

#### Squeezing High Performance Out of CPR Compressions

A Symposium of the 8th Scientific Congress of the European Resuscitation Council Stavanger, Norway, May 11, 2006



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#### COURSE INTRODUCTION / METHOD OF PARTICIPATION

To complete this course, you must do the following:

- Read the Needs Statement/Overview and Course Objectives.
- Study the Terminology.
- Study the Content Review.
- Complete the comprehensive Post Test (online at www.medcomrn.com/zoll
  or via the answer form at the back of this booklet).

The American Medical Association designates this continuing medical education activity for up to 1 hour of category 1 credit towards the AMA Physician's Recognition Award. Each physician should claim only those hours of credit that he/she spent in the educational activity. To receive Continuing Medical Education credit for the course, complete the comprehensive post test and activity evaluation form online at www.medcomrn.com/zoll or via the forms at the back of this booklet and follow the instructions to receive your certificate.

To receive 1 hour of Continuing Nursing Education credit for the course, complete the comprehensive post test and activity evaluation form online at www. medcomrn.com/zoll or via the forms at the back of this booklet and follow the instructions to receive your certificate.

#### NEEDS STATEMENT / OVERVIEW

For years, cardiopulmonary resuscitation (CPR) has taken a secondary position relative to defibrillation in lifesaving. In the following pages, four noted experts on the theory and practice of CPR discuss the latest science on the performance, hemodynamics and cellular biochemistry of CPR, the latest American Heart Association (AHA) and European Resuscitation Council (ERC) recommendations on compressions, and laboratory studies of the effectiveness of manual vs. automatic CPR. These experts point out the critical importance of CPR quality in influencing outcomes in resuscitation.

#### INTENDED AUDIENCE

Physicians, nurses, paramedics, emergency medical technicians (EMT) and other healthcare professionals who may be required to perform CPR.

#### PURPOSE AND OVERALL GOAL

The purpose of this activity is to provide physicians, nurses, and other healthcare professionals with an understanding of the current theory on the chemistry and hemodynamics of CPR, and the importance of performing CPR correctly per the AHA recommendations.

#### **OBJECTIVES**

This booklet is a collection of transcripts from multiple presenters at the Symposium of the 8th Scientific Congress of the European Resuscitation Council, Stavanger, Norway. Objectives for each presentation are contained within their respective section of the booklet.

#### FACULTY DISCLOSURE STATEMENT

The authors/presenters of the sections contained herein have indicated that they have no financial arrangements or affiliations with any of the corporate organizations offering financial support or educational grants for this activity.

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## Terminology

**AED** Automated External Defibrillator, a device that analyzes the heart's rhythm for abnormalities and directs the rescuer to deliver an electrical shock, if necessary

**ADP** Adenosine diphosphate, a common body chemical found after energy is released

**ATP** Adenosine triphosphate, a common body chemical in which energy is stored in living organisms

**Coronary perfusion** Blood flow through the coronary vessels that supply blood to the myocardium

**CPP** Coronary Perfusion Pressure, the aortic to right atrial pressure gradient during the relaxation phase of CPR. CPP is a surrogate measure for actual blood flow to the myocardium. CPP values during resuscitation have also been shown to be predictive of resuscitation success

**Defibrillation** An electrical shock delivered through the chest to the heart to try to help the heart reestablish an effective rhythm

**Dysrhythmia** Cardiac rhythm disturbances

**Gaussian distribution** The normal distribution, which often takes the form of a bell curve when graphed

Histogram A graphical display of tabulated frequencies

**Ischemia** A cellular reaction to a local deficiency of blood, generally due to a blockage

Myocardium Heart muscle

Myocytes Muscle cells

**Pentazocine** An opioid receptor agonist

PO<sub>2</sub> Myocardial oxygen

**Ventricular fibrillation** Chaotic electrical activity of the heart, resulting in little or no blood flow

## Section 1: Go With the Flow: Blood Flow and its Importance in Improving Resuscitation

By: Wanchun Tang, MD, FCCP, FCCM Weil Institute of Critical Care Medicine Rancho Mirage, CA, USA

#### INTRODUCTION

Some perfusion from red blood cells continues for about four minutes after ventricular fibrillation, but it is essential for resuscitation to maintain the recommended rate of CPR compressions, and without pauses. Manual compressions are less effective than automated. It is important for all healthcare workers to be aware of these results

#### PURPOSE AND OVERALL GOAL

The purpose of this section is to offer data on the state of the heart after the heart stops, and how this affects CPR practices.

#### **OVERVIEW**

This section offers data on the state of the heart after the heart stops, and how this affects CPR practices. The material is organized around the following categories:

- Introduction
- The Red Cells Continue Bringing Oxygen
- Electrophysiology
- Difficulties of CPR
- Two Rescuer CPR
- Conclusion

#### SECTION OBJECTIVES

After completing this section of activity, the learner should be able to:

- Describe how perfusion and myocyte activity continue after ventricular fibrillation.
- Describe the importance of maintaining a high rate of chest compressions without interruptions.

## Section 1: Content Review

#### INTRODUCTION

This section will address two issues. First, it will look into the mechanisms of why immediate defibrillation is only effective during the first three to four minutes of untreated ventricular fibrillation (VF). After that, chest compressions are the only factor that will result in a successful resuscitation. And the second issue addresses why manual chest compressions are less effective.

About five years ago, there was a study at the Weil Institute of Critical Care Medicine concerning actively protecting the myocardium during cardiac arrest using a drug called Pentazocine, an opioid receptor agonist. After induced cardiac arrest, when myocardial PO<sub>2</sub> was measured, the study showed that myocardial PO<sub>2</sub> did not go to zero until four minutes later. That was a surprise to the investigators and totally against the traditional textbook physiology. When the cardiac output ceases, it's expected that the heart will become ischemic immediately. The experimenters were worried about the accuracy of the sensor and recalibrated it many times. They still could not explain why myocardial PO<sub>2</sub> did not go to zero immediately (see Figure 1).

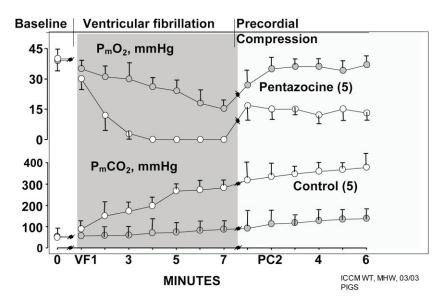


Figure 1

#### THE RED CELLS CONTINUE BRINGING OXYGEN

This question was only answered five years later when Dr. Mike Fries from Germany, using a technology called orthogonal polarization imaging, was able to look deep into the tissue. Figure 2 demonstrates the movement of the red cells.

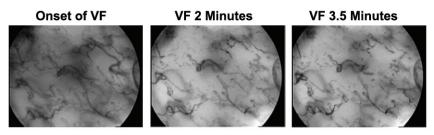


Figure 2

During normal circulation, one observes rapid movement of the red cells. When VF is then induced, the rapid movement continues even though the cardiac output is zero—those red cells are continuously bringing oxygen to the myocardium—and there is no ischemia.

At two minutes the red cells were still going even though the heart had been stopped all that time.

Three and a half minutes later there was still some small activity going. Then the experimenters realized that activity was closely related to the resting pressure between the left side and the right side. In other words, it took three to five minutes until the arterial and venous pressures balanced, and, until they did, the red cells continued to flow, bringing oxygen to the myocardium.

#### **ELECTROPHYSIOLOGY**

Electrophysiology can also explain why CPR should be resumed immediately after the first three or four minutes of VF.

There is a reentry mechanism (Figure 3). At the beginning of VF there are a few large reentry wave fronts. During VF the myocytes do not stop contraction. They just are not organized and they work independently.

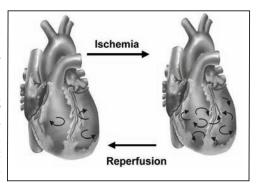


Figure 3

Following prolongation of cardiac arrest, more and more wave fronts develop. In other words, more and more myocytes contract and relax independently. That is what increases the difficulty of defibrillation (Figure 4).

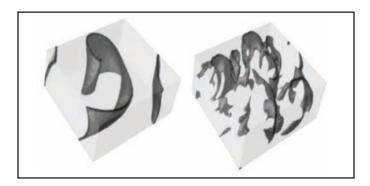


Figure 4

By some unknown mechanism, when heart perfusion is induced, this process can reverse—even though the myocytes cannot spontaneously defibrillate, they can reverse this disintegration process.

Figure 5 shows a micro-current of the myocardium that confirms the experimental hypothesis. At the beginning of VF, there are large and few wave fronts. After five to six minutes, more and more myocytes contract independently.

After 1 minute of VF, you see few wave fronts with very high amplitude and rapid frequency. This confirms that a large number of myocytes are still contracting and relaxing spontaneously and cooperating.

Following the prolongation of cardiac arrest, the amplitude goes down and the frequency slows. This means more and more myocytes are working independently.

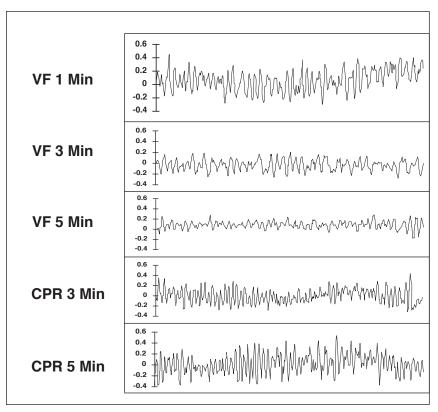


Figure 5

After you start reperfusion and begin chest compressions, there are a lot of myocytes still contracting independently, but CPR can actually "line up" all that independent activity of the myocytes and make them contract more nearly simultaneously. As more and more myocytes contract simultaneously, coarse VF occurs—and that is very easy to defibrillate.

Our hypothesis has been confirmed by mapping technology that tells us that the VF waveform really represents how many myocytes are actually contracting together or independently.

Chart 1 is data from a study done by Dr. Lars Wik. Our study is consistent with Dr. Wik's study that shows after five minutes of cardiac arrest, one needs to do CPR first to improve the outcome of cardiac arrest.

## CPR first prior to defibrillation in OHVF

(Survival to hospital discharge)

Response time < 5 min:</li>

CPR first (3 min): 23 % ( 9/64)
Defibrillation first: 29 % (12/55)
OR 0,70 (IC 95% 0,26-1,91) p=0,61

· Response time > 5 min:

CPR first (3 min): 22 % (14 /40)
Defibrillation first: 4 % (2/41)

OR 7,42 (IC 95% 1,61-34,3) p=0,006

Wik L, et al. JAMA 2003; 289:1389-95

ICCM, WT, 01/04

#### Chart 1

In the United States, during the last two decades, the incidence of VF as a first presenting rhythm has decreased very rapidly (Figure 6). This is because of the widespread use of the so-called primary and secondary prevention strategies—beta blockers and calcium channel blockers. These medications make VF very short-lived—one only sees VF for the first few minutes. Chest compression then becomes even more important.

#### Reduced Incidence of VF as the First Presenting Rhythm

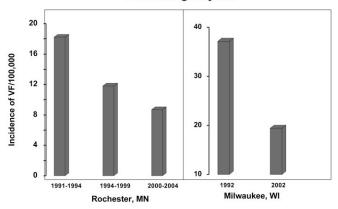


Figure 6

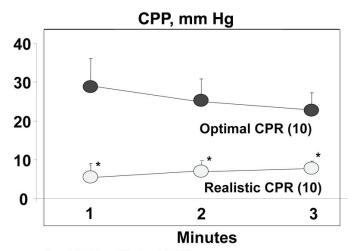
#### DIFFICULTIES OF CPR

The next issue to be addressed is why performing good CPR is so difficult. Even with ideal conditions, the maximum flow generated by CPR compressions is about 20% of normal cardiac output. This has been demonstrated in both the laboratory and the clinical setting. The new AHA/ERC guidelines put the emphasis on chest compressions: push harder and push faster, allow complete chest recoil, and minimize interruptions.

It is very hard for a human being to comply with all these requirements. This has been shown in the papers by Dr. Lars Wik and by Dr. Ben Abella in out-of-hospital situations and in-hospital, respectively. The conclusion is, regardless of how well people are trained, they just can't do good CPR.

The two papers emphasized that both the rate and the depth were poor– slow rate and shallow depth.

We wanted to set up a study to separate these two variables to see which one was most important. We hypothesized that if you kept the rate constant and reduced the depth by 25%, as the other studies reported, perhaps depth really wouldn't matter. We used mechanical compression set to 100 times per minute and reduced depth of compression by 25%. The outcome surprised the experimenters (Figure 7).



\*p < 0.0001 vs "Optimal CPR"

Figure 7

The Coronary Perfusion Pressure (CPP) is significantly different—it's too low. And this level totally excludes the possibility of successful resuscitation. That means that compression depth is really very important, and a reduction in depth of only 25% has profound effects in terms of how the heart is perfused; what we call "realistic CPR" actually generates only about 10% of the flow compared to baseline.

Figure 8 shows the outcome. ROSC was achieved in only about 12% of cases with realistic CPR and 100% of the time with optimal CPR.

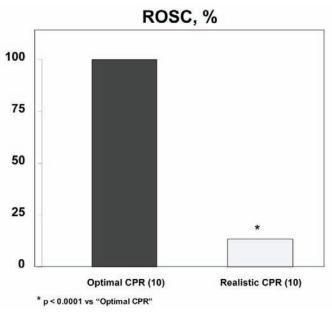


Figure 8

We videotaped CPR being performed well and poorly, using orthogonal polarization imaging. The only difference was that the poor CPR was performed with 25% shallower compressions (Figure 9).

## **Quality of Chest Compression**

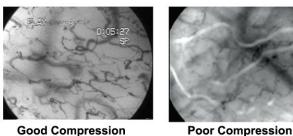
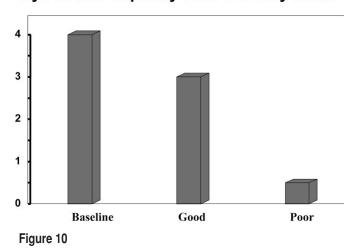


Figure 9

We were then able to quantify the activity seen and call it the myocardial capillary flow velocity index. The calculated result is shown below (Figure 10).

## **Myocardial Capillary Flow Velocity Index**



The poor CPR generated less than 10% of the flow as compared with baseline.

#### TWO RESCUER CPR

We decided to measure whether human beings could perform what we called "good CPR." We took two average rescuers in their thirties and asked them to perform two-person CPR and we measured compression rate and CPP and also microcirculation flow (Figure 11).

#### **How Effective is the Manual Chest Compression?**

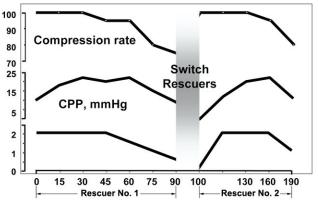


Figure 11

The best they could do was maintain "good CPR" for about 90 seconds - actually it's not 90 seconds. One can already see the rate dropping, even though we used a metronome to try to maintain rate, the rate is dropping. The coronary perfusion pressure is dropping, and the myocardial microcirculation is dropping.

At 90 seconds, we switch rescuers—another way to continue good quality CPR. It should have been quick to switch from the head side to the body side, but it took ten seconds to switch and then fully 60 seconds to return to optimal CPR. And then the rate started declining again. Our conclusion was that a human being just cannot do this task effectively for very long.

#### **CONCLUSIONS**

Some conclusions were inescapable. When cardiac arrest is prolonged to greater than four minutes, regardless of the first presenting rhythm, reestablishing blood flow to the vital organs is the single most important determinant of successful resuscitation.

The second conclusion is rather negative: significantly improved outcomes with CPR can only be established when constant and effective chest compressions with minimal interruptions are provided. This can probably only be done by a mechanical device.

What we see right now widely applied in the United States is defibrillators. One hopes that a few years from now we will see a device called an automatic mechanical chest compressor standing beside the defibrillator.

## Section 2: We Do Good CPR – Right?

By: Benjamin S. Abella, MD, MPhil University of Chicago, Chicago, IL, USA

#### INTRODUCTION

Some of the first work looking at CPR quality was observational studies in a hospital in the mid 1990s. But they were subjective studies. In 2004, Aufderheide in Wisconsin looked at paramedic delivery of CPR objectively during out-of-hospital cardiac arrest. It is important for all healthcare workers to be aware of these results.

#### PURPOSE AND OVERALL GOAL

The purpose of this section of the activity is to offer data on the state of current CPR practices.

#### **OVERVIEW**

This section offers data on the state of current CPR practices. The material is organized around the following categories:

- Introduction
- Testing CPR
- In-Hospital Arrest
- The Danger of Relying on Mean Rates
- Resuscitation
- Mechanism of Chest Compressions
- Solutions
- Conclusion

#### SECTION OBJECTIVES

After completing this section of activity, the learner should be able to:

- Describe the results of recent objective trials of CPR.
- Describe the importance of maintaining a high rate of compressions.

## Section 2: Content Review

#### INTRODUCTION

Dr. Tang reviewed some of the physiology underlying the need for good CPR compressions. This section will step back a little bit, and discuss some of the data that shows how well CPR is performed in the early 21st century.

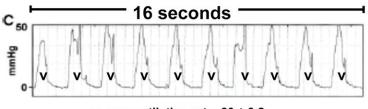
Some of the first work looking at CPR quality was done in Arizona in the mid 1990s, namely, observational studies in a hospital. But these were subjective studies, and they didn't cause many changes. However, in 2004, Aufderheide in Wisconsin looked at paramedic delivery of CPR during out-of-hospital cardiac arrest. He looked at it objectively, which was key.

There have been a number of studies looking at CPR quality using mannequins. These studies don't really simulate the stressful environment of real arrest. There are other studies that look at CPR in real arrest, but do it subjectively. Some of these have problems with methodology, because an objective measure is needed.

#### **TESTING CPR**

Aufderheide and his group did tracings much like those shown in Figure 1 below, where you can see each ventilation marked with a "v."

## Hyperventilation in out-of-hospital cardiac arrest



mean ventilation rate: 30 ± 3.2

Aufderheide et al, 2004

Figure 1

This is from a tracheal pressure monitor, and what he found was that ventilation rates during paramedic cardiac arrest were quite high, rates in the 20s and 30s. He then took these rates and replicated them with experimental pigs, and found that all the pigs died. These rates were highly detrimental. There are a variety of pathophysiologic reasons why this might be the case, but suffice it to say that hyperventilation was a big problem in actuality, and then it proved to be a big problem for survival.

We looked at compression rates in a quantifiable fashion in our hospital, the University of Chicago, in 2005. Figure 2 shows a frequency histogram of chest compression rates. On the bottom axis are rates, 10 to 20, 20 to 30, etc. The Y-axis is the frequency at which that rate came up. This is the entire cohort of the arrests in our study, broken into 30-second segments, for which we calculated rates.

## **Chest Compression Rates**

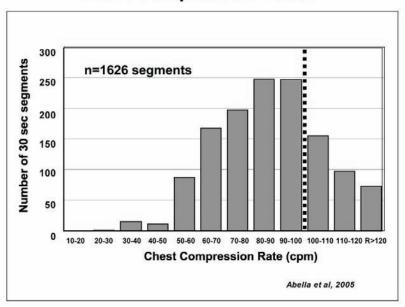


Figure 2

You can see a reasonably Gaussian distribution. Note: the vertical dotted line represents the AHA standard, 100 compressions per minute.

You can immediately see that rates are extremely variable. Also, a lot of rates are too low. When chest compression rates get in the 70s and 80s, as we often find during a real arrest, CPP falls off essentially to zero. So rates in the 70s or 80s were barely doing anything for patients. And we found this was quite common among trained physicians, even ACLS trained physicians.

#### IN-HOSPITAL ARREST

To determine if the rate really mattered during in-hospital arrest, we took the data from animal tests (Figure 3) and did a post hoc analysis. We broke it down to those who survived (shaded bars on the histogram) and those who did not (white bars).

## **Compression Rate and Survival**

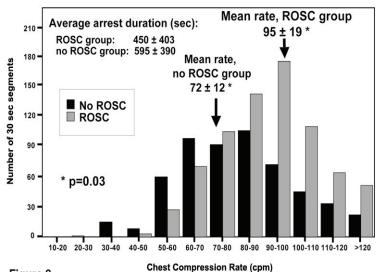


Figure 3

When one looks at a group that survived, their mean chest compression rate was actually very close to AHA Guidelines. It was quite surprising how close it was. When one looks at the group that did not survive, they had a much lower mean chest compression rate, in the low 70s.

There are problems in post hoc analysis. This was not a randomized controlled trial. A control group would be impossible with humans—it would simply be unethical to subject one group to a low chest compression rate. Nonetheless, the marked difference in rate was significant. This was one of the first demonstrations in human cardiac arrest that chest compression rate has an importance in survival.

We were very excited by this, which led us to embark on a study using a defibrillator, much like Lars Wik used in Norway, to look at chest compression rates, ventilation rates, and other parameters of CPR, taken together during in-hospital cardiac arrest. This led to our joint publications in *JAMA* last year. Now we will look at some of that data.

#### THE DANGER OF RELYING ON MEAN RATES

Figure 4 shows a frequency histogram, with chest compression rate on the X-axis, the frequency at which that rate comes up on the Y-axis.

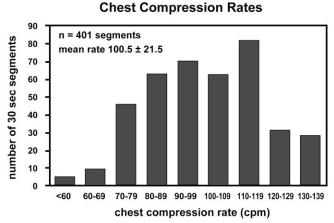


Figure 4

This demonstrates an important issue. When you look at the mean rate for all of the compressions, it was 100. A hundred compressions per minute—one might think that's perfect. But the important lesson here is that mean rates and mean CPR characteristics can be very deceiving.

As we all think about CPR quality, we have to look deeper than mean rates. Because a patient doesn't care, physiologically, about an average. If a patient gets a minute of very bad CPR, followed by a minute of very good or overly good CPR, the average comes out good, but does the patient say, "Well, that's fine. It averages out." No. The patient cares that a minute was very bad CPR, and the patient may not recover. Note that there is a very wide distribution with a lot of low chest compression rates that are definitely bad CPR.

When we looked at ventilation rates, we saw a very similar finding (Figure 5).

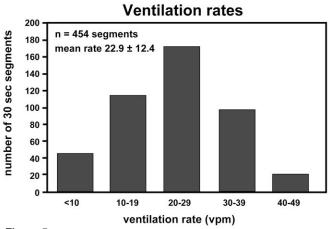


Figure 5

We found that ventilation rates were quite high, with a mean higher than what is standard. In the 2005 AHA/ERC Guidelines, there's been a de-emphasis on ventilations. Nonetheless, whichever guidelines you're using currently, ventilation rates should be at the low end. We're seeing 20, 30, 40—consistent with Aufderheide and consistent with some mannequin studies as well. So, it's very fast.

It is easy to imagine why this is the case. Try to do twelve ventilations a minute in a room with people screaming, adrenaline flowing, and twenty people pushing around. Anyone with a bag is going to squeeze that bag and keep on squeezing. Human beings find it very hard to step away and be calm and not touch something for five seconds during a cardiac arrest. We think we understand the human factors behind this conclusion. In this case, it's so bad that even the mean is way off.

When we look at chest compression depth, again we find a Gaussian distribution (Figure 6).

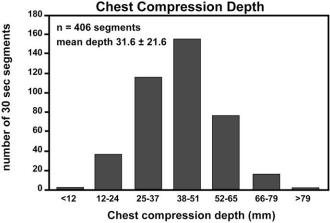


Figure 6

This chart is in millimeters. The American Emergency Cardiac Care (ECC) Guidelines are 1.5 to 2 inches, which translates to 38-51 millimeters as seen in the tallest bar. Once again, we can see some compressions that are too shallow, some too deep—a wide distribution. It's possible that the data is skewed a little bit in favor of showing good results, because we don't know if backboards were used in all of the arrests. And, given the current measurement technology, if a backboard wasn't used, it would over-calculate and think the compression was deeper than it was. Probably this curve is, in reality, shifted a little bit to the left.

This study led to two publications looking at in-hospital and out-of-hospital arrest survival. These studies are discussed in other sections of this course. Here we want to look briefly at the study by Lars Wik. His results are shown in Table 1 on the next page.

In that study, there were very low chest compression rates, with lots of pauses. The results were very consistent in both the in-hospital and out-of-hospital setting. The important theme here is that this is an endemic problem. It is not a specific institution, a specific setup.

## **Out-of-hospital CPR quality**

Performance of CPR During the First 5 Minutes and Entire Episode of CPR\*

	First 5 Minutes of CPR	Entire Episode of CPR
No flow (n = 176)		
NFR, %	49 (21)	48 (18)
NFR <sub>adi</sub> , %	42 (19)	38 (17)
Compression (n = 176)†		
Compressions/min	60 (25)	64 (23)
Compression rate, /min	120 (20)	121 (18)
Depth per episode, mm	35 (10)	34 (9)
38-51 mm with complete release	27 (30)	28 (25)
T∞ deep (>51 mm), median (IQR)	0 (0-3)	0 (0-5)
Too shallow (<38 mm)	59 (37)	62 (33)
Incomplete release, median (IQR), %	0 (0-1)	0 (0-2)
Duty cycle, %	41 (5)	42 (4)
Ventilation (n = 163)	0.44.03	44.44.70
Ventilations/min	8 (4.6)	11 (4.7)

#### Table 1

Wik et al 2005

This same problem has also been demonstrated by others. Valenzuela came out with a study last year (Table 2), looking at out-of-hospital arrests.

**Out-of-hospital CPR quality** 

	First 5 Minutes	Entire Effort	Р
Time with CCs, %	40±21	43±18	NS
Time without CCs, %	$60 \pm 21$	57±18	NS
Longest period with CCs, seconds	65 (46, 84)	122 (68, 206)	0.0001
Average period with CCs, seconds	46 (30, 67)	55 (43, 74)	NS
Longest period without CCs, seconds	95 (70, 147)	172 (109, 246)	0.0001
Average period without CCs, seconds	56 (41, 87)	57 (40, 78)	NS

Table 2 Valenzuela et al., 2005

This study, too, found long pauses, and, in essence, low chest compression rates by virtue of the pauses.

In summary, wherever CPR quality has been studied, experimenters have found low chest compression rates, high ventilation volumes, and shallow depths. Those are the most important CPR errors that are commonly found.

What can be done? The Guidelines address this in several ways. First of all, they changed the ratio from 15 to 2 to 30 to 2, to de-emphasize ventilations, and bring compressions up. Importantly, as well, they put specific language into the Guidelines that CPR performance should be monitored, and that we should develop systems approaches to improve resuscitation quality, and to address deficiencies. This is a challenge to everyone to help improve the quality of CPR.

#### RESUSCITATION

I want to move on to a related topic, but one on which you haven't necessarily seen the data. This is work we have in press in *Resuscitation*. My colleague, Donna Edelson, did some analysis of our SISTER data. (SISTERS are a collaborative group of CPR quality investigators).

It turns out that pauses matter, but pauses also matter in relation to defibrillation. Some people still believe defibrillation is the most important thing we can do for resuscitation, but our data says defibrillation is directly affected by the CPR quality immediately preceding that shock.

Figure 7 shows the impact of compression depth or shock success. Again, shallow compressions significantly affect outcome.

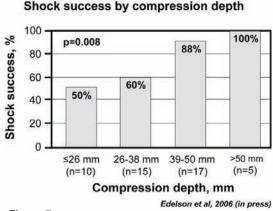


Figure 7

Figures 8 shows the data related to pauses in CPR before a shock. The X-axis shows the length of the pauses immediately before defibrillation—less than 10 seconds, 10 to 20, 20 to 30, and greater than 30. The Y-axis is the percentage of time this shock was successful. There was a very striking relationship. As the pause increased, the chance of success markedly fell.

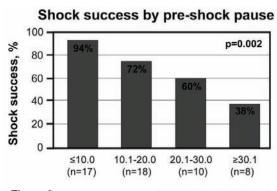


Figure 8

Edelson et al, 2006 (in press)

This is probably consistent with some current thinking, in that those who know about AICDs know that a very quickly administered shock is generally quite successful. But the success rate falls off very rapidly.

This has very striking implications for those who use AEDs. There are many AEDs that are currently on the market that have analysis times in the 20-second range. So they're not as good as they could be. The possibility of success is already dropping off at 20 seconds. It's very important to keep these pause times short.

#### MECHANISM OF CHEST COMPRESSIONS

This brings us to the question of why this might be the case—why pauses and depth matter before a shock. Here we need to discuss mechanisms. Figure 9 shows what might be going on in myocardial cells.

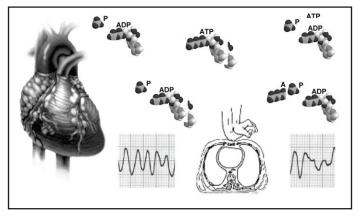


Figure 9

For the heart to work, it needs to keep its membrane potential intact, and it needs to keep its calcium sequestered within cells.

These processes are ATP dependent. Ventricular fibrillation, exhibited here, is a very energy-intensive process—such that ATP molecules are broken up. Membrane potentials degrade. Myocardial function decreases, and the heart is less receptive for a shock.

If the person receives good CPR immediately before a shock, the body can reconstitute ATP. It can reconstitute myocardial function. It's almost as if the heart says, "Yes, now I'm ready for a shock." This is an important mechanism that we believe explains why good CPR is so important for defibrillation efficiency.

#### **SOLUTIONS**

There are a variety of solutions to the problems of CPR, one of which is mechanical CPR. But there are other issues to consider in the whole spectrum of how to address the ECC challenge. One: we need to improve our training modules through simulators, and through other methods. Also, we need software that filters out compressions from AEDs or analysis of rhythms. We need to be able to watch the heart rhythm, right through the compressions, and have the apparatus say "Shock," so that you could take your hands off, just before a shock. That would be ideal.

Figure 10 shows data, again from our colleagues in Norway, where they were able to filter out compressions and see underlying data.

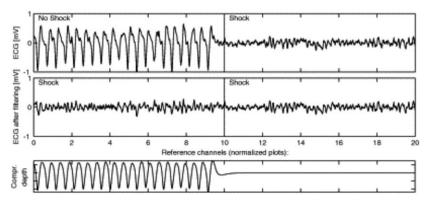


Figure 10

Finally, there are really two solutions to improvement in CPR quality during arrest. One is mechanical CPR, and the other is technology that gives feedback used to measure how a patient is doing during CPR, record it, and then spit it out. It could give oral messages to the team saying, for example, "Speed up compressions," or something like that. Figure 11 is a device made by ZOLL that has a sensor on the chest that detects chest compression rate and depth. Such devices, with feedback enabled, will allow manual CPR to improve.



Figure 11

#### **CONCLUSION**

Will these solutions be able to match mechanical CPR? The data to come in the next few years will tell us. These are both solutions that may have application in different places. They may have strengths and weaknesses for different environments. Nonetheless, there are a number of approaches, and it's time to implement them now.

## Section 3: Harder, Deeper, Faster: Manual CPR and Mechanical CPR — Getting the Best from Both

By: Lars Wik, MD, PhD Ulleval University Hospital, Oslo, Norway

#### INTRODUCTION

Several studies have shown that compressions with appropriate depth are very important. We also know that the appropriate rate is important. The third thing, which is also very important, is the pauses: The fewer pauses the better. It is important for all healthcare workers to be aware of these facts.

#### PURPOSE AND OVERALL GOAL

The purpose of this section of the activity is to offer data on the importance of depth and rate of compressions and lack of pauses in performing CPR.

#### **OVERVIEW**

This section offers data on the importance of depth and rate of compressions and lack of pauses in performing CPR. The material is organized around the following categories:

- Introduction
- Laboratory Study
- Trapezoidal Waveforms
- Compression Depth
- Conclusion

#### SECTION OBJECTIVES

After completing this section of activity, the learner should be able to:

- Describe the importance of maintaining the proper depth in performing CPR.
- Describe the importance of maintaining a high rate of chest compressions in performing CPR.
- Describe the importance of minimizing pauses in performing CPR.

## Section 3: Content Review

#### INTRODUCTION

Many studies have shown us how CPR must be performed today in order to delay the process of death. We have learned from several studies that compressions with appropriate depth are very important. We also know that the appropriate rate is important because even though there is a tendency for ROSC to increase up to about 100 compressions a minute, the rate then tends to decrease.

The third issue, which is also very important, is the question of pauses. The fewer pauses the better.

The question remains: how are we performing in the real world? The data suggests we are not actually performing very well. The conclusion is clear. We are not doing what is meant to be done when we confront a cardiac arrest. And, we are doing a lot of things which are not meant to be done. We are simply not doing chest compressions 50% of the time. We must change all that.

We are also not compressing well enough. When we get feedback from a machine we press harder and deeper. But when we are focusing on other aspects of performing CPR, we go back to the old ways—we press slower and weaker.

The number of compressions with correct depth increases with feedback, but decreases after we remove feedback. And, how does CPR influence hemodynamics and flow? That's the key question because if it doesn't influence hemodynamics and flow, it's only empty statistics.

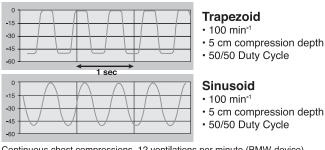
#### LABORATORY STUDY

There was a study in our lab at the Ulleval University Hospital in which we built a new mechanical device in order to mimic the typical clinical manual CPR performance. Jo Kramer-Johansen was the principal author on this paper and Figure 1 displays some of the data.

We looked into what we called compression waveforms.

We can provide compressions in a trapezoidal way or in a sinusoidal way.

Manual CPR is typically a sinusoidal waveform. We are not able to do trapezoidal chest compressions manually. Machines can, but humans aren't able to hold the chest down long enough. Figure 1 on the following page shows two graphs of waveforms of how CPR can be performed.



Continuous chest compressions, 12 ventilations per minute (BMW-device)

Figure 1

There are clinical differences in the matter of hemodynamics and flow generated by these two approaches. If we look at mean arterial pressure, developed by the typical out-in-the-street CPR, we reach lower mean arterial pressures than we reach with a trapezoidal form of CPR. And remember, in our testing it was a machine that performed the manual CPR. So we cannot say that the performer was getting fatigued or factors like that. Humans always tend to perform 100% sinusoid chest compressions.

The other result was that there was a difference in mean right atrial pressure. These two numbers, mean arterial pressure and the right atrial pressure come together and dictate the coronary perfusion pressure (CPP). The data are shown in Table 1.

	Trapezoidal	Sinusoidal	
Mean Arterial Pressure (mmHg)	36 + 10	30 + 7	p < 0.01
Mean Right Atrial Pressure (mmHg)	35 + 11	29 + 9	p<0.01
Maximum Coronary Perfusion Pressure (mmHg)	14.9	11.6	p=ns
Mean Common Carotid Flow (ml/min)	50 + 32	35 + 22	p<0.01
Mean Cerebral Cortical Flow (% of baseline)	50 + 40	40 + 35	p<0.01

Table 1

Ideally we would like to have a mixture of the benefits of the trapezoidal way of doing CPR and the sinusoidal way of doing it – because we would like to have a great aortic pressure with a low right atrial pressure.

We are now looking into the waveforms and studying the waveforms to see how we are performing CPR. In a few years we may be able to report some interesting data on this.

The other factor we noted was that the cerebral blood flow was higher with the trapezoidal compressions than with the sinusoidal compressions-let's say the typical clinical way of doing CPR.

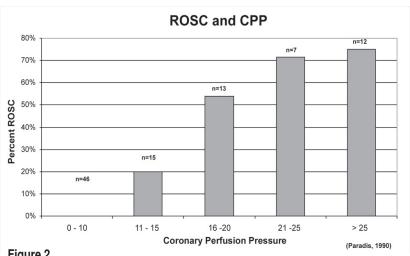
The conclusions from this study were that better hemodynamics were achieved with trapezoidal waveforms with 20% higher blood pressure and 40% higher cerebral blood pressure. But there was no significant difference in coronary perfusion pressure.

#### CORONARY PERFUSION PRESSURE

We have learned from the study, Paradis, 1990 in JAMA, that there is a limit that must be reached in order to restart hearts, dictated by the coronary perfusion pressure.

A coronary perfusion pressure of about 15 mmHg is needed in order to restart a heart. Most of the patients on whom we are performing manual-sinusoidal type-CPR don't come close to 15 mmHg in CPP and they are not resuscitated. So here we have an opportunity to improve our survival rates, because we first of all have to restart the heart in order to restart the brain and the body.

Figure 2 below shows that in patients with low coronary perfusion pressure, there are few to no instances of ROSC. For those with higher CPP, there was ROSC. And in animal experiments from 1982 and 1984, it was documented that there is a direct relationship between the compression force and the blood flow.



So first of all, we must stop performing what might be called "simple healing." If you put your hands on the chest like most physicians do and pray, that's simple healing—or laying on of hands. We must do much more. We have to compress. If we add some force to the chest, blood flow goes up, and more will survive.

But this relationship between survival and force of chest compressions—which has been shown directly on animals—has not been documented in humans until recently. The data from the SISTER study does show this: in the study we looked into compression depth and related that to survival. The harder you press, the higher the survival.

So the quality of CPR is one of the most important factors for survival, together with time, of course.

#### **CPR AND DRUGS**

If we now look at what we know about how poorly people are performing, we have a challenge.

We wanted to see if there is a relationship between the quality of CPR we are performing out in the field with respect to the epinephrine dose and the effects of that epinephrine dose, compared with optimal CPR performed by a mechanical device.

We used the same device to mimic human-performed clinical CPR, based on the SISTER data manual CPR, and compared that with mechanical CPR. We gave a dose of epinephrine and measured the coronary perfusion pressure. Our findings were impressive.

When you administer epinephrine with manual CPR, it takes a very long time before you see any effect from the epinephrine, because we are giving epinephrine in the arm, and the circulation is too slow.

However, if we give epinephrine with good chest compressions, the flow is higher and the epinephrine you put in the vein will get to the organs in time. We even saw increased coronary perfusion pressure, and the effect comes earlier than compared to a manual technique.

This illustrates that when you're doing mechanical CPR, the drug action comes earlier than when you're doing manual CPR, if you have an effect at all. The results say that in the mechanical setting, it takes 69 seconds before you see any drug effect and in manual more than two minutes.

Note that these results are in pigs. The distance from a pig's foreleg to its chest is much shorter than arm-to-chest in humans.

The other result, which is striking, is that with mechanical compressions, the brain flow increased over manual CPR. If you look at the femoral and internal carotid, the flow increases as well. This is data from Morten Pytte's work from our group, which is currently in press in *Resuscitation*.

#### CONCLUSION

We have to change the way we are performing. This is a good message to take to heart: don't think that drugs alone will have any effect in the field if you don't perform good quality CPR. In the future, there is a possibility that we will need to do all the drugs studies again with good quality CPR.

Finally, my conclusion for "Harder, Deeper, and Faster:" the best results will depend on increased flow and drug effects.

#### **QUESTIONS AND ANSWERS**

**Q:** Where people cannot afford machines, they are still dependent on humans doing CPR. And I think the emphasis must be, therefore, if you want to save as many as possible, having humans who do CPR do it well, do it hard, fast, and deep, and not push for machines to do it, because then the reach will only be to a very small percentage of the population who need it.

**WIK:** I agree with you. First of all, mechanical CPR is not meant to take away manual CPR. It is an additional tool that will come, the sooner the better, but in some societies and in some environments, it is probably not wise to introduce mechanical CPR everywhere. Because if you think that you can just stand back and prepare the device and put it on, some people will wait too long.

Q: I think we like to push hard, fast, and deep, but what is too hard what is too fast and what is too deep? Could you suggest some answers?

WIK: We are talking about resuscitation now, I guess. When you are talking about fast, it's clear? There is no reason to go above 100 per minute. The human brain is not able to compress your own hands fast enough in order to mimic the trapezoidal curve. So you do it as fast as you can. And deep, that's the big problem. Because the fact that tiny little girls, or men, for that matter, don't necessarily need 45 centimeters. Maybe they need 33.5. But we don't know. So we don't really know the ideal depth of chest compressions per individual. That's unknown. But what we know is that to teach the public, and teach ourselves, we need to have some figures to remember, or a distance to try for. So the answer to your question on depth is this: press deep enough so your colleague will feel a pulse in the groin or in the carotid artery. That's deep enough.

## Section 4: Of Men (Women) and Machines: Getting High Performance CPR with Mechanical Devices

By: Jerry Overton, MPA Richmond Ambulance Authority, Virginia Commonwealth University, Richmond, VA

#### INTRODUCTION

In the Richmond EMS, the technicians apply the automatic CPR device immediately upon reaching the patient's side, and continue the CPR all the way through to the end of the cardiac arrest. It is important for all healthcare workers to be aware of the positive results they have achieved.

#### PURPOSE/OVERALL GOAL

The purpose of this section of activity is to describe one EMS system's experience with automated CPR.

#### **OVERVIEW**

This section describes one EMS system's experience with automated CPR. The material is organized around the following categories:

- Introduction
- A High Performance System
- Response Statistics
- Placement of Autopulse®
- The Effect of Environment

#### SECTION OBJECTIVES

After completing this section of activity, the learner should be able to:

- Describe how the automated CPR machines are used in Richmond, VA.
- Describe the data on the use of automated CPR machines in Richmond, VA.

## Section 4: Content Review

#### INTRODUCTION

This section will discuss the experience with a mechanical CPR device in Richmond, Virginia. Figure 1 below is a benchmarking study that was done with other United States systems, Canadian systems, and European systems. Richmond is the single column to the left, and we have 1,450 emergency transports per 10,000 population—one of the busiest per capita systems in the United States.

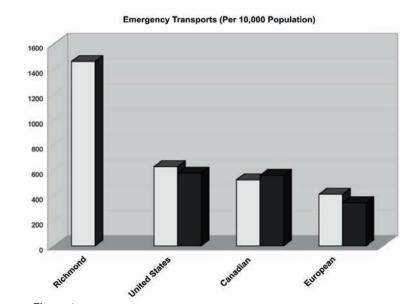


Figure 1

The patients that we're responding to are very sick. When you look at our cardiac arrest percentage, when you look at our chest pain percentage, and when you look at our dysrhythmia percentage, you'll see that these patients have high risk factors that are dependent upon the socio-economic factors that we have in Richmond. The town has a high age group, and we have a low socio-economic setting.

## A HIGH PERFORMANCE SYSTEM

The Richmond system is one of the very few high performance EMS systems in the United States. When we talk about high performance EMS, we mean both at the dispatch center and in the field. In the dispatch center, we prioritize our calls depending upon the patient condition, the patient assessment being taken by EMT dispatchers. We use dispatch life support. In other words, we're giving pre-arrival instructions, including instructions on over-the-phone CPR to our calling parties. We use a flexible deployment of resources, and we're an accredited International Academy of Emergency Dispatch call center.

In the field, we have mandated response times. We are required by city law to be on the scene of a life-threatening emergency in eight minutes or less, 90% of the time. As a result, we are all prepared for advanced life support, and every ambulance has every piece of equipment on it. So we began to look at our cardiac arrest analysis and response. We have to be performance-based in that also.

We're a data-driven system. When we look at analysis, we look at, obviously, witnessed versus un-witnessed cardiac arrest, who witnessed the arrest, whether it was our first responders or a bystander. We also look at it by the different ECGs, the different ways of presenting.

#### RESPONSE STATISTICS

Figure 2 below shows our analysis of number of arrests by hour of day.

# 2005 Arrests By Hour of Day

Responses Only

Distribution of Cardiac Arrest Responses By Hour Of Day

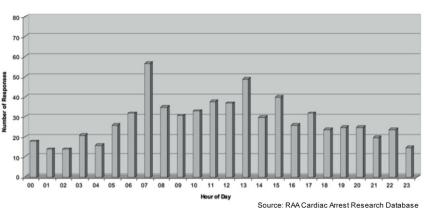


Figure 2

We do this, too, by day of week, which guides us in the deployment of our resources.

We use GIS mapping (shown in Figure 3).

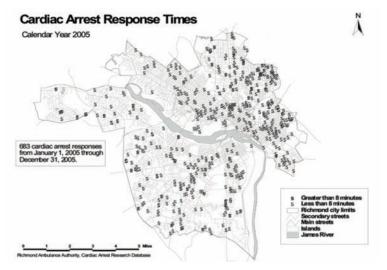


Figure 3

In Figures 4 and 5, you will see completely different dispersal patterns depending upon time of day. In Figure 4, you can see the time period from noon to 4 p.m. Note that there is a concentration of cardiac arrests in the core city at this time.

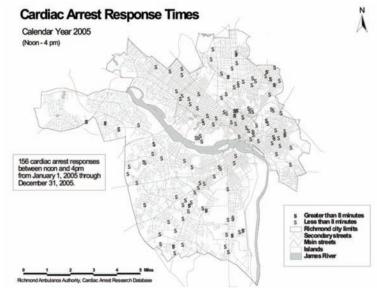


Figure 4

But if you then look at the time period from 4:00 p.m. to 8:00 p.m., you can see that more of the cardiac arrests are occurring outside the core city in the peripheral city.

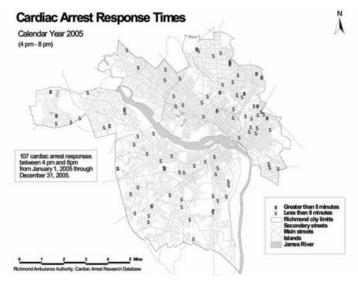


Figure 5

As a result, we use very sophisticated computer algorithms and system software to be able to re-deploy our ambulances at different times to be able to make that eight minute response time. We also use onboard computer systems because proper deployment of ambulances is much more effective than trying to speed the ambulances to a call, and have a wreck. This also bears on where we're placing our AutoPulses.

# AUTOPULSE MECHANICAL CPR

At Richmond Ambulance, we currently use the ZOLL AutoPulse. This device consists of a backboard and a chest band. The patient is placed on the board and the band is secured across the chest. The device then provides mechanical CPR continuously throughout the rescue, freeing the EMS team to attend to other aspects of the resuscitation.

Our history with the AutoPulse device dates back to April 2003, when we had three devices. We had some preliminary successes, and in December 2003, we increased that number to 11 devices so we were able to deploy an AutoPulse device to every cardiac arrest in Richmond. In November 2005, we increased that to 19 devices, so it became required equipment on every vehicle. A difference between 2003 and 2005 is that every vehicle had an AutoPulse by 2005. Previously we were deploying a field supervisor unit with an ambulance, and the field supervisor unit had the AutoPulse device—which meant there was a delay in getting mechanical CPR to some of our patients.

## TRAINING WITH AUTOPULSE

It's fairly easy to train on the AutoPulse device, but it's still using a mannequin. That's a very easy scenario. Anyone involved in pre-hospital care knows that this isn't the real world.

Figure 6 at right shows a typical scenario.

The patient is lying, in this instance, on his stomach. You can see in the next image that the shirt has been torn off immediately. The AutoPulse is quickly taken out and applied, and in fact, once the AutoPulse is applied to the patient, a button is pushed which takes literally a matter of seconds. The device is left on through CPR, and this is significant.

There's been considerable discussion about turning the device off when the patient becomes intubated. We do not turn the device off. We intubate right through the operation of the AutoPulse. Similarly, we do not turn the device off when we're defibrillating a patient, so that once the device is applied, it continuously provides automated CPR through every function that our pre-hospital care team is providing.









Figure 6

### AUTOPULSE AND THE RESCUE ENVIRONMENT

Not every cardiac arrest happens in a convenient parking lot. These are real environments that are not ideal, whether it's coming up the stairs into a parking lot or going down the stairs in some very narrow apartment buildings, or coming around corners. Richmond, Virginia is one of the oldest cites in the United States. We have some very challenging environments. What we were doing, of course, was having to do either manual CPR to bring the patients down or up in these settings, and afterward apply a mechanical CPR device, or we had to wait for the ambulance.

Now, with the AutoPulse, we apply the device immediately upon reaching the patient's side, and it continues the CPR all the way through to the end of the cardiac arrest. We can transport patients down steps and around corners while delivering CPR.

Another problem is the fact that people's bodies are not uniform. The fact of the matter is that we have an obesity situation in the United States. However, this has not been a factor in the application of mechanical CPR. We have had no patient who was too large for the AutoPulse.

## AUTOPULSE UTILIZATION AND OUTCOMES

In 2005, we used the AutoPulse on approximately 85% of our cardiac arrests in the field (Figure 7).

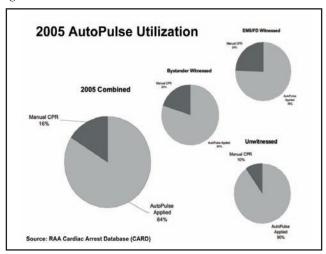


Figure 7

Figure 8 shows a summary of our 2005 results with the AutoPulse. Of 227 attempted resuscitations, the AutoPulse was used a total of 191 times (84%). One hundred twenty-eight (128) or 67% of the patients were transported for treatment. (We cease resuscitation in the field in Richmond). Of those transported, 55 of 128 achieved ROSC at some point, and 36 (38%) had ROSC upon arrival at the ED. Overall, ROSC at ED was achieved in 36 of 191 AutoPulse cases, or 18% of all uses. Of those transported, ROSC was present at arrival at ED in 27%, and 24% of EMS and bystander-witnessed arrests, respectively, and in 15.9% of unwitnessed arrests.

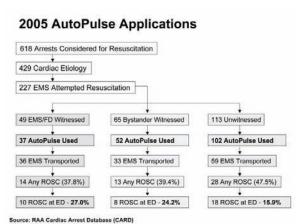
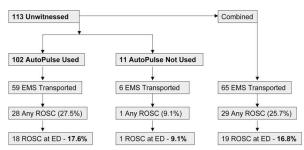


Figure 8

Figure 9 looks more closely at the unwitnessed arrest cohort, and compares outcomes between AutoPulse and manual CPR. Overall ROSC at ED was seen in 18 of 102 patients treated with AutoPulse (17.6%) versus 1 in 6 (9.1%) in the manual group. As expected, of those transported, the variance is similar, 30% in the AutoPulse group versus 16% in the manual CPR cohort.

# 2005 AutoPulse Applications



Source: RAA Cardiac Arrest Database (CARD)

Figure 9

# **CONCLUSION**

Now that we have the AutoPulse fully deployed throughout Richmond Ambulance, we are seeing statistically significant outcomes. The return of spontaneous circulation is exactly double that achieved when AutoPulse is not used.

NOTE: You may also complete this test and activity evaluation online at www.medcomrn.com/zoll. You will receive your certificate immediately upon successful completion of the test and evaluation.

Please mark answers on Test Answer Form.

Note: There is only one correct answer to multiple choice questions unless otherwise noted. A score of 80% or more is required to pass.

# Section 1: Go With the Flow: Blood Flow and its Importance in Improving Resuscitation (Wanchun Tang)

1.	The red cells continue supplying oxygen to the heart for aboutafter ventricular fibrillation.
	<ul> <li>a. 3-5 minutes</li> <li>b. 8-10 minutes</li> <li>c. no time at all</li> <li>d. At least 20 minutes</li> </ul>
2.	Immediate defibrillation is only effective during the firstof untreated VF.
	<ul><li>a. 30 minutes</li><li>b. 10 minutes</li><li>c. three to four minutes</li><li>d. 30 seconds</li></ul>
3.	During the last two decades, the incidence of VF as a first presenting rhythm has
	<ul><li>a. decreased</li><li>b. stayed the same</li><li>c. increased slightly</li><li>d. increased dramatically</li></ul>
4.	One way to continue good quality CPR is to  a. switch the rescuers b. use a metronome c. compress more shallowly d. slow the rate of compression
5.	Even with ideal conditions, the maximum blood flow you can generate with manual CPR is of cardiac output.  a. almost 90% b. about 10% c. about 20% d. about 35%

# Section 2: We Do Good CPR — Right? (Benjamin Abella)

6.	The pro studies	blem with the early studies of CPR quality in Arizona was that the
	a. b. c. d.	had too small a sample were subjective were objective were only on mannequins
7.	The AH	A standard for compressions is a minute.
	a. b. c. d.	50 75 100 120
8.	In the 2	005 AHA guidelines for CPR there's been a de-emphasis on
	a. b. c. d.	ventilations compressions compression rate clearing the airway
9.	One thi	ng that interferes with keeping compression rates up is
	a. b. c. d.	long pauses resilience of the chest patient resistance distractions
10.	As the p	pause before defibrillation increases
	a. b. c. d.	spontaneous recovery often occurs the chance of success increases the chance of a success markedly falls the mean rate of compressions goes up
		: Harder, Deeper, Faster: Manual CPR and cal CPR — Getting the Best from Both (Lars Wik)
11.		mber of compressions with correct depth increases with feedback, after we removed feedback.
	a. b. c. d.	increases again decreases stays the same stops
12.		est circumstances, compressions can be performed in a sinusoidal or waveform.
	a. b. c. d.	square oval trapezoidal flat

13.	The ide	al would be to generate a great aortic pressure with a low
	pressur	e.
	a.	right atrial
		fibrillation left atrial
	с. d.	right ventricular
14.	We nee	d a coronary profusion pressure of about millimeters of y in order to restart a heart.
	a. b. c. d.	15 12 30 100
15	***	
15.	epinepl	is weak, it takes a very long time before you see any effect from the nrine. Because we are giving epinephrine and the ion is too slow.
	a.	in the leg
	b.	in the chest
	С.	in the arm
	a.	orally
		: Of Men (Women) and Machines: Getting High
Pe	rforma	nce CPR with Mechanical Devices (Jerry Overton
Pe	<b>rform</b> a Experie	nce CPR with Mechanical Devices (Jerry Overton) nce shows that it is best to deploy AutoPulse devices
Pe	<b>rforma</b> Experie a.	nnce CPR with Mechanical Devices (Jerry Overton) once shows that it is best to deploy AutoPulse devices on each vehicle
Pe	<b>rforma</b> Experie a. b.	nce CPR with Mechanical Devices (Jerry Overton) once shows that it is best to deploy AutoPulse devices on each vehicle at random stations in the city
Pe	<b>rforma</b> Experie a. b.	nnce CPR with Mechanical Devices (Jerry Overton) once shows that it is best to deploy AutoPulse devices on each vehicle
Pe	Experie a. b. c. d.	nce CPR with Mechanical Devices (Jerry Overton) once shows that it is best to deploy AutoPulse devices on each vehicle at random stations in the city
<b>Pe</b> 16.	Experie a. b. c. d.	nce shows that it is best to deploy AutoPulse devices  on each vehicle at random stations in the city at hospitals in the EMT's home  is much more effective than trying to speed the nces to a call.
<b>Pe</b> 16.	Experie  a. b. c. d.  ambula	nce shows that it is best to deploy AutoPulse devices on each vehicle at random stations in the city at hospitals in the EMT's home is much more effective than trying to speed the nces to a call.  Proper deployment of ambulances Centralizing all the ambulances
<b>Pe</b> 16.	Experie a. b. c. d. ambula a. b. c.	ence shows that it is best to deploy AutoPulse devices  on each vehicle at random stations in the city at hospitals in the EMT's home  is much more effective than trying to speed the nces to a call.  Proper deployment of ambulances Centralizing all the ambulances Parking ambulances outside the city
<b>Pe</b> 16.	Experie a. b. c. d.  ambula a. b. c. d.	ence shows that it is best to deploy AutoPulse devices  on each vehicle at random stations in the city at hospitals in the EMT's home  is much more effective than trying to speed the nees to a call.  Proper deployment of ambulances Centralizing all the ambulances Parking ambulances outside the city Driving slowly and cautiously
<b>Pe</b> 16.	Experie a. b. c. d.  ambula a. b. c. d. In Rich	ence shows that it is best to deploy AutoPulse devices  on each vehicle at random stations in the city at hospitals in the EMT's home  is much more effective than trying to speed the nces to a call.  Proper deployment of ambulances Centralizing all the ambulances Parking ambulances outside the city
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<b>Pe</b> 16.	Experie  a. b. c. d.  ambula  a. b. c. d.  In Rich: defibril a.	ence shows that it is best to deploy AutoPulse devices  on each vehicle at random stations in the city at hospitals in the EMT's home  is much more effective than trying to speed the nces to a call.  Proper deployment of ambulances Centralizing all the ambulances Parking ambulances outside the city Driving slowly and cautiously mond, they the AutoPulse device when they're lating a patient. slow the rate of only turn off
<b>Pe</b> 16.	Experie  a. b. c. d.  ambula  a. b. c. d.  In Rich: defibril a.	ence shows that it is best to deploy AutoPulse devices  on each vehicle at random stations in the city at hospitals in the EMT's home  is much more effective than trying to speed the nces to a call.  Proper deployment of ambulances Centralizing all the ambulances Parking ambulances outside the city Driving slowly and cautiously mond, they the AutoPulse device when they're lating a patient.  slow the rate of only turn off do not turn off

- 19. The Autopulse is turned off:
  - a. During intubation
  - b. At the end of the code
  - c. When the ambulance is moving
  - d. When going down the stairs
- 20. The Richmond experience showed that Autopulse increased the number of patients in ROSC at arrival to the Emergency Department.
  - a. Not al all
  - b. 10%
  - c. 25%
  - d. 90%

# **Test Answer Form**

NOTE: You may also complete this test and activity evaluation online at www.medcomrn.com/zoll. You will receive your certificate immediately upon successful completion of the test and evaluation.

Name		Date _		
License #		SS#		
Address				
City	State		Zip	

Circle the correct answer							
1.	a	b	C	d			
2.	a	b	c	d			
3.	a	b	c	d			
4.	a	b	c	d			
5.	a	b	c	d			
6.	a	b	С	d			
7.	a	b	c	d			
8.	a	b	С	d			
9.	a	b	С	d			
10.	a	b	c	d			
11.	a	b	c	d			
12.	a	b	c	d			
13.	a	b	С	d			
14.	a	b	c	d			
15.	a	b	c	d			
16.	a	b	С	d			
17.	a	b	c	d			
18.	a	b	c	d			
19.	a	b	c	d			
20.	a	b	c	d			

# Continuing Medical Education Activity Evaluation Form (For Continuing Nursing Education credit please see the following page)

Name of Participant (Please Print)	Educatio	Date					
	Squeezing High of CPR C						
Please circle scores which best express your reactions to this activity							
Overall program organization     Level of information     Extent of new information, skills, knowledge     Usefulness of information in your practice     Extent to which you will change your current practices based on information from this program. (Please elaborate in the	Disorganized Poor Not At All No Use	1 2 3 4 5 1 2 3 4 5 1 2 3 4 5 1 2 3 4 5	Very Organized Excellent Great Extent Great Use				
General teaching effectiveness of the presenter     Extent to which this program fulfilled its stated educational objectives. Please rate each objec-	No Change Ineffective	1 2 3 4 5	Significant Change Very Effective				
tive individually:  At the conclusion of this 1 hour activity the participant will be able to:  Describe how perfusion and myocyte activity continue after ventricular fibrillation.	Not At All	1 2 3 4 5	Great Extent				
<ul> <li>Describe the importance of maintaining a high rate of chest compressions without interruptions.</li> </ul>	Not At All	1 2 3 4 5	Great Extent				
Describe the results of recent objective trials of CPR.	Not At All	1 2 3 4 5	Great Extent				
Describe the importance of maintaining a high rate of compressions.	Not At All	1 2 3 4 5	Great Extent				
Describe the importance of maintaining the proper depth in performing CPR.	Not At All	1 2 3 4 5	Great Extent				
<ul> <li>Describe the importance of maintaining a high rate of chest compressions in perform- ing CPR.</li> </ul>	Not At All	1 2 3 4 5	Great Extent				
<ul> <li>Describe the importance of minimizing pauses in performing CPR.</li> </ul>	Not At All	1 2 3 4 5	Great Extent				
Describe how the automated CPR machines are used in Richmond, Va.	Not At All	1 2 3 4 5	Great Extent				
Describe the data on the use of automated CPR machines in Richmond, Va.	Not At All	1 2 3 4 5	Great Extent				
Check Appropriate Box:							
☐ Staff M.D. ☐ Non Staff M.D. ☐ Other	☐ Resid	dent 🗆 Fe	llow				
Comments:							
Suggestions for Future Programs:							
Signature:							
*Form must be signed to receive CME credit.							

# Nursing Educational Activity Evaluation

# Please print and complete both sides of this form.

Name			Credentials					
(Last)	(FIRST)	(IV	1.I.)					
License #			Licensing State					
Home Address								
City			State					
Zip								
Full Facility Name	CATE	IERE COURSE WAS COMPLE	TED					
Facility Address								
racinty Address								
City			State	_ Zip				
Facility Phone #			Ext#	_				
Course #: <b>ZOLL100-CE</b> Contact Hour(s): 1	Course Title:	Squeezing High Per Compressions	formance Out of C	 CPR				
Time needed to complet	e total program:							
hours	_minutes	Date:						
Note: Times are to be show	Note: Times are to be shown in hours and 15 minute increments.							
Was this program offere	d through an inst	ructor or taken indep	endently as home s	study?				
☐ Through Instructor	☐ Indepe	endently						
	1	,						

I have reviewed the activity content and completed the Post Test. Please circle the response that best describes your ability to:

Describe how perfusion and myocyte activity	Strongly Agree				Strongly Disagree	Does Not Apply
continue after ventricular fibrillation.  • Describe the importance of maintaining a high rate of chest compressions without interrup-	1	2	3	4	5	0
tions.  • Describe the results of recent objective trials of	1	2	3	4	5	0
CPR.  • Describe the importance of maintaining a high	1	2	3	4	5	0
rate of compressions.	1	2	3	4	5	0
<ul> <li>Describe the importance of maintaining the proper depth in performing CPR.</li> <li>Describe the importance of maintaining a high</li> </ul>	1	2	3	4	5	0
<ul> <li>Describe the importance of maintaining a right rate of chest compressions in performing CPR.</li> <li>Describe the importance of minimizing pauses</li> </ul>	1	2	3	4	5	0
<ul> <li>Describe the importance of infinitizing pauses in performing CPR.</li> <li>Describe how the automated CPR machines are</li> </ul>	1	2	3	4	5	0
used in Richmond, VA.	1	2	3	4	5	0
<ul> <li>Describe the data on the use of automated CPR machines in Richmond, VA.</li> </ul>	1	2	3	4	5	0

Please give us your reactions to the program by circling the response that best describes your feelings:

	Strongl Agree	y			trongly risagree	Does Not Apply
1. Relationship of objectives to overall purpose/goal(s) of activity.	1	2	3	4	5	0
2. The material was effectively organized.	1	2	3	4	5	0
3. The language was clear, understandable, and at an appropriate level.	1	2	3	4	5	0
4. The program is an effective educational presentation.	1	2	3	4	5	0
5. The information included will benefit my practice.	1	2	3	4	5	0
6. The material presented was new to me.	1	2	3	4	5	0
7. This is a helpful and convenient way to earn	1	2	3	4	5	0
CE credit.	Exc	ıt			Poor	
8. Overall, I would rate this program:	1	2		3	4	5
9. (Optional) If you would like to contact us with a improving this program, suggestions for new please write them in the area below.						

Thank you for taking the time to complete this form.

To receive Continuing Education credit for the course, turn in your completed Test Answer Form and Educational Activity Evaluation to your Instructor. If you are taking the course on a home study basis through Medcom/Trainex, return the materials to: CE Coordinator, Medcom, Inc., 6060 Phyllis Drive, Cypress, CA 90630.

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