Introduction
Cardiopulmonary resuscitation (CPR) is critical to the survival of the sudden cardiac arrest patient. Since its introduction about 50 years ago and continuing to the 2005 American Heart Association (AHA) Guidelines, CPR has been considered standard resuscitation practice.\(^1\) Multiple studies have shown that CPR improves post resuscitation myocardial function, neurological outcomes, and survival. Yet even when performed by experts, manual chest compressions provide only about 1/3 of normal blood supply to the brain, and a meager 10-20% of normal blood flow to the heart.\(^2\) Discharge rates following in-hospital resuscitation are poor, reported by the National Registry of CPR (NRCPR) to be 18% for adults.\(^3\) Even more dismal are the survival rates after out-of-hospital cardiac arrest, hovering around 4-9%.\(^4\)

Presenting Rhythms in Cardiac Arrest

In the out-of-hospital arrest victim, it was originally thought that ventricular fibrillation (VF) was the initial rhythm about 80% of the time. With the realization that early defibrillation was effective for those in VF and with the technologic innovations that have made defibrillation easier, there was a shift in the educational and therapeutic focus to defibrillation as early as the 1992 Guidelines, and chest compressions were relegated to a secondary role. More recently it has been found that only about 40% of victims demonstrate VF.\(^5\) It is likely that a larger number of sudden cardiac arrest victims have VF or rapid ventricular tachycardia (VT) at the time of collapse, but by the time of first rhythm analysis the rhythm has deteriorated to asystole.

NRCPR data from January, 2000 through March, 2004 show that VF and VT were the first documented pulseless rhythms in only 23% of in-hospital adult cardiac arrests. Asystole was the initial rhythm in 35% and pulseless electrical activity (PEA) in 32%.\(^3\)

VF that occurs out-of-hospital is the manifestation of severe, undiagnosed heart disease, primarily coronary artery disease (CAD). The decline in the prevalence of VF as the initial rhythm of cardiac arrest is thought due to primary and secondary prevention, early diagnosis, and successful medical and interventional therapies for CAD.\(^5\) Perhaps patients with CAD are living longer and once they suffer from cardiac arrest, they have reached end stage heart disease where asystole or PEA is more common than VF.\(^6\) The use of beta blockers and calcium channel blockers in out-patients decreases time in VF, so that it is seen only for the first few minutes of arrest.\(^7\) Most arrests in the hospital environment are due to respiratory failure and cardiovascular collapse. Hospital clinicians frequently witness a bradycardia in the unstable patient that progresses to a non-perfusing rhythm.
The treatment for VF/pulseless VT is defibrillation – and it works when administered early. NRCPR data shows that return of spontaneous circulation (ROSC) for ≥20 minutes in adults with VF/VT was 62.0%, and survival to discharge was 36.0%. On the other hand ROSC for adult patients in asystole was 38.4% and for those in PEA 42.9%. Survival to discharge for asystole and PEA was disappointingly small around 11%. Survival isn’t always so grim with PEA and asystole, as Figure 1 shows that survival in children is more than double that of the adults in the NRCPR database.

![Figure 1. Survival to discharge by first documented pulseless rhythm from NRCPR data from Jan, 2000 through Mar, 2004](image_url)

Is survival in children better than for adults because children were more often in an ICU prior to arrest and had earlier recognition of deterioration and earlier interventions? Do children receive respiratory support and drugs sooner? Or could the fact that compressions are performed when a bradycardia appears prior to the loss of a pulse influence the outcome? This new finding leads us to ask: Can there be improvement in the management of PEA and asystole in the adult patient?

**Role of CPR**

So how does CPR fit into all of this? When the presenting rhythms are either PEA or asystole, it is well understood that effective CPR is the first and main treatment that is critical to survival followed by the identification of any reversible cause. For VF the AHA Guidelines 2000 for CPR and Emergency Cardiovascular Care advised that defibrillation be performed as the first
intervention, regardless of the length of time the patient had been down.\textsuperscript{8} In contrast, the AHA 2005 Guidelines now advise that for out-of-hospital cardiac arrest which is not witnessed by EMS compressions may be given first for about two minutes, prior to checking for a pulse and delivering defibrillation.\textsuperscript{1} For in-hospital arrest the provider should still use the defibrillator as soon as it is available.

Why the change to compressions prior to defibrillation when the patient has been down for a time? Let’s look at the electrophysiology of VF. At the beginning of VF there are a few large reentry wave fronts. In VF myocytes don’t stop contracting; rather they are not organized and work independently. During this process the critical energy source of the myocardium, adenosine triphosphate (ATP), which is vital for successful defibrillation, rapidly breaks down into adenosine diphosphate (ADP).

As VF disintegrates, global myocardial ischemia results in myocardial contractile dysfunction, and more reentry wave fronts develop. More myocytes begin to contract and relax independently, causing the waveform to move from a coarse VF (amplitude \( > 0.2 \text{ mV} \)) to a fine VF (amplitude \( \leq 0.2 \text{ mV} \))\textsuperscript{9} and eventually asystole. Chest compressions (especially those of high quality) provide reperfusion and regenerate ATP, which enables the myocytes that are acting independently to ‘line up’ and contract nearly simultaneously. The rhythm then likely moves back into a coarse VF which is more shockable.\textsuperscript{7}
How Do We Measure the Effectiveness of CPR?

The effectiveness of CPR is best reflected by coronary perfusion pressure (CPP). CPP is calculated as the difference between the aortic and right atrial pressures during the relaxation phase of chest compression, which is when the coronary arteries are perfused. See figure 2. The driving pressure for perfusing the myocardium arises from the aortic diastolic pressure, with the simultaneous right atrial pressure acting as an impediment or resisting force to antegrade coronary flow by decreasing the transmyocardial flow gradient.

The link between CPP and the likelihood of ROSC is documented in a landmark study by Paradis et al. CPP was measured in 100 ED cardiac arrest patients. Figure 3 shows a definite correlation between peak CPP achieved during CPR and ROSC. Eleven (79%) of the 14 patients with a CPP greater than 25 mm Hg had ROSC, while no patient with a peak CPP of less than 15 mm Hg experienced such a return. Thus, ROSC and survival from an arrest are clearly linked to the ability to achieve a CPP greater than 15 mm Hg.
There are several other determinants of the perfusion pressure during chest compressions in cardiac arrest – vascular volume, vascular resistance and intrathoracic pressure.¹¹ Adequate CPP cannot be obtained if the volume is low because there is not enough blood to move. Causes of low blood volume include excessive blood loss, vascular fluid extravasation, and marked dilation of the venous system. Epinephrine with its alpha adrenergic agonist activity produces peripheral vasoconstriction resulting in increased central aortic diastolic pressure and thus enhanced CPP.

There are two phases to a chest compression - systole is the compression phase and diastole is the decompression or relaxation phase. A negative intrathoracic pressure during the diastolic phase of chest compression helps aid venous return to the heart. Incomplete decompression increases the frequency and duration of positive intrathoracic pressure, inhibiting venous blood return to the right heart and decreasing the hemodynamic effectiveness of CPR. Excessive ventilations can also increase intrathoracic pressure, further inhibiting venous return. Thus the AHA Guidelines 2005 recommend full chest recoil between each compression and 8-10 breaths/minute.¹

Another factor to consider in maintaining CPP is the reduction of no flow time (NFT). NFT is the down time spent without compressions when assessing for a pulse, performing rescue breathing, intubating, defibrillating, etc. The hands-off time interval when using AEDs according to the Guidelines 2000 has been reported to be as long as 28 seconds.¹² Kern found that with a cycle of 15 compressions, the first 5-10 compressions-relaxations are ‘building up’ the coronary perfusion gradient and that it is not optimal until at least one-third of the series has been completed.¹³ Then, with the cessation of chest compressions, this diastolic gradient falls off rapidly, often returning to near zero within 5-10 seconds. Yu’s research showed the impact of pauses from 3 to 20 seconds on CPP following the initial 3 minutes of CPR in domestic swine placed into VF; see figure 4.¹⁴ Efforts to decrease or limit interruptions are paramount for providing the best perfusion during CPR.

![Figure 4. Relationship between duration of interruption in compressions and coronary perfusion pressure (Yu)](image-url)
Depth of compressions is an important factor in the achievement of higher CPP. Ristagno reported at the 2006 Resuscitation Science Symposium on a study with 10 domestic swine in which electrically induced VF was untreated for 5 minutes, followed by 3 minutes of chest compressions given according to the AHA Guidelines. The pigs were randomized to receive compressions at 6 cm depth (recommended depth) vs 4 cm (70% of the depth) via the Thumper® (a pneumatic piston device manufactured by Michigan Instruments) at a rate of 100/minute. CPP was significantly lower in the animals in which compressions were performed with 70% of the recommended depth; see Figure 5. Each animal in which compressions were performed in accordance with the Guidelines was successfully resuscitated and survived for more than 72 hours. No animal which had compressions after a 30% reduction in depth had ROSC (p=<0.001).

![Figure 5. CPP related to compression depth (Ristagno)](image)

*Guidelines 2005 recommend 100 compressions per minute.* When chest compression rates fall into the 70s and 80s, as we often find during real resuscitations, CPP falls essentially to zero.

**CPR Challenges**

So we are trained health professionals, right? We can do CPR with our eyes closed. Let’s take a look and see. A recent seminal study of in-hospital CPR by Abella yielded some surprising results. It was
found during the first 5 minutes of CPR at the University of Chicago Hospitals that:

- Chest compression rates were too low: less than 90/min 28.1% of the time, and less than 80/min 12.8% of the time.
- Compression depth was too shallow (< 38 mm or 1.5") 37.4% of the time.
- The no flow fraction (NFF) yielded a mean of 0.24 with 40.3% of the segments having a NFF of > 0.20. (Note: a 10-second pulse check every minute of CPR would yield an NFF of 0.17).\(^{17}\)

WHAT? That was my thought as well. In 1998 Ochoa reported on a study of 38 hospital clinicians which found that in the second minute of chest compressions on a Laerdal Skillmeter™ Resusci Anne® manikin, only 24.9% were done correctly.\(^{18}\) Furthermore, most rescuers did not perceive any fatigue until after 3 minutes. 26% of those studied did not perceive any fatigue after 5 minutes, even though a decrease in performance was observed after only one minute. This is the reason AHA *Guidelines* recommend switching providers every two minutes.

At the 2006 Emergency Nurses Association conference in Texas 400 nurses participated in a “CPR Challenge”. These trained professionals performed one or two minutes of compressions on a manikin, using an automated external defibrillator (AED) with CPR feedback. A sensor was placed on the sternum, which constantly measured the rate and depth of their compressions. At the end information was wirelessly transmitted to a computer with code documentation software, which rated their compressions to determine how many were in the “target zone” defined as rate 90-110/minute and depth 2-3”. This exercise occurred in a relaxed, stress free atmosphere. The results are shown below in Figure 6. On average the compressions delivered were out of the target zone 40% of the time.

![Figure 6. ENA 2006 CPR Challenge results](imageURL)
Mechanical CPR with a Load-distributing Band

We know why CPR is important and why the focus is shifting to consistent, high quality, uninterrupted chest compressions. In addition, we now know that we are not performing compressions very well, but how do we break the cycle? We can use a load distributing band (LDB) for delivering mechanical CPR with the AutoPulse®.

The AutoPulse® is a revolutionary LDB resuscitator that moves more blood, more consistently than is possible with human hands. Easy to use and battery operated, its unique LifeBand® squeezes a vast surface area of the chest. AutoPulse minimizes no-flow time by providing consistent, high-quality compressions without causing fatigue. In doing so, it eliminates the interruptions that can cause CPP to fall. As a result, patients receive optimized compressions that deliver improved blood flow and the possibility of better outcomes.

Placing the patient onto the backboard of the AutoPulse is easy and takes only a few seconds. The device automatically calculates the patient’s chest size, shape and resistance, and then applies only that force necessary to compress the chest the prescribed amount for each individual patient. Its simple user interface allows rescuers to apply the device rapidly and then focus on more advanced skills and critical problem solving. The settings can easily be changed from a 30:2 compression to ventilation ratio for Basic Life Support (BLS) providers to continuous compressions for Advanced Cardiac Life Support (ACLS) providers. The AutoPulse is also safe for the healthcare professional, who no longer has to risk personal safety while conducting compressions in the back of a moving ambulance or on a gurney. In addition, it can enhance the effectiveness of defibrillation by “priming the pump” and/or helping to convert a rhythm from non-shockable to shockable. Further, it allows caregivers better access to the patient and is often viewed as helping to “organize” the chaotic code situation.

AutoPulse Clinical Support

Sure sounds good, but does it really work? There is a growing body of clinical evidence that supports the benefit of the AutoPulse. There are two important animal studies on the AutoPulse. The first study, conducted by Halperin at John Hopkins University Medical Center, was designed to evaluate regional blood flow and CPP with the AutoPulse as compared to manual CPR. In order to eliminate the effects of fatigue and inconsistencies inherent in manual CPR, the Thumper device was used as a surrogate due to its hemodynamic equivalence to manual CPR. Ventricular fibrillation was induced in 20 pigs and after one minute CPR was performed using either the AutoPulse or the Thumper in random order for 4 minutes, and then the method of delivering CPR was switched for the next 4 minutes. The values needed to calculate CPP were measured, as was regional blood flow to the heart and the brain (using neutron-activated microspheres). For the “BLS” scenario, no epinephrine was used. In the “ACLS” scenario, a 0.5 mg bolus of epinephrine was administered followed by 4 mcg/kg/min infusion of epinephrine.
CPR was initiated simultaneously with the administration of epinephrine. Results (see Figure 7 below):

- Without the use of epinephrine (BLS), the AutoPulse was able to produce a mean CPP of 21 mm Hg – well above the important 15 mm Hg threshold necessary for ROSC as previously described. In comparison, the mean CPP was only 14 mm Hg for conventional CPR.
- The AutoPulse produced 36% of normal coronary flow vs. only 13% produced by conventional CPR without the use of epinephrine.
- When epinephrine was administered to the animals early in the course of the arrest (“ACLS”), the AutoPulse generated blood flow to the heart and brain that was equivalent to pre-arrest levels of flow. This result predates the recent article by Pytte in Resuscitation which shows that with manual CPR the effect of epinephrine is minimal yet mechanical support provides sufficient perfusion for the drug to be effective.

### Figure 7. Hemodynamic parameters in AutoPulse study vs conventional CPR with porcine model (Halperin)

<table>
<thead>
<tr>
<th></th>
<th>Myocardial Flow (% of Pre-arrest Level)</th>
<th>Cerebral Flow (% of Pre-arrest Level)</th>
<th>CPP at 1 minute (mm Hg)</th>
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<tbody>
<tr>
<td><strong>BLS</strong></td>
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<tr>
<td>AutoPulse</td>
<td>36% (+12%)</td>
<td>36% (+10%)</td>
<td>21 (+2)</td>
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<tr>
<td>Conventional CPR</td>
<td>13% (+3%)</td>
<td>28% (+11%)</td>
<td>14 (+2)</td>
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<tr>
<td></td>
<td>p=0.07</td>
<td>p&lt;0.6</td>
<td>p&lt;0.0001</td>
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<td><strong>ALS</strong></td>
<td></td>
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<tr>
<td>AutoPulse with epinephrine</td>
<td>127% (+36%)</td>
<td>129% (+27%)</td>
<td>45 (+3)</td>
</tr>
<tr>
<td>Conventional CPR with epinephrine</td>
<td>29% (+11%)</td>
<td>31% (+6%)</td>
<td>17 (+2)</td>
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<td></td>
<td>p&lt;0.02</td>
<td>p&lt;0.003</td>
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Another porcine study reported in 2006 by Ikeno at Stanford University Medical Center was designed to evaluate the affect of the high levels of CPP and blood flow produced by the AutoPulse on survival. VF was induced for eight minutes during which no interventions were performed (typical down time of pre hospital arrest). Then compressions were initiated for 4 minutes, after which ACLS interventions were performed for another 4 minutes. Compressions were randomized between the Thumper and the AutoPulse. End points for the study were ROSC, 24 hour survival and neurological status at 24 hours. 16 of the 22 pigs receiving the AutoPulse had ROSC and survived 24 hours, while none of the pigs receiving conventional CPR survived. See Figure 8.
Figure 8. 24 hour survival with AutoPulse vs conventional CPR (Ikeno)

88% of the AutoPulse survivors were found to be neurologically normal (i.e. they did all the ‘typical’ things that pigs do [walk around, eat, snort, etc.]). In fact, the arm of the study with conventional CPR was closed because the researchers were concerned that all they were doing was just sacrificing pigs.

Impressive results, but how does the AutoPulse perform on humans? In a study reported by Timerman in Sao Paulo, Brazil, 16 terminally ill patients who arrested in the hospital had catheters inserted into their aorta and right atrium to calculate CPP. After a minimum 10 minutes of failed ACLS and 30 minutes during which catheters were placed, the intubated patients received AutoPulse CPR alternating with manual CPR by residents for 90 seconds each. There was a 33% increase in CPP with the AutoPulse despite the long delay (p=<0.015). Further, the manual CPR group barely averaged 15 mm Hg CPP, the minimum threshold established by Paradis for ROSC. See Figure 9.
Figure 9. Human study of CPP with AutoPulse vs conventional CPR (Timerman)

Casner’s study with the San Francisco Fire Department investigated out-of-hospital patients who received CPR with the AutoPulse an average of 15 ± 5 minutes after the dispatch call was received. These 69 patients were matched to 93 patients treated with manual CPR from the study period or the preceding 12 months. The primary endpoint was to determine the impact of the AutoPulse on patients delivered to the ED with ROSC. Use of the AutoPulse was associated with an increased rate of ROSC compared to manual CPR (39% vs 29%; p=0.003). This was most pronounced when the presenting rhythm was asystole or PEA.

A similar short-term survival study was reported by Swanson at the 2006 American Heart Association Scientific Sessions on the use of the AutoPulse with EVAC Ambulance, serving Volusia County in Florida. The AutoPulse was used with 269 victims out-of-hospital by certified paramedics until ROSC or until death was declared. A manual CPR comparison group contained 607 patients. CPR with the AutoPulse increased short-term survival overall significantly compared to manual CPR (28% vs 18%, p=0.001). End tidal CO2 was significantly improved at 4 sequential time points following intubation using the AutoPulse compared to manual CPR with a p value <0.01 for each. (See Figures 10 and 11 below.)

Another important data point is that while there was no significant difference in outcomes when patients presented with shockable rhythms (33% for manual CPR vs 31% for AutoPulse CPR; p=0.85), PEA and asystole showed significantly increased short-term survival (PEA: 17% vs 32%; p=.04; asystole: 10% vs 24%; p=0.01). This difficult patient population is very perfusion dependent, and the use of AutoPulse appears to be of significant benefit.
The AutoPulse Pre-hospital International Resuscitation trial (ASPIRE) was a randomized (“cluster” randomization), multi-center trial which took place in 5 sites in North America: Vancouver, BC; Calgary, Alberta; Columbus, OH, Pittsburgh, PA and Seattle, WA. 1,071 patients were actually enrolled in the trial, however the researchers focused the majority of their analysis on a “primary comparison” group for which the primary cause of the arrest was cardiac. This group contained 767 patients: 373 who were treated with manual CPR and 395 treated with the AutoPulse. The primary endpoint of the study was 4 hour survival, used to simulate hospital admission. The secondary endpoint was survival to discharge. The trial was terminated based upon statistically insignificant secondary endpoint data, thus the results are inconclusive. The problems with ASPIRE appear to be related to study issues and not the AutoPulse itself. Among the implementation issues were inconsistent implementation and very late use of the AutoPulse (an average of 12 minutes after 911 dispatch), as well as spotty training and monitoring at the various sites. There were also issues with the study design, including the fact that multiple protocols were involved and changes amongst the protocols allowed even after the trial started. The one site that changed protocols had a disproportionate impact on the combined data from all five sites.

An important long-term survival study was conducted in Richmond, Virginia. In this pre hospital study by Ong survival rates were compared for 499 patients treated with manual CPR from January 1, 2001 - April 1, 2003 to 284 patients treated with the AutoPulse from December 20, 2003 - March 31, 2005. The mean time interval for the EMS ambulance to arrive at the location was 6.1 minutes, and the median time interval from the crew at the patient side to the AutoPulse attached and started was 3.6 minutes. ROSC in the field increased from 20.2% with manual CPR to 34.5% with the AutoPulse. Hospital admission rose from 11.1% to 20.9%. The most impressive result was the increase in survival to discharge from 2.9% to 9.7%! See Figure 12. The benefit was relatively robust across a range of patient subsets, especially for those patients with VF initially, bystander witnessed events, and recipients of bystander CPR.
A new randomized multi-center study is about to begin at several sites in the United States and Europe, lead by Lars Wik, MD, PhD, a world-renowned researcher in the field of cardiac resuscitation. It will be important in this research to pay attention to the definition and consistency of the method for using the AutoPulse, to measuring the multiple time intervals with precision, and to ensure the quality of manual CPR administered in both groups.

**Conclusion**

Consistent high-quality CPR is an essential element in survival from cardiac arrest, yet it is delivered inadequately and with interruptions by care providers. The heart needs a consistent coronary perfusion pressure above 15 mm Hg for survival to be possible. The AutoPulse can take control of CPR in cardiac arrest - giving clinicians peace-of-mind knowing that the best possible CPR is being delivered, thus eliminating the variables that occur when performed by humans and improving the likelihood of neurologically intact survival - the ultimate goal of resuscitation.

**References**


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