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# Impact of Manual CPR on Increasing Coronary Perfusion Pressure

In sudden cardiac arrest cases, the ability to adequately perfuse the brain and heart during resuscitation is of critical importance. The problem is that manual chest compressions during CPR provide only one third of normal blood supply to the brain.[1] Even more troubling, manual CPR provides only 10% to 20% of normal blood flow to the heart.[1] In fact, the role of CPR to provide flow to the heart muscle may have historically been under-emphasized. While defibrillation is the definitive therapy for ventricular fibrillation (VF), its efficacy is severely limited by response time, and its success is also dependent on circulation.[2] Quite literally, it may be impossible to restart a heart after several minutes without first providing adequate coronary blood flow.[2]

This paper discusses defibrillation and manual CPR, explores the issues relating to coronary blood flow, and describes a new approach to perfusing the heart and brain during cardiac arrest.

## Time Limitations of Defibrillation

When defibrillation is indicated for sudden cardiac arrest caused by VF, time to treatment is critical to its success. In a study of cardiac arrests in casinos that had automatic external defibrillators (AEDs) applied, a 74% survival rate was observed in patients receiving a first shock within 3 minutes of arrest.[3] If the first shock was not delivered within 3 minutes, the survival rate dropped to 49%, suggesting that even in an ideal setting where arrests are witnessed and defibrillators are readily available, time is a critical factor in determining survival.

According to the study:

> *Intervals of no more than 3 minutes from collapse to defibrillation are necessary to achieve the highest survival rates.*[3]

But in the EMS setting, a 3-minute interval from arrest to first shock is generally not possible, and EMS agencies rarely achieve the high survival rates seen in the casino study.

To address this reality, many communities have implemented EMT-D programs and Public Access Defibrillation (PAD) programs. These programs are important and need to be supported, but they are unable to provide early defibrillation to the majority of patients who need it.

Over 70% of cardiac arrests outside the hospital occur at home where no AED is available, and therefore the vast majority of cardiac arrest patients do not receive defibrillation within the crucial first 3 minutes.[4]

Another limitation is that defibrillation is not indicated in at least half of all cardiac arrest patients. That's because more than 50% of patients in cardiac arrest do not exhibit VF when the rescuer arrives.[5]

So, while defibrillation is the definitive treatment for VF, countershock alone does not ensure survival. Recognition of these facts has led to a search for additional strategies to improve myocardial perfusion as a means of increasing survival in cardiac arrest.

## The Important Role Circulation Plays in Defibrillation

A growing body of evidence suggests that beyond a delay of about 3 minutes, reestablishing blood flow before defibrillation may improve the efficacy of electrical countershock. In fact, animal studies have demonstrated an increased survival when, after several minutes of arrest, subjects receive a brief round of CPR before defibrillation.[1]

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When a Seattle EMT-D program did not produce the anticipated improvement in human survival, the protocol was modified to include CPR before defibrillation.

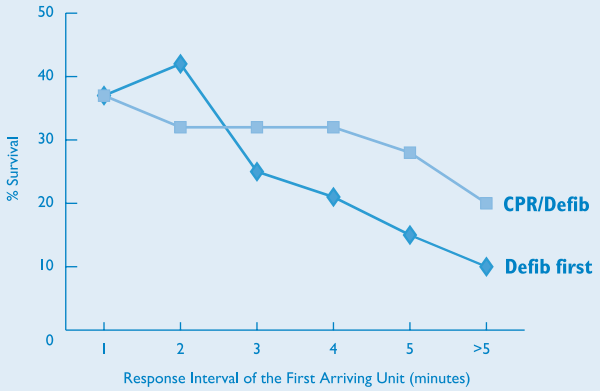


**Figure 1. % Survival According to Response Intervals (minutes) of the First Arriving Unit[2]**

The revised protocol directed the provision of approximately 90 seconds of CPR before defibrillation for patients in cardiac arrest. Under this protocol, for patients whose initial presenting rhythm was VF, the number of neurologically intact survivors increased to 23% when compared with a historical control of 17% neurologically intact survivors ($p=0.01$).[2] This result includes both witnessed and unwitnessed arrests.

The efficacy of administering defibrillation first appears to diminish over time and, after approximately 3 minutes, better results are achieved by delivering CPR prior to defibrillation. The improvement in overall survival was greatest in patients with a response interval of 4 minutes or more after arrest (Figure 1). Survival for these patients as a group increased from 17% to 27% ($p=0.01$).[2]

Though not a prospective, randomized trial, this study of more than 1000 patients does speak to the crucial role of circulation, as provided by rescuer CPR, in increasing survival.

A recently reported prospective study followed a similar protocol where patients were randomly treated with either 3 minutes of CPR first or with immediate countershock. This prospective study showed that for patients treated after a response interval of 5 or more minutes, a significantly higher number had Return of Spontaneous Circulation (ROSC), survival to discharge and one-year survival when CPR was provided before defibrillation. See Table 1. In this patient group, 58% experienced ROSC vs. 38% of the control group, and the one-year survival was 20% vs. 4% for the control group.[6]

Both the Cobb and Wik studies highlight the importance of circulation for increasing survival. The question arises as to how well CPR establishes circulation.

## Myocardial and Cerebral Perfusion in CPR

Even when performed by experts, manual CPR produces only about 30% of blood flow to the brain and a meager 10% to 20% of normal blood flow to the heart.[1] Due to its unique physiological characteristics the heart is more difficult to perfuse.

## The Mechanism of Heart Perfusion

When beating spontaneously, the heart is perfused in the relaxation phase (diastole) that occurs between contractions. As the heart contracts (systole), blood is ejected out of the left ventricle, past the aortic valve, and into the aorta. At this stage, though, the blood doesn't flow freely into the coronary arteries or the myocardium. The reason: The force of the heart's contraction is greater than the force driving blood into the coronary arteries. It is only with the

**Table 1. Survival For Patients With a Response Interval of 5 or more minutes[6]**

| | ROSC | Survival to Discharge | 1-Year Survival |
|---|---|---|---|
| Group "A": 3 minutes of CPR | 37 (58%) | 14 (22%) | 13 (20%) |
| Group "B": Defibrillation First | 21 (38%) | 2 (4%) | 2 (4%) |
| | p<0.03 | p<0.003 | p<0.003 |

return of diastole that blood flows from the aorta into the coronary arteries, perfusing the heart muscle.

During CPR, the relaxation phase of chest compressions is similar to diastole, and maintaining the proper balance between compression and relaxation ensures adequate blood flow to the heart. This is the foundation for the American Heart Association's recommended CPR duty cycle of 50% (the ratio of time spent under compression to time spent in the relaxation phase). [7]

How do we assess the adequacy of blood flow to the heart during CPR? Commonly, the effectiveness of manual CPR is assessed by checking for a pulse generated by chest compression. The presence of a pulse is a positive sign, especially when it can be palpated with each compression. But it does not necessarily mean that blood flow to the heart is adequate. [8]

According to the American Heart Association:

> *No studies have shown the clinical utility of checking pulses during ongoing CPR. …The important pressure for perfusion of the myocardium is coronary perfusion pressure.*[8]

## The Importance of Coronary Perfusion Pressure

Coronary perfusion pressure (CPP) is an indicator of coronary flow. When CPP increases, so does blood flow to the myocardium.[9]

CPP is the difference between the aortic pressure and the right atrial pressure during diastole expressed in millimeters of mercury (mm Hg).

CPP (mm Hg) = AP (mm Hg) – raP (mm Hg)

Aortic pressure (AP) is the driving force behind coronary blood flow, but blood flow to the myocardium is resisted by the pressure in the coronary venous system. Therefore, the driving force for coronary blood flow (aortic pressure) less the pressure resisting flow (right atrial pressure) yields the blood pressure gradient for that vascular bed, and blood flow is related to this pressure gradient.

Measurement of CPP is an invasive research technique and is not routinely available or practical in the CPR setting. However, the desirability of increasing CPP has tremendous clinical significance and is a useful tool in clinical research.

## Increased CPP Correlates with Survival and ROSC

The clearest link between CPP and the likelihood of a return of spontaneous circulation (ROSC) is documented by Paradis et al.[10] CPP was measured in 100 Emergency Department (ED cardiac arrest patients. A definite correlation was noted between peak CPP and ROSC (Figure 2).

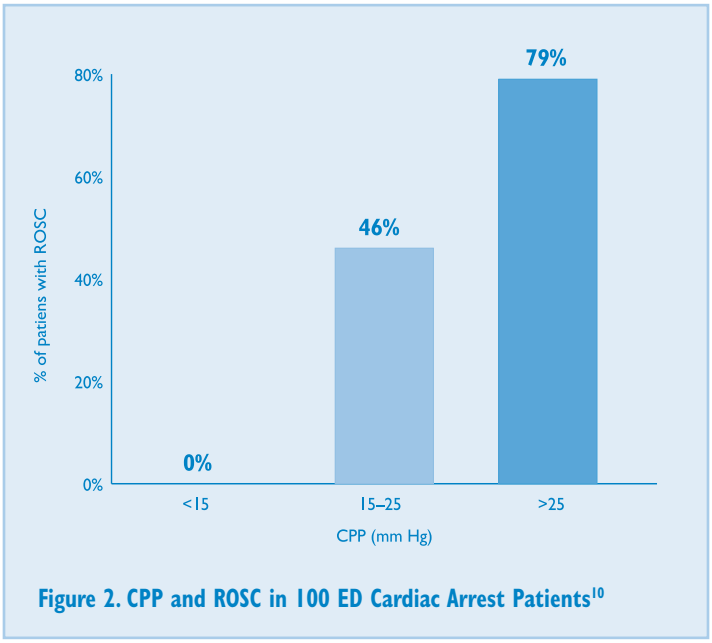


**Figure 2. CPP and ROSC in 100 ED Cardiac Arrest Patients[10]**

Eleven (79%) of the 14 patients with a CPP greater than 25mm Hg had a return of spontaneous circulation, while no patient with a peak CPP of less than 15mm Hg experienced such a return.

According to the study:

> *Return of spontaneous circulation and survival from an arrest have been clearly linked to the ability to achieve a CPP greater than 15mm Hg.*[10]

The problem is the difficulty in achieving and maintaining CPP above 15mm Hg through conventional CPR. In the 100 patients studied by Paradis, conventional CPR provided a mean CPP of only 12.5mm Hg, indicating that conventional CPR cannot reliably provide the CPP necessary for adequate ROSC and survival. As the result of this information, efforts to develop new methods for increasing myocardial perfusion during CPR have been sought.

## Rescuer CPR Performance

As discussed before, CPR typically does not perfuse the heart or brain well under the best of circumstances. In addition, there are inconsistencies inherent in manual CPR.

Several facts emerge from the literature evaluating CPR performance.[11,12]

- Rescuers have difficulty accurately determining the correct depth of compression.
- Rescuer fatigue sets in within 1 minute of CPR, measurably affecting the quality of chest compressions.
- Rescuers cannot accurately perceive their own fatigue.

These shortcomings are not related to individual rescuers, but rather to the complex and demanding nature of manual CPR.

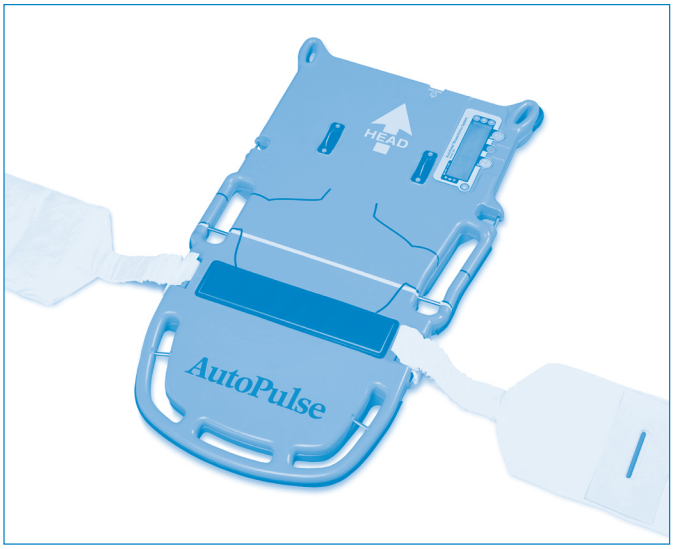


**Figure 3. The AutoPulse™ Non-invasive Cardiac Support Pump**

## Physical Demands of Manual CPR

According to the AHA Guidelines 2000, the chest must be compressed at a rate of 100 compressions per minute, to a depth of 1½ to 2 inches, with proper hand placement, and a 50% duty cycle should be maintained (50% of time under compression, 50% percent under relaxation).[7] This is simply more than most rescuers can physically achieve and certainly more than anyone can perform beyond a few minutes.

In 1998 Ochoa reported on a study of 38 hospital clinicians which found that in the second minute of chest compressions, only 24.9% were done correctly.[11] Furthermore, most rescuers did not perceive any fatigue until after 3 minutes. And 26% of those studied did not perceive any fatigue after 5 minutes, even though a decrease in performance was observed after only 1 minute. Based on this study and a similar study of EMS rescuers by Hightower,[12] it would seem vital to rotate rescuers regularly.

In an attempt to create a useful guideline, another study evaluated teams of two and three rescuers performing compressions, rotating after periods of 1, 2 or 3 minutes. The researchers concluded that to maintain technically correct performance, chest compressions should be performed over periods of 1 minute with at least three rescuers rotating every 1 minute.[13] Unfortunately, this scheme still results in less than optimal CPR, not to mention the impracticality of administering CPR in this way.

## Effects of Pausing Manual CPR

Frequent rotation of rescuers may improve performance, but it introduces a new concern. When compressions are stopped, even for a few seconds, CPP drops significantly and ROSC becomes less likely. [1]

Earlier CPR guidelines called for a ratio of 5 compressions to 1 ventilation, while current AHA guidelines specify a 15:2 ratio. One reason for this change is that CPP is higher after 15 uninterrupted chest compressions than after 5 compressions.[1] Any benefit gained from ventilating every fifth compression is outweighed by the subsequent loss of CPP. Additional evidence shows that pauses in compressions decrease both CPP and the probability of ROSC.[1]

In summary, data reveals that even the most highly trained rescuers can rarely perform manual CPR correctly for more than 1 minute, and even when they can, they are unable to perfuse the heart and brain sufficiently for recovery. For these reasons, manufacturers have attempted to develop mechanical devices that can avoid rescuer fatigue while providing accurate, consistent chest compressions.

## The Ideal CPR Device

In a recent article reviewing the literature on clinical and laboratory use of external and noninvasive mechanical CPR devices, the author states:

> *The goal must be to provide mechanized equipment that is easy to apply and use on the patient as early as possible. The device must also produce a haemodynamic profile better or at least as good as optimally performed manual ECC.*[15]

Several mechanical CPR devices have been developed, but most of these have no known data that show consistently improved CPP over properly performed manual CPR.

Furthermore, most of the devices:[15]

- Have operational limitations due to application time.
- Are cumbersome to install and operate.
- Are heavy and unstable on the chest.

They do not meet the criteria for either improved hemodynamics or ease of use.

## The AutoPulse™ Non-invasive Cardiac Support Pump

The AutoPulse Noninvasive Cardiac Support Pump from ZOLL Medical Corporation is a new device that deploys in seconds to provide automated chest compressions at a consistent rate and depth and standard duty cycle during CPR. In 2001, the FDA cleared the use of the AutoPulse as an adjunct to manual CPR for commercial distribution.

The AutoPulse is a portable, automated chest compression device intended for use as an adjunct to manual cardiopulmonary resuscitation in the adult atraumatic cardiac arrest population. The AutoPulse offers a more efficient method of generating chest compressions during CPR, and results from recent studies provide evidence of the improvement in blood flow when the AutoPulse is used compared to conventional CPR.

The device consists of a single, integrated platform that contains a microprocessor-based control system, an electromechanical drive system, and a user interface panel. A single-patient-use chest compression assembly provides pre-programmed compressions to the patient's chest.

In addition, the AutoPulse has been specially designed to be:

- Rapidly deployable in the field.
- Automatically adjustable to the patient.
- Practical for rescuers of all skill levels.

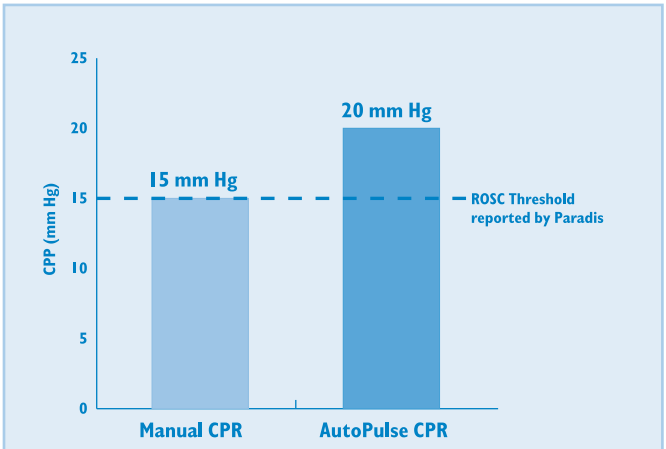


**Figure 4. Human Study CPP Result**[16,10]

## Efficacy of the AutoPulse—Human Study

In a study presented at the NAEMSP 2003 Annual Meeting, the AutoPulse demonstrated a significant increase in CPP compared to the CPP generated from aggressively performed manual CPR. Subsequent to IRB approval, a total of 31 sequential subjects with in-hospital sudden cardiac arrest were screened, and 16 were enrolled. All subjects received prior treatment for cardiac disease and most had systemic co-morbidities. Following a minimum of 10 minutes of failed ACLS and catheter placement, the intubated and ventilated subjects received alternating manual and AutoPulse chest compressions for 90 seconds each.

The AutoPulse demonstrated a significant increase in CPP compared to the CPP generated from aggressively performed manual CPR. Specifically, the AutoPulse was able to produce a mean CPP above the previously described 15mm Hg threshold necessary for return of spontaneous circulation which manual chest compressions did not achieve.[16]

The mean CPP generated with AutoPulse and with manual CPR treatments are shown in Figure 3. For the 16 patients in the study the mean

CPP for AutoPulse was 33% higher than manual CPR (20mm Hg vs. 15mm Hg, $p < 0.05$).

## Efficacy of the AutoPulse—Animal Study

An animal study was also conducted that allowed actual blood flow measurements to be performed in addition to CPP measurements. This study, led by Henry Halperin M.D. at Johns Hopkins University, School of Medicine, was performed to assess hemodynamics with the use of the AutoPulse device as compared to manual CPR. In order to eliminate the effects of fatigue and inconsistencies inherent in manual CPR, the Thumper® system (Michigan Instruments) was used to provide a consistent form of manual CPR.[17]

Ventricular fibrillation (VF) was induced in 10 pigs and after one minute, CPR was performed. AutoPulse CPR (A-CPR) and conventional CPR (C-CPR) were performed in random order.

For the Basic Life Support or "BLS" scenario, no epinephrine was used. For the Advanced Life Support or "ALS" scenario, a 0.5mg bolus of epinephrine was administered followed by 0.004mg/kg/min infusion of epinephrine. CPR was initiated simultaneously with the administration of epinephrine.

Coronary perfusion pressure was measured as well as regional blood flow. Regional flows were measured with neutron-activated microspheres.

The results (Table 2) provided very encouraging evidence of the potential for improved hemodynamics with AutoPulse compared with conventional CPR:

- Without the use of epinephrine (BLS), the AutoPulse was able to produce a mean CPP of 21mm Hg—well above the important 15mm Hg threshold necessary for return of spontaneous circulation as previously described. In comparison, the mean CPP was only 14mm Hg for conventional CPR.
- The AutoPulse produced 36% of normal coronary flow vs. only 13% produced by conventional CPR without the use of epinephrine (BLS).
- When epinephrine was administered to the animals early in the course of the arrest (ALS), the AutoPulse generated blood flow to the heart and brain that were equivalent to pre-arrest levels of flow.

Based on these data, the AutoPulse clearly meets the criterion for demonstrating a hemodynamic profile better or at least as good as optimally performed manual CPR. And its easy operation and rapid deployment in the EMS setting more than satisfies the requirement for practical, efficient use in real-world situations.

**Table 2. Hemodynamic Parameters Measured in a Porcine Model of Ventricular Fibrillation (n = 10)**

| | | Myocardial Flow (% of Pre-Arrest Level)* | Cerebral Flow (% of Pre-Arrest Level)* | CPP (mm Hg)* |
|---|---|---|---|---|
| BLS | AutoPulse | 36%(±12%) | 36%(±10%) | 21(±2) |
| | Conventional CPR* | 13%(±3%) | 28%(±11%) | 14(±2) |
| | | p=0.07 | p<0.6 | p<0.001 |
| | | | | |
| ALS | AutoPulse with epinephrine | 127%(±36%) | 129%(±27%) | 45(±3) |
| | Conventional CPR** with epinephrine | 29%(±11%) | 31%(±6%) | 17(±2) |
| | | p<0.02 | p<0.003 | p<0.001 |

*Mean ±S.E.

**For purposes of the study, the Thumper was used to provide consistent manual CPR to eliminate the effects of fatigue and inconsistency inherent in human CPR.

## Summary

Given the necessity of perfusing the heart well and the difficulty of performing adequate manual CPR, many have sought to develop a mechanical adjunct capable of performing chest compressions. The ideal device, according to experts, must improve hemodynamics and be easy to use.

The portable, easy-to-deploy AutoPulse outperforms manual CPR in generating blood circulation to all organs—including the heart. It has been shown to increase CPP in both animals and humans over and above optimally performed manual CPR. Further, it has been shown in animals to produce levels of blood flow that are greater than pre-arrest levels of flow with the use of epinephrine.

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