Review Article

Circulatory Support for the Patient in Cardiac Arrest: Some Research Perspectives

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ABSTRACT

Cardiopulmonary resuscitation (CPR), as a treatment for the patient in cardiac arrest, has been met with varying degrees of success. Two of the problems which have been identified include variations in the application of the technique and the initiation of the treatment with respect to the time of arrest. Modifications to the technique, such as active compression-decompression CPR, interposed abdominal compression CPR, and open chest CPR, have not significantly improved survival. It is clear that the major problem is the technique’s inability to provide the necessary myocardial and cerebral blood flow required for recovery. It would seem that extracorporeal perfusion as a resuscitative technique should solve the problems associated with CPR. Extracorporeal circulatory support has met success in some applications but has not been found to be a universal answer for the patient in cardiac arrest. The purpose of this paper is to review the current status of CPR and extracorporeal circulatory support, overview the techniques employed, and summarize applied research that is ongoing in this institution.

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CPR: The Success and the Failure

The success rate of CPR as a treatment for the cardiac arrest patient varies with respect to the definition of “success”. A success rate which refers to the return of spontaneous circulation (ROSC) can be strikingly different from the success rate which refers to recovery and discharge from the hospital. For example, one study has reported that the “success rate” of in-hospital CPR is nearly 45% but, unfortunately, less than 15% of those patients actually leave the hospital (1). A literature review, which defines the “success” of in-hospital CPR as survival to discharge, indicates that rates of 0% to 28% have been reported (2). Another similar review of out-of-hospital application of CPR indicates that discharge from the hospital occurs 2-44% of the time (3). Cummins et al. (4) reported on a comprehensive seven-year study of nearly 1300 patients who had out-of-hospital cardiac arrest. Overall, only 18% of that group survived to hospital discharge.

In addition to the confusion regarding the definition of the success of CPR, it is equally difficult to isolate those factors which contribute to the success of CPR. Varying technique, the aggressiveness and timing of the application of CPR, and the potential use of modifications to standard CPR technique make the analysis of these factors difficult. Data pertaining to survival from out-of-hospital cardiac arrest, for example, is subject to numerous patient-related factors which include age and sex, the cardiac rhythm while in arrest, and any underlying cardiac disease or medical condition the patient may have prior to the arrest (5). While age has not been shown to be a consistent variable with regard to the success of CPR, it does appear that older patients tend to have poorer survival data (2,6). Those patients who have ventricular fibrillation in arrest have a much greater chance of survival than those patients who have asystole as the initial rhythm of arrest (7). Pre-existing medical conditions such as cancer, renal failure, sepsis, cerebral vascular accident, left ventricular dysfunction, irreversible organ failure, or recurrent arrest correlate poorly with survival (2,8). Time and incident factors also play a role in survival. These factors include whether or not the arrest was witnessed and whether or not CPR was immediately initiated by a bystander or an individual specifically trained in cardiac support skills. In the seven-year study by Cummins et al. (4), it was clear that patients who had a witnessed cardiac arrest had a significantly greater chance of survival (26%) than those whose cardiac arrest was not witnessed (4%). In this study, the patients who had a witnessed cardiac arrest were divided into bystander initiated CPR and delayed CPR (initiated by an EMT or paramedic). The data indicated that survival significantly improved if a bystander initiated CPR (27%) over the group whose CPR was delayed (13%).

CPR: Why Doesn’t It Work?

The overwhelming problem with conventional closed chest CPR has always been its inability to adequately produce the blood flow to the brain and heart needed for survival (9-12). This fundamental problem has been noted repeatedly in animal studies which have documented coronary and cerebral blood flows during CPR to be less than 5% and 10% of pre-arrest values respectively (13,14). It is assumed the data from these animal studies are directly applicable to the human patient. Numerous animal and human studies have determined that coronary perfusion pressure (CPP) is the single best predictor of the success of myocardial recovery (8,15,16). It is likely that, during arrest, the coronary vasculature is fully dilated due to the global ischemia that has ensued. Coronary blood flow, therefore, will be directly related to the amount of CPP that can be generated with CPR. These studies have determined that a CPP of at least 15 mmHg is required to have any hope of successful myocardial resuscitation. It has been suggested that, when CPP is maintained at a level approaching 25 mmHg, approximately 80% of the patients in cardiac arrest will likely be resuscitated (17).

Restoration of coronary and cerebral perfusion flow are major determinants of the outcome of CPR (8). The duration of time during which the patient has no flow (from cardiac arrest to initiation of CPR) and the duration of CPR to ROSC are both crucial to the survival of the patient. The “window of effectiveness” (a time reference) to the start of CPR is actually quite small. It has been determined that if CPR is initiated beyond 4-6 minutes after the patient has had a cardiac arrest, it will no longer independently improve survival (4,18-20). Most investigators agree that the single most important factor related to survival is the duration of cardiac arrest. Also, if CPR is continued beyond 10-12 minutes without advanced life support procedures available and implemented, it will not be effective. The limited blood flow produced by CPR and the chance of successful defibrillation both likely decrease as resuscitation time increases (21-23).

Numerous secondary affects of cardiac and circulatory arrest influence the potential recovery of the patient undergoing CPR (24). These include the ischemic and necrotic reactions of blood and tissues throughout the body which set the stage for multiorgan failure. The release of inflammatory and vasoactive mediators during the ischemic period and reperfusion activate complement, neutrophils, lipids, and cytokines. As was previously described, after the initial resuscitation a large portion of cardiac patients die during hospitalization, likely due to multiorgan failure or other complications related to the resuscitation.

The financial cost of CPR should also be considered in a discussion of its ineffectiveness. It is assumed that approximately 90% of the 350,000-400,000 persons who experience out-of-hospital sudden cardiac arrest in this country per year are served by a local emergency medical teams whose directive is
to initiate and maintain a resuscitative effort (25). Since less than 20% of all of these individuals will likely live to be discharged from the hospital, it could be argued that the expense greatly outstrips the benefit. It has also been determined that the cost of the hospital stay rises exponentially as the rate of survival to discharge decreases (26). In other words, non-survivors of the successful use of CPR can consume enormous resources.

CPR: Innovations and Modifications - Non Invasive

The American Heart Association has recommended that standard CPR involve active chest compressions to a depth of 1.5-2.0 inches at a rate of 80-100 times per minute with an active chest compression to passive chest expansion time ratio of 1:1 (27,28). Many of the modifications to standard CPR have come about in an attempt to control some of the variables associated with the technique (e.g. depth and force of compression) or provide feedback information about their effectiveness. Other modifications attempt to directly increase coronary and cerebral blood flow thereby increasing the potential for a successful resuscitation. While some of these modifications have been proven to be beneficial in animal studies and some patient trials, clinical acceptance has been lacking. In reality, in spite of the modifications, successful outcome from CPR has not significantly changed over the last 30 years (6).

Active Compression-Decompression (ACD) CPR

One of the most unusual reports of the use of a mechanical device to assist in the delivery of CPR occurred when a toilet plunger was used to not only produce the compression phase of CPR, but also was used to produce an active decompression phase (29). The standard method of CPR, as originally described by Kouwenhoven in 1960 (30), proposes an active compression phase but passive relaxation of the chest wall. Providing an active decompression phase in the cycle, as is proposed by this newer technique, would theoretically produce a negative intrathoracic pressure which would improve venous return and left ventricular filling. Under some circumstances of resuscitation, it may also increase the ventilation of the patient during CPR.

Research and clinical trials have been undertaken with handheld suction devices which, when applied to the patient’s chest, can be used to apply the compression phase of CPR and provide an active decompression of the chest wall (27,31,32). One such experimental device is the Cardiopump manufactured by Ambu International. Measuring 10-14 cm in diameter, these devices may also have built in monitoring systems to provide feedback regarding both the force and depth of chest compression. These studies have shown significant increases in cardiac output, systolic aortic pressure, coronary perfusion pressure, and minute ventilation when ACD CPR is compared to standard CPR.

Interposed Abdominal Compression CPR

Some of the modifications to standard CPR require sophisticated equipment not readily available to the bystander who witnesses the arrest (33). Such would be the case with the use of chest compression with high airway pressure ventilation or the inflation of a pressure suit (MAST: Military Anti-Shock Trousers) over the legs and abdomen. Interposed abdominal compression (IAC) CPR, on the other hand, is a technique that could be accomplished by anyone who is appropriately trained. The fundamental concept of IAC CPR is counterpulsation and is not unlike the familiar concept of intra-aortic balloon pumping. This technique is performed by two individuals - one performing the chest compressions of standard CPR and the second performing abdominal compressions in a 1:1 ratio (34). The abdominal compressions are performed with the hands centered over the umbilicus and are applied during the early relaxation phase of the chest compression (i.e. during diastole). In theory, as a concept related to counterpulsation, this technique should provide an augmentation of diastolic pressure and improve blood flow to the coronary and cerebral circulation. Abdominal compression may also increase venous return to the right heart thus increasing cardiac output. While no evidence of abdominal trauma in experimental studies has been noted, trial applications involving human patients have excluded patients under 16 years of age, pregnant women, and patients with known abdominal aneurysm, palpable masses, or hepatosplenomegaly (35,36).

Early experimental and clinical results with IAC CPR have shown significant hemodynamic improvement over standard CPR but have not shown significant improvement in resuscitation outcome or survival (34-39). These animal and human studies have shown significant improvement in coronary perfusion pressure, cardiac output, and common carotid blood flow but were unable to find significant differences between CPR and IAC-CPR in the number of defibrillation attempts required, number of animals resuscitated, time to spontaneous perfusion, resuscitation outcome, twenty-four hour survival, or survival to hospital discharge.

Simultaneous Compression-Ventilation CPR

Two mechanisms of blood flow during CPR have been proposed (40-44). Early reports speculated that compression of the heart between the sternum and spinal column (the cardiac pump mechanism) was responsible for forward blood flow. Later reports have argued that the cardiac pump concept cannot totally account for forward flow in many patients and have postulated a thoracic pump mechanism to explain blood flow during CPR. The increase in intrathoracic pressure during chest compression tends to compress the arteries in the chest, moving blood toward the peripheral circulation. Ventilation during standard CPR and modifications such as IAC CPR likely increase intrathoracic pressure and improve blood flow in one of two ways. If intrathoracic pressure is increased during the relaxation phase of the CPR cycle, arterial compression moves blood toward the
periphery. If, however, intrathoracic pressure is only increased during the compression phase of the CPR cycle, the sudden decrease in intrathoracic pressure during the relaxation phase should improve venous return and, subsequently, cardiac output. Simultaneous compression-ventilation (SC-V) CPR serves to ventilate the patient during the compression phase of the CPR cycle, thereby increasing the pressure gradient between intrathoracic and extrathoracic vasculature allowing for forward blood flow. Retrograde flow is prevented by venous valves at the at the thoracic inlet thereby preventing thoracic to extrathoracic flow through the large central veins. Intrathoracic pressure falls during the relaxation phase and improves cardiac output through augmentation of venous return (37,45). Ventilation systems have been designed to administer high pressure ventilation (at airway pressures of 80 mmHg) timed to coincide with chest compression (46). Proposed complications related to this technique include pulmonary barotrauma and an increase in cerebrospinal pressure which may decrease cerebral blood flow.

CPR: Innovations and Modifications - Invasive

Open Chest CPR

Open chest CPR has been shown to significantly increase diastolic blood pressure and cerebral blood flow in animals and humans over that observed with the closed chest technique (13,33,47-52). This technique is generally reserved, however, for those instances where a thoracotomy has already been performed (the operating room) or when the approach is facilitated by penetrating chest injuries. The technique used and the position of the hand or hands in the chest can have significant affects on the success of this form of CPR (53).

Direct Mechanical Ventricular Assistance

The application of direct mechanical ventricular assistance (DMVA) has been shown to further increase perfusion flow and pressure beyond that found with closed and open chest CPR techniques (13,54). First described in 1966 (55), DMVA consists of a cup which is placed around the ventricles which have been accessed via a thoracotomy. Through the pneumatically driven action of a diaphragm in contact with the ventricles, this device actuates cardiac output by alternately compressing and expanding the ventricles to provide systole and diastole. To date only one limited clinical trial of this device has been completed (56). A total of 22 patients were placed on DMVA following failure to respond to conventional CPR. An average time of 81 minutes elapsed between the initial cardiac arrest incident and the start of DMVA. None of these patients survived likely due to the late application of the device.

CPR: Monitoring and Pharmacology

The monitoring of end-tidal carbon dioxide (ETCO₂) partial pressure or percent concentration has been found to be helpful in the assessment of the effectiveness of CPR (8,57,58). When cardiac output is normal or high, ventilation is the rate-limiting factor for CO₂ removal and the ETCO₂ becomes an indicator of arterial pCO₂. When the cardiac output is low, however, pulmonary blood flow is the rate-limiting factor for CO₂ removal and determines ETCO₂. Therefore, under the circumstances of CPR, ETCO₂ is a direct reflection of pulmonary blood flow which is related to cardiac output and coronary blood flow. A decrease in ETCO₂ will signal problems in the delivery of CPR such as malplacement of the endotracheal tube or inadequate chest compressions. An increase in ETCO₂ during CPR is generally associated with an increase in coronary blood flow and myocardial resusciability. A sudden increase in ETCO₂ during CPR often marks the return of spontaneous circulation. As an index of successful outcome, it has been found that an ETCO₂ of greater than 10 mmHg is associated with effective CPR.

Epinephrine, administered intravenously or via the endotracheal tube, has been used as an adjunct to CPR for many years. While the optimal dose of epinephrine is unknown, it has been shown in animal studies that the vascular tone established with 0.1-0.2 mg/kg doses of epinephrine will augment cerebral and coronary blood flow during resuscitation and perhaps improve the chances of early restoration of spontaneous circulation (59).

EXTRACORPOREAL CIRCULATORY SUPPORT (ECS): A SOLUTION TO THE FAILURE OF CPR?

As one reviews the literature pertaining to extracorporeal circulatory support (ECS) for the patient in cardiac arrest, several categories of application are generally found: (1) support of patients who arrest while undergoing high risk angioplasty or valvuloplasty procedures in the catheterization laboratory, (2) support of patients who have arrested due to cold exposure (hypothermia caused by either cold water submersion or cold environmental conditions), (3) support of patients who have arrested in the hospital due to a variety of medical conditions or surgical procedures (outside of the catheterization laboratory), and (4) support of patients who are the victims of either witnessed or unwitnessed cardiac arrest outside of the hospital.

Numerous reports speak to the success and failure of ECS for patients in cardiac arrest who fall into these four categories of application. Since many of these reports combine arrested patients from several or all of these categories, it is difficult to draw conclusions with regard to the success of ECS in this patient population as a whole. Perhaps the most straightforward data to follow are those which report the success or failure of supporting patients who arrest in the catheterization laboratory. Circulatory support for those patients typically takes place as an elective procedure for the patient who has a high risk for acute vessel closure and subsequent arrest or an emergency procedure implemented following a cardiac arrest. Acute vessel closure has been reported to occur in approximately 5% of the patients.

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Raithel et al. reported on a group of 29 ECS patients who had a high risk for abrupt coronary closure during PTCA. Notably, all 6 survivors in this study came from that 14 patient group. Similarly, Mooney et al. reported on a group of 11 patients, 5 of whom had a cardiac arrest following a complication in the catheterization laboratory (67). All five of these patients had a long term survival, whereas only two of the patients who had non-catheterization related cardiac arrest survived. Whittaker et al. reported the use of ECS in 14 patients undergoing high risk angioplasty, 8 having elective support and 6 having emergency support (68). Four of the 14 died, all in the emergency support group. It was concluded that the outcome from ECS in these patients is related to the time delay in deciding to initiate the support and the effectiveness of resuscitation efforts prior to the implementation of ECS. It was also concluded from these studies that the application of ECS is more successful in the elective PTCA patient group than other patients who experience cardiac arrest.

Little data exists summarizing the use of ECS outside of the catheterization laboratory. Phillips et al. detailed several case reports from a 15 patient study which employed ECS in patients who did not respond to standard CPR (69). These cases presented with a variety of etiologies for cardiac arrest and profound shock including hypothermia, trauma, and myocardial infarction. Four of the 15 became long term survivors of the technique. In a follow-up report, Phillips reported that the patient population had expanded to a total of 22 patients with 7 long term survivors (70). Hartz et al. reports the application of ECS in 29 patients who had in-hospital cardiac arrest for a variety of reasons (71). Only one patient in this group survived. Reichman et al. used ECS in 38 in-hospital patients in cardiac arrest who were refractory to conventional CPR attempts (72). While 7 of these patients were failed angioplasties, most presented due to other medical or surgical problems. Ninety-five percent of the 38 patients were returned to a stable cardiac rhythm and 50% were successfully weaned from ECS. Only six (16%) of the 38 patients, however, were eventually discharged from the hospital. Hill et al. (73) have entered data from 17 institutions into a Registry which documents the use of emergent ECS. Of the 187 patients included in this database, 130 had experienced either a witnessed or unwitnessed cardiac arrest. Thirty day survival in this patient group was only 21.4%. The analysis of this data predictably found that patients who had an unwitnessed cardiac arrest or had CPR for more than 30 minutes prior to ECS were poor candidates for the procedure.

The application of ECS for the patient in cardiac arrest has clear advantages over conventional CPR. The technique allows for immediate improvement in tissue perfusion and acid-base imbalance well beyond that possible with conventional or modified CPR (74,75). It also provides for the opportunity to be creative with respect to resuscitation of the heart and brain by allowing alteration of patient perfusion pressure, temperature, blood constitution (hemodilution), and drug delivery (72,76). To be successful in cardiac arrest, the support must be implemented before the duration of ischemia causes irreversible damage to the myocardium and/or other organ systems. One cannot assume that survival is assured merely due to the fact that the patient is being reperfused with a device that can provide a blood flow far in excess of that provided by CPR techniques (77).

Detailed protocols for ECS have been described elsewhere (66,68,78-81). At the outset, it is the opinion of these authors that the set-up and operation of the ECS system must be directed by a perfusionist. The patient preparation for ECS will, of course, be subject to the medical or surgical presentation. Usually, a modified Seldinger technique using a stepped dilation of the femoral vessels is used for cannulae insertion although the exact cannulation protocol will vary with the experience of the physician and the institution (61,66,68,69,80-83). Other large vessels could be used for cannulation (e.g. right internal jugular) depending on the requirements and circumstances of the support. Prior to cannulae insertion, the patient will be anticoagulated with 150-300 U/kg heparin. The femoral and iliac vessels may be studied angiographically prior to cannulation to determine suitability for the advancement of the cannulae, although this is likely only possible for patients undergoing elective PTCA support (68). Thin-walled polyurethane cannulae with an end-hole and multiple side-holes are generally used. The 50-60 cm, 17-30 F, venous cannula is advanced to the level of the cavoatrial junction to provide adequate venous drainage and the 15-30 cm, 12-21 F, arterial cannula is advanced into the iliac artery.

The circuit used nearly always employs preassembled components which include a polypropylene hollow fiber membrane oxygenator and nonocclusive centrifugal pump. Polyvinylchloride tubing of 3/8” internal diameter is generally used to connect these components together. The oxygenator chosen has a blood-outside-fiber blood path design which provides a low resistance to the flow of blood through the system. In contrast to gravity venous drainage into a venous reservoir, which is usually employed with the bypass of heart surgery, these closed and non-compliant systems employ an aspiration of the venous blood by the centrifugal pump. Since this is the case, pump flows of up to 6 liters per minute should be expected in spite of the fact that a relatively small diameter venous cannula is employed. A heater-cooler is used to maintain blood temperature or, in the case of the hypothermic patient, to warm the blood. An oxygen tank (e.g. E-cylinder), gas flow meter, and blender are used in the gas delivery system. The system is built to be portable and battery powered for transport and application in various areas of the hospital. A system as described should be able to be assembled and primed within five minutes (78,83). The circuit is
Patient management while on bypass includes adjustments of patient blood flow, volume, and anticoagulation (81). It is not uncommon for fluid to be administered to support adequate bypass flows. Patient data collected should include arterial blood pressure, right atrial pressure, pulmonary artery and wedge pressures, arterial and venous blood gases, mixed venous oxygen-hemoglobin saturation, electrolytes, and anticoagulation status. System data should include blood flow, gas flow, and oxygen concentration. Heparin is administered to maintain the activated clotting time (ACT) at a value greater than 300-480 sec (67,79).

Many of the reported complications of ECS come out of the literature describing the use of this support for PTCA and in-hospital or hypothermic arrest. Primary complications appear to involve the cannulation procedure (66,79,84). These include bleeding, occlusions, pseudoaneurysms, vessel injury, venous thrombosis, infections, and femoral or peroneal nerve injury. Other complications include embolism, infection, hemolysis, or ischemia.

**ONGOING COLLABORATIVE RESEARCH**

Research at The Ohio State University involving ECS and the cardiac arrest patient has been a collaborative effort between the Division of Circulation Technology and Department of Emergency Medicine and is under the direction of Dr. Mark Angelos. A focus of our investigation has been the use of nuclear magnetic resonance spectroscopy as a tool to evaluate changes in myocardial phosphate dynamics during cardiac arrest, ventricular fibrillation, and reperfusion. Nuclear magnetic resonance spectroscopy involves the transmission of high-frequency radio waves into a tissue placed into a strong magnetic field (85-87). Absorption of this radio frequency by atomic nuclei in the magnetic field will generate signals which then can be used to develop a frequency spectrum. Certain atomic nuclei possess a property known as spin whereby the nuclei spin around their axes like a small bar magnet. When these nuclei are placed in a magnetic field (such as the core magnet used in magnetic resonance), these nuclei will align in a specific orientation. When a short (several microseconds) burst of high-frequency radio waves is added the nuclei resonate producing their own small magnetic fields. The area under the peak signal in the spectrum is directly proportional to the sum of the resonating nuclei or its concentration.

Our investigations have centered on the naturally occurring isotope $^{31}$P. The spectrum produced includes peaks which represent the concentrations of inorganic phosphate (Pi), phosphocreatine (PCr), and adenosine triphosphate (ATP). In addition, it has been determined that the spectral separation between the Pi and Per peaks correlates to intracellular pH measurements. With this information it is possible to study the myocardial high-energy phosphate changes which occur with various circumstances related to cardiac arrest, ventricular fibrillation, and reperfusion with CPR or ECS.

One such issue is the recovery of high-energy phosphates (HEP) following cardiac arrest and the institution of CPR. It has been shown that the ventricular fibrillation which accompanies cardiac arrest rapidly consumes HEP. By using ECS in a model of cardiac arrest, one can simulate the flow likely produced by CPR (or any other reperfusion technique) and study the depletion and recovery of HEP in the myocardium which occurs following cardiac arrest and the initiation of CPR. Tracking the changes in these HEP will give some idea of the oxygen demands and flow requirements involved in arrest and reperfusion.

Angelos et al. (88) designed a study which induced a 10 minute nonperfused ventricular fibrillation in swine followed by 50 minutes of reperfusion with ECS at a flow rate of 30 ml/kg/min. The 10 minute ventricular fibrillation period simulated the duration of time a patient might be in cardiac arrest prior to initiation of CPR. The ECS flow rate of 30 ml/kg/min simulated the optimal CPR flow rate one could hope to achieve with CPR, about 25% of the cardiac output in this experimental model. It was found that, following 10 minutes of ventricular fibrillation, myocardial PCr levels dropped to nearly zero, ATP levels fell to approximately 50% of the baseline value, and intracellular pH became increasingly acidic. Following the institution of reperfusion, the myocardial PCr levels came back to normal and, in fact, exceeded baseline levels. Over 50 minutes of reperfusion, ATP levels increased slightly to approximately 65% of the baseline value and intracellular pH improved somewhat. Using microsphere flow determinations, it was found that myocardial blood flow during the reperfusion period was significantly greater than baseline, likely due to coronary vasodilation, and that both myocardial oxygen delivery and consumption were elevated. The results of this study concluded that, in spite of the myocardial hyperemia and increased oxygen delivery, only a partial recovery of myocardial ATP and acidosis occurred. It could be assumed that the blood flow associated with CPR would fail to meet the energy demands of the fibrillating heart.

In another study, Angelos et al. used a low flow ECS (10 ml/kg/min) after 10 minutes of unsupported ventricular fibrillation to simulate the blood flow that would be consistent with CPR in a swine model of cardiac arrest (89). After 5 minutes at this flow, 0.12 mg/kg of norepinephrine was administered and...
the ECS flow was increased to 50 ml/kg/min to simulate a more aggressive resuscitation with this technique. At this point, defibrillation and weaning off ECS was attempted. Interestingly, all animals were returned to spontaneous circulation with defibrillation and were able to be weaned from support. Measurement of blood flow to various organ systems utilizing microsphere techniques demonstrated that the animal selectively perfused the cerebral, myocardial, and adrenal vascular beds at the expense of the pulmonary and mesenteric circulations during the early ROSC period.

These two studies, as well as others (90,91), demonstrate the value of ECS as a means of simulating the blood flow consistent with CPR, the blood flow that could be achieved with full support, or any other blood flow that might be anticipated with other resuscitative techniques or procedures. Studies of blood flow distribution, oxygen utilization, high energy phosphate consumption, and drug interventions are possible with these research tools.

THE FUTURE

The opinion that ECS is a viable resuscitative technique for the patient in cardiac arrest is not shared by all investigators. We are familiar with the history of intra-aortic balloon pumping and the fact that not until criteria for use and early intervention were established did it become a successful tool for cardiac support. Similarly, early intervention with ECS and defined criteria for its implementation will perhaps result in more obvious success. Early intervention to provide at least some myocardial and cerebral blood flow seems to be a key. The development of portable partial support systems which might be used by specially trained paramedical personnel is not beyond the realm of possibility. Ease of vascular access and the development of miniaturized circuits and hardware would be two areas of consideration to make this possible. Recovery of myocardial function without the associated recovery of neurological functions is a common problem in these patients. Additionally, the global ischemia associated with a period of no blood flow sets the stage for various tissue reactions to occur once reperfusion begins. Research must be done to determine if modifications to the way reperfusion is performed and the recipe of the perfusate will increase the success of ECS. It is clear that the perfusionist has a vital role in the research and development of these devices and techniques for this special patient population.

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