorta was a pressure reduction during systole and elevation during diastole. The results of the first hemodynamic study were presented in 1958 and were published in 1961 (7). During the next few years many investigators utilized this system and others to study the effects of counterpulsation on cardiac output, heart rate, left ventricular pressure work, oxygen consumption, and coronary flow (14). Increased coronary flow, decreased coronary AVO_2 difference, and reduced left-ventricular pressure work were documented. These findings have been supported by an extensive literature generated by a large number of investigators. It is now clear that counterpulsation does reduce the left ventricular oxygen demand and increase the oxygen supply by increasing the coronary flow. Although counterpulsation has no independent effect on cardiac output, the response of the left ventricle to reduced systolic pressure and increased oxygen supply is generally an increase in cardiac output. When the left ventricle is so damaged that it cannot produce an increased stroke volume during counterpulsation, it is possible that drug-induced lowering of vascular resistance may provide increased perfusion.

In 1961, Jacobey published the first study that documented the potential efficacy of counterpulsation as a treatment for coronary occlusions (12). The Agress microsphere coronary occlusion preparation was utilized to provide a diffuse occlusion of coronary vessels in the 200-300 micron range (1). Counterpulsation significantly improved the survival (from about 25% in controls to about 75% in treated animals) of the animals subjected to this coronary occlusion preparation. Postmortem X-ray contrast injection studies of the hearts disclosed development of collateral channels in the treated survivors, which did not exist in surviving control animals. This was the first indication that counterpulsation could quickly enhance the development of coronary collateral circulation, suggesting the possible application of counterpulsation to the treatment of coronary insufficiency and angina. Many laboratory studies confirmed these findings; clinical studies utilizing the intra-aortic balloon and external counterpulsation support the laboratory results.

Two problems plagued conventional counterpulsation: the requirement for cutdowns of one or both femoral arteries, and hemolysis which limited the duration of an assist procedure. Hemolysis was caused by negative pressure, turbulence, and flutter in the femoral arteries at the tips of the cannulae. The velocity head created when blood is withdrawn rapidly from an

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artery exceeds the static pressure in the artery, reducing the total pressure so that the artery collapses. This causes a fluttering action, which restricts the stroke volume that can be withdrawn, and induces hemolysis.

The Intra-Aortic Balloon

In 1958, Birtwell and Clauss produced counterpulsation by introducing a catheter which supported a long slender balloon into the descending aorta via the femoral artery. Saline was pumped in and out of this balloon by means of the counterpulsating actuator. This work was continued and presented in 1962 (8). In the same year, Moulopoulos published results of his experience with a similar device using gas as the inflation medium (15).

The Artificial Heart Program Office later contracted an extensive development program, which resulted in systems that are now finding clinical acceptance. The principle remains the same. Gas, usually helium or CO_2, is pumped in and out of a long slender balloon supported in the descending aorta by a catheter introduced via the femoral artery. The gas increases the diastolic pressure and displaces blood from the aorta. The removal of the gas reduces the aortic pressure during cardiac systole. The stroke volumes are of the same order as those in conventional counterpulsation, and the hemodynamic effects are similar.

Two advantages are provided by the intra-aortic balloon: The effects are created close to the ascending aorta, and the hemolysis associated with conventional counterpulsation is almost eliminated. Even though a surgical procedure is still required, the intra-aortic balloon is used following cardiopulmonary bypass and open-heart surgery to treat preinfarction angina and cardiogenic shock (10,13,18), and to support the circulation of patients undergoing coronary bypass surgery. The circulation of patients with critically damaged hearts has sometimes been supported for weeks.

External Counterpulsation

In 1963, Dennis, Osborn, and Birtwell independently reported successes in achieving counterpulsation without invading the vascular bed by applying pressure pulsations to regions of the body synchronous with cardiac activity (4,5,10,16). Positive pressure applied to the lower portion of the body compresses the vascular bed and displaces both arterial and venous blood. The venous blood is moved antegrade into the central venous pool. Release of pressure encourages arterial blood flow retrograde into the aorta. Thus the limbs become the actuator providing a potential arterial stroke volume of 100-200 ml, which permits effective counterpulsation pressure effects.

Studies of the effects of external counterpulsation in the venous system (Fig. 3) disclose that the valves in the major veins prevent regurgitant flow and provide a venous pumping effect. Further, the blood volume of the venous bed of the limbs is quickly displaced into the central venous pool providing the effects of transfusion. As a result, there is no large venous flow pulse. These effects in the venous system cause external counterpulsation to provide greater increases of cardiac output than other counterpulsation methods.

An external counterpulsation system (Synchronous Cardiac Pulsator 400-1, Flow Corporation, Watertown, Mass.) was produced for clinical studies (Fig. 4). A rigid conical housing encloses the legs of the patient. Water-filled seals fill the dead space between the patient's limbs and the rigid housings. The hydraulic actuator displaces about 2 liters of water into the seals

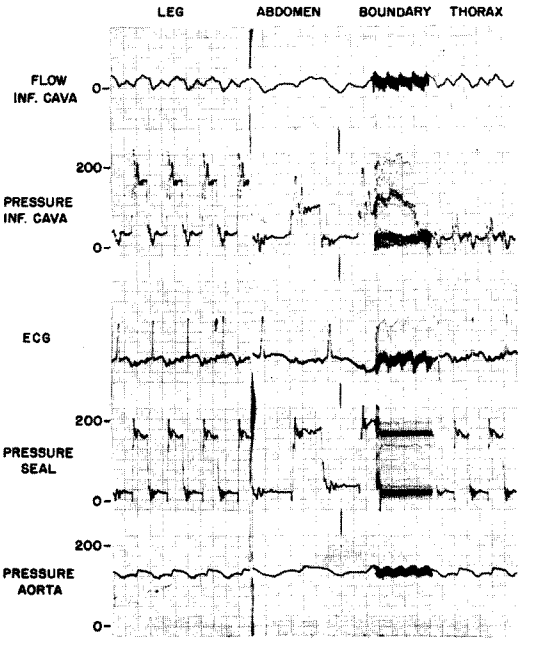
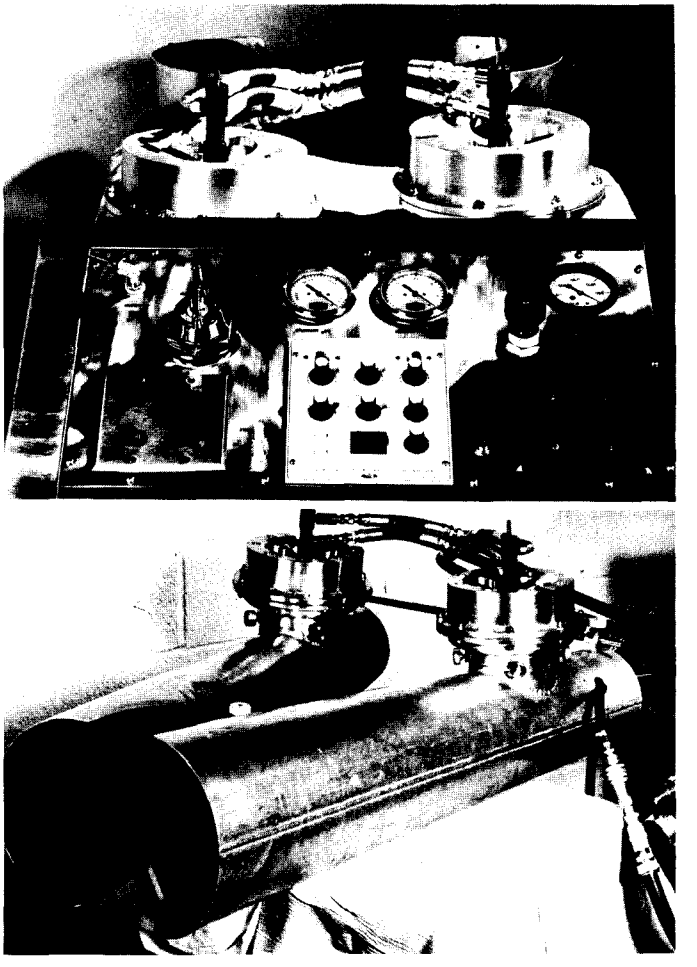


Fig. 3. Effects on the pressure of the inferior vena cava by external counterpulsation.

Fig. 4. External counterpulsation system. Top: Console housing the hydraulic pump. Bottom: Rigid conical housing encloses the legs of the patient.



to provide external pressure of approximately 200 mmHg. The driving power for the hydraulic actuator is provided by a 1-HP high-pressure hydraulic pump housed in the console. A synchronous electronic control operates a servo valve that energizes the hydraulic actuator.

A schematic diagram of the hydraulic power supply for this system is shown in Figure 5. The large stroke volume required for effective external counterpulsation must be displaced rapidly (80-120 msec). Accumulators in the output of the hydraulic pump provide high instantaneous flow rates. The pump output must be adequate to maintain the pressure in the accumulators. The high-pressure low-volume pulse from the hydraulic power supply is converted in the patient pressure actuator to a low-pressure high-volume pulse.

Patient motion has been the most troublesome problem encountered in the development of external counterpulsation. When the limbs are totally enclosed at the foot end in the pressure system, an ejection force equal to the cross-sectional area of the thighs times the external pressure is produced. The limbs act as pistons and tend to be accelerated out of the system. This motion causes a damping effect and accounts for much of the stroke volume requirement. It is also obviously undesirable to move the patient back and forth even if it were not uncomfortable.

The most effective method for restricting patient motion is longitudinal tethering of the walls of the water-filled seals pressurizing the limbs. These tethers restrict the displacement that can take place between the inner and outer walls of the seal. The outer wall is in frictional contact with the rigid housing. The inner wall is in contact with the body. The motion between the body and the system is thus restricted.

It has been demonstrated that the frequency response of ECP systems is fast enough to permit synchronous operation at heart rates up to 200 beats per minute. The damping associated with body motion and tissue displacement requires stroke volume of about 2 liters to produce an external pressure pulse of 200-mmHg peak. This load utilizes the full output of the 1-HP hydraulic pump. This is as large an electrical load as should be placed on the standard electrical system in most hospital rooms. Most applications of counterpulsation require that the system be taken to the patient, so portability and a reasonable power requirement are essential features. It is also necessary that all ancillary devices needed for the procedure be incorporated in the console.

New systems now being tested have been designed to reduce the power requirement and increase the hemodynamic effectiveness of external counterpulsation. Primary emphasis has been placed on determining the optimum external pressure waveform and ambient pressure. A complex mathematical model of the cardiovascular system has enabled a computer analysis of the interaction between the external pressure actuator and the vascular bed. A mechanism has been developed to permit retrieval of most of the energy stored during the pressure cycle, reducing the power requirement of the system.

Preliminary studies have indicated that the arterial blood volume in the limbs can be increased by applying sub-atmospheric pressure during the suction phase of the pressure cycle. This increases the potential stroke volume and improves the hemodynamic effects of external counterpulsation. A new, lighter, form-fitting pressure actuator has been produced that simplifies the creation of negative pressure and reduces body motion and the associated damping effect.

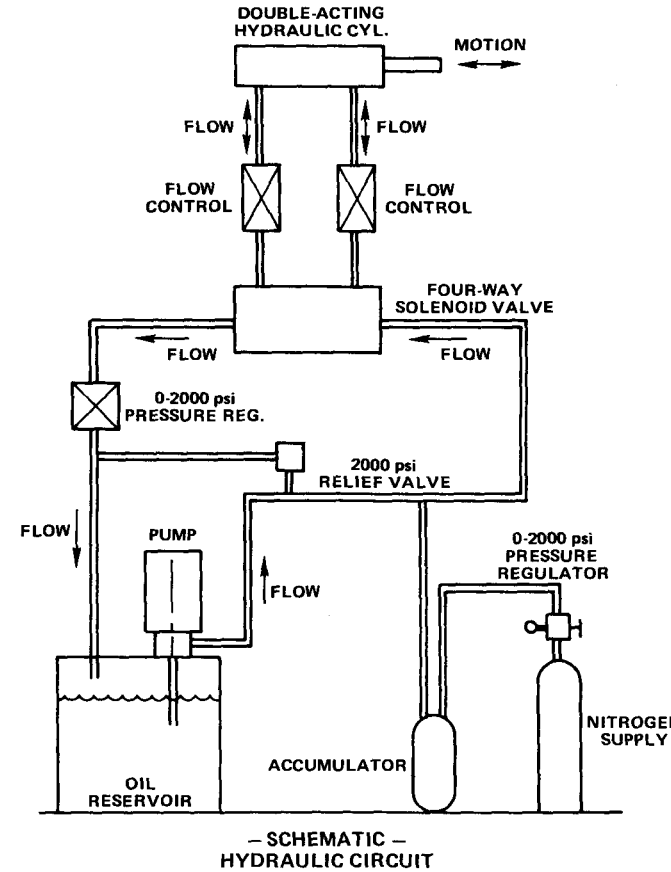


Fig. 5. Schematic diagram of the hydraulic power supply for the external counterpulsation system.

Clinical application of external counterpulsation has been demonstrated to be an effective means of increasing patient survival.

A multi-hospital study of the treatment of patients in cardiogenic shock related to myocardial infarctions was reported by Soroff et al. (21). Judged by accepted clinical signs, all patients had been in the shock state for several hours before treatment with external counterpulsation. The survival rate in 25 patients was improved from 15 percent to 48 percent.

Banas et al. reported effective, long-term results in the treatment of patients with angina pectoris using five daily treatments with external counterpulsation (3). The patients entered the study with Class 3 and 4 angina, which was improved to Class 1 and 2. Pre-treatment and post-treatment coronary angiograms were compared to assess the development of collateral circulation. Many of the patients demonstrated identifiable collateral channels that were not apparent before the treatment.

A multi-hospital study to determine the effects of treatment with external counterpulsation during the first 24 hours after admission to the hospital for myocardial infarction has recently been reported. Mortality was reduced in the treated group by more than 50 percent (2).

These early clinical results support the findings of extensive laboratory studies and justify continued clinical evaluation. The external counterpulsation systems require further development, the treatment protocols need improvement (17), and more clinical data must be collected and evaluated.

Summary

A sustained research effort has proved the efficacy of the basic concept of counterpulsation. Time has been recognized as a most significant determinant of the success of treatment to prevent irreversible ischemic damage to the myocardium during periods of coronary occlusion. The principal goal of this research effort has been the development of the safest and most practical method for producing effective counterpulsation. External counterpulsation has been developed to the point that successful clinical application has been achieved. Although the present techniques are somewhat primitive, external counterpulsation increased survival in patients with myocardial infarction and cardiogenic shock and relieved angina pectoris.

EDITOR'S NOTE: MEDICAL INSTRUMENTATION policy "not to name names" was established to prevent the implication of product endorsement. Not to confuse commendation with endorsement, we must recognize our dependence on responsible, ethical support from industry. The contributions of some companies, both in research and development and in providing equipment, merit their mention by name: Avco-Everett Research Laboratories, Inc., Everett, Mass.; Cardi-Assist Corporation, Fox River Grove, Ill.; Datascope Corporation, Paramus, N.J.; and Hemodyne, Incorporated, North Scituate, R.I. *D.E.H.*

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Next to knowing the truth itself, is to know the direction in which it lies.
And this is the peculiar praise of a sound conjecture.

Diseases of the Heart, Lect. XX