9-2009

# Methods for Calculating Coronary Perfusion Pressure During CPR

Michael P. Otlewski

Leslie A. Geddes

Michael Pargett

Charles F. Babbs
*Purdue University*, babbs@purdue.edu

# Methods for Calculating Coronary Perfusion Pressure During CPR

Michael P. Otlewski, Leslie A. Geddes, Michael Pargett, and Charles F. Babbs

Weldon School of Biomedical Engineering, Purdue University, West Lafayette, Indiana, 47907, USA.

**Abstract**

Coronary perfusion pressure (CPP) is a major indicator of the effectiveness of cardiopulmonary resuscitation in human and animal research studies; however methods for calculating CPP differ among research groups. Here we compare the 6 published methods for calculating CPP using the same data set of aortic (Ao) and right atrial (RA) blood pressures. CPP was computed using each of the 6 calculation methods in an anesthetized pig model, instrumented with catheters with Cobe pressure transducers. Aortic and right atrial pressures were recorded continuously during electrically induced ventricular fibrillation and standard CPR. CPP calculated from the same raw data set by the 6 calculation methods ranged from -1 (signifying retrograde blood flow) to 26 mmHg (mean ± SD of 15 ± 11 mmHg). The CPP achieved by standard closed chest CPR is typically reported as 10–20 mmHg. Within a single study the CPP values may be comparable; however, the CPP values for different studies may not be reliable indicators of the relative efficacies of different CPR methods. Electronically derived, true mean coronary perfusion pressure is arguably the gold standard metric for representing coronary perfusion pressure.

**Key words:** blood flow, blood pressure, cardiopulmonary resuscitation, ventricular fibrillation

Cardiovascular Engineering (2009) 9:98–103

Supported in part by National Institutes of Health (NIBIB Grant NGAR2IEBOO1540) and a grant from the Purdue Trask Fund.

## Introduction

Since Kouwenhoven, Jude, and Knickerbocker described external chest compressions to maintain circulation during ventricular fibrillation (VF) (Kouwenhoven et al. 1960), researchers have been working to refine this idea with more effective and efficient techniques. Investigators have tested various compression forces, rates, and duty cycles, investigated interposed chest and abdominal compressions, and proposed rhythmic abdominal compressions only (Maier et al. 1984; Criley et al. 1976; Halperin et al. 1993, 2004; Ralston et al.1982; Babbs 1993; Geddes et al. 2007), as well as adjunct devices to be applied during CPR to enhance its positive effects (Lottes et al. 2007; Cohen et al. 1992; Mauer et al. 1999; Lurie et al. 1990, 2001, 2002; Tang et al. 1997). To evaluate and compare the effectiveness of the various types of CPR compressions and devices, a metric must be chosen that will describe how well each method maintains coronary and systemic blood flow during cardiac arrest and CPR.

Most authorities agree that it is most vital that blood flow and oxygen delivery to the brain and the heart muscle itself be maximized during CPR (Kern 2000). It is difficult to measure coronary blood flow directly, using an electromagnetic flowmeter (Rubertsson et al. 1995; Geddes 1984) or radio-labeled microspheres (Voorhees et al. 1980; Halperin et al. 1986; Strohmenger et al. 1996). It is much simpler to measure the coronary perfusion pressure (CPP), which is the driving force for coronary blood flow, and CPP is directly proportional to the flow when coronary vascular resistance is constant. As a result, many researchers report the efficacy of CPR experiments (novel compression styles, adjunct devices, etc.) in terms of the CPP produced by each. CPP is defined as the difference between aortic and right atrial blood pressures (Kern 2000). Sample records of aortic and right atrial pressures with a normally beating heart and during VF with CPR are shown in Fig. 1. CPP with standard closed-chest CPR during VF is typically reported as 10–20 mmHg (Kern 2000).

Depending on the types of CPR, however, the aortic (Ao) and right atrial (RA) pressure waveforms can vary widely during the chest compression and decompression phases, but as yet there is no set rule for selecting the time points at which to measure the blood pressure difference. Accordingly, two researchers viewing the same Ao and RA pressure records from the same episode of VF with CPR may reach different conclusions about the value of that CPR. Therefore, the results of different studies may not be comparable, and multiple comparisons of different CPR techniques may not agree, depending on how CPP was measured.

The use of mean CPP may circumvent this problem as well as provide a more accurate depiction of coronary blood flow throughout CPR. The most general method for computing mean CPP computes the average difference between aortic and right atrial pressures over the entire compression-decompression cycle (Geddes et al. 2007). This value is the true, electronically derived mean perfusion pressure. It is equivalent to the area between the Ao and RA pressure curves, integrated over one cycle, and divided by the duration of the cycle. Since it is a measure of coronary perfusion pressure throughout compression and decompression, this measure accounts for both antegrade and retrograde blood flow (typically during the compression phase), and it eliminates the need for arbitrary or idiosyncratic time points for CPP calculation.

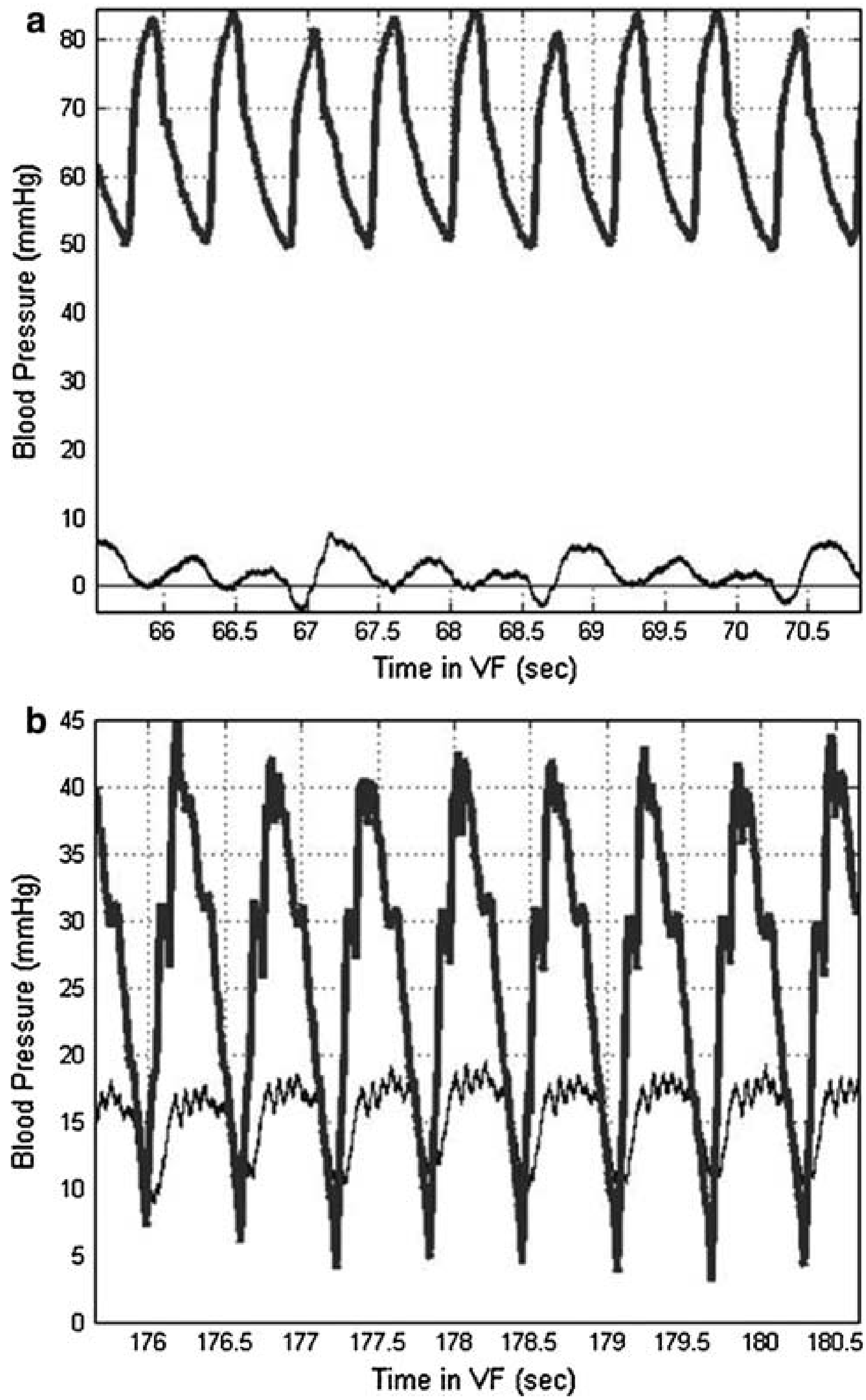


Fig. 1 In a are shown aortic (thick line) and right atrial (thin line) blood pressures with the normally beating heart. In b are shown aortic (thick line) and right atrial (thin line) blood pressures with standard CPR during VF. Note the difference in vertical scales.

## Methods

All experimental protocols were approved by the Purdue Animal Care and Use Committee and are in accordance with National Institutes of Health guidelines for ethical animal research. Domestic pigs were intubated and anesthetized to surgical depth. 100% oxygen was given with isoflurane anesthetic. Lead II ECG was recorded. Blood pressure was recorded using Cobe pressure transducers via catheters inserted into the right atrium and the thoracic aorta. ECG and right atrial and aortic pressures were recorded on a Physiograph strip-chart recorder (Narco Inc) and recorded digitally with Labview (1,000 data samples/sec for each channel). A bipolar pacing electrode was advanced into the right ventricle for inducing ventricular fibrillation. A catheter was inserted into the left femoral vein for the collection of blood samples and the administration of drugs.

Ventricular fibrillation was induced by electrical stimulation of the right ventricular (RV) myocardium using the Physiograph stimulator connected to the RV bipolar electrode. Fibrillation was confirmed by the characteristic ECG waveform and concurrent loss of pulsatile blood pressure. After ventricular fibrillation was confirmed, the pneumatic chest Thumper® (Michigan Instruments, Grand Rapids, MI) was activated to compress the chest and set at 100 lbs force, 100/min compression rate, and 50% duty cycle. After 30 chest compressions, 2 breaths at 60 $cmH_2O$, 100% oxygen were administered. Compressions were then resumed. This cycle of 30 compressions and 2 breaths was completed twice and then defibrillation was attempted with transchest electrodes. A damped sine wave defibrillation countershock of 100–150 J was administered by a defibrillator (Hewlett- Packard). If defibrillation was not successful, CPR was resumed and a higher energy dose was given. After successful defibrillation was achieved, rhythmic ECG and corresponding blood pressure were restored.

For the normally beating heart and the episode of VF with CPR, CPP was calculated from the digital blood pressure records using 6 different methods reported in the literature and described by the blood pressure recordings in Figs. 2, 3, 4, 5, 6, and 7. The thicker waves represent aortic blood pressure during CPR, the thinner waves, right atrial blood pressure. The vertical lines indicate the time at which the pressure measurements were recorded. The shaded areas represent the segments of time over which the areas between the curves were calculated. Areas marked with an X show retrograde blood flow. The data set used for the calculations in this study was previously published in Geddes et al. 2007. For each method, the CPP results were calculated for 3 consecutive blood pressure cycles and averaged.

Each of the six calculation methods was performed as follows:

1. ***Peak diastolic:*** right atrial blood pressure was subtracted from time-coincident aortic blood pressure at the point of lowest aortic pressure during diastole (decompression).

2. ***Mid-diastolic:*** right atrial blood pressure was subtracted from time-coincident aortic blood pressure at the midpoint of the diastolic (decompression) interval.

3. ***End-diastolic:*** right atrial blood pressure was subtracted from time-coincident aortic blood pressure at the point just before the beginning of the rise in pressure due to the subsequent compression.

4. ***Peak systolic:*** right atrial blood pressure was subtracted from time-coincident aortic blood pressure at the point of highest aortic pressure during systole (compression).

5. ***Diastolic mean:*** the area between the aortic and right atrial blood pressure curves during the diastolic interval (decompression) was calculated and divided by the time length of the interval.

6. ***CPI or true mean:*** CPI is the area between the aortic and right atrial blood pressure curves throughout the entire blood pressure cycle (compression and decompression), integrated over one minute. For the sake of comparison with the other methods of calculation, the same three blood pressure cycles were integrated, rather than using an entire minute's worth of cycles. True mean is CPI/60 to remove the time element and allow for direct comparison to other CPP values.

The authors had full access to the data and take responsibility for its integrity. All authors have read and agree to the manuscript as written.

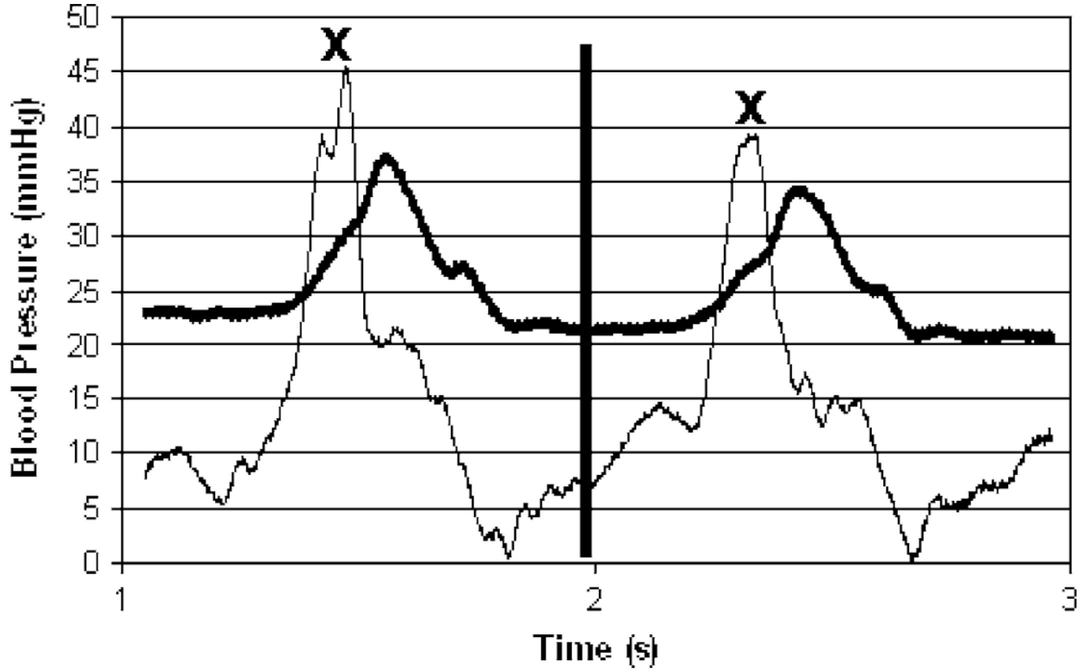


Fig. 2 The peak diastolic method, as described by Niemann et al. 1985 and Fries et al. 2006

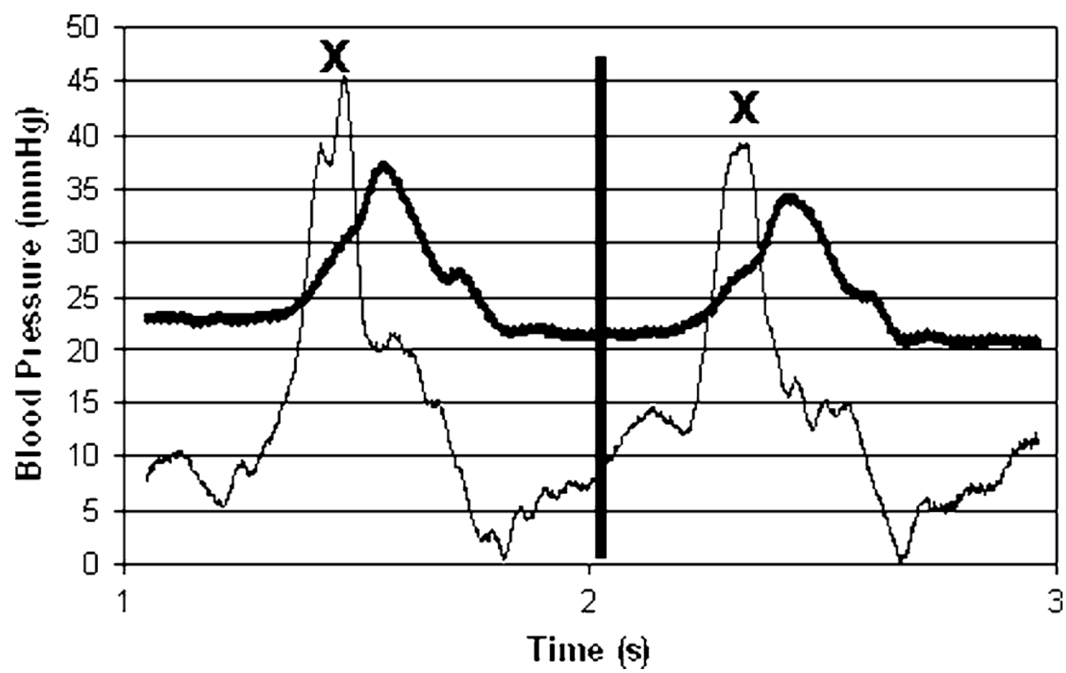


Fig. 3 The mid-diastolic method, as described by Niemann et al. 1982, Sanders et al. 1985, and Tang et al. 1997

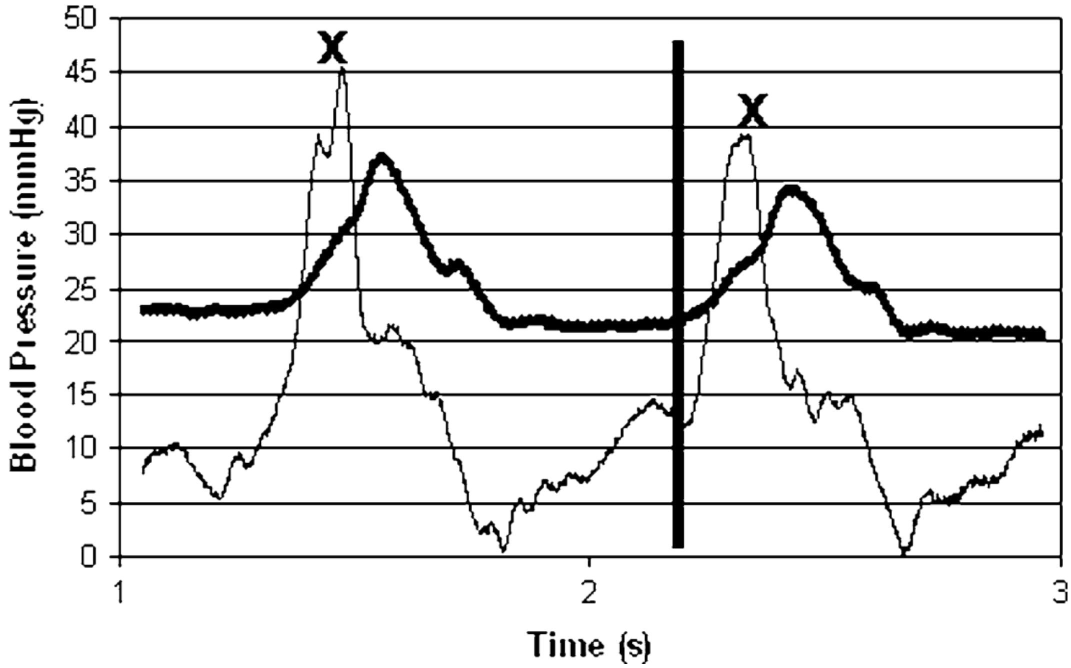


Fig. 4 The end-diastolic method, as described by Paradis et al. 1990 and Cairns and Niemann 1998

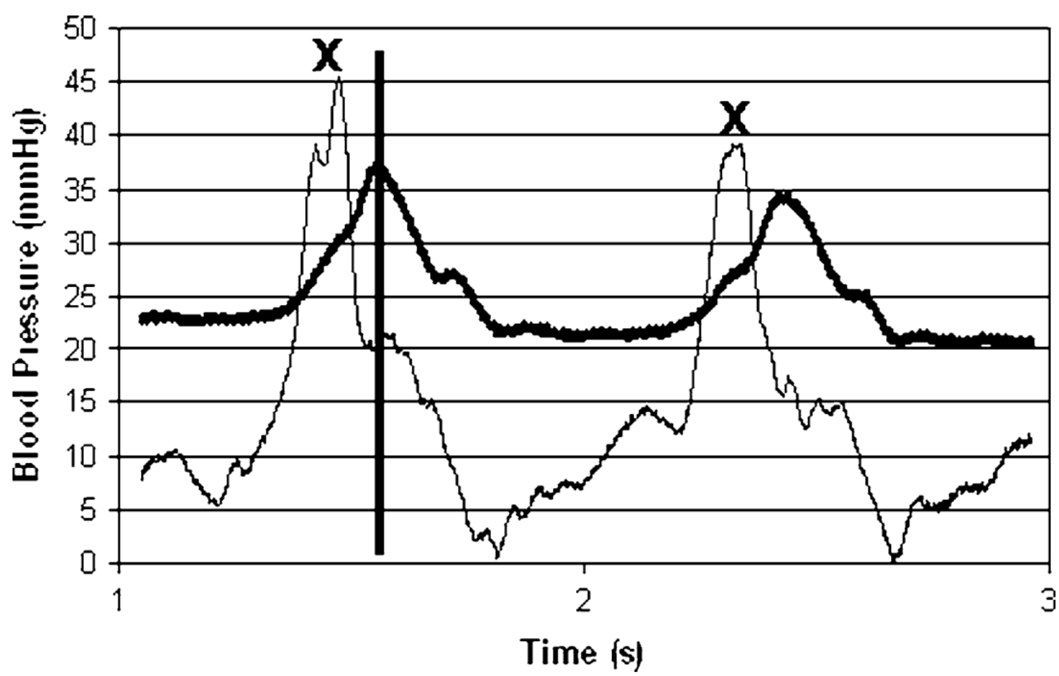


Fig. 5 The peak systolic method, as described by Niemann et al. 1982, 1985, and Raessler et al. 1988

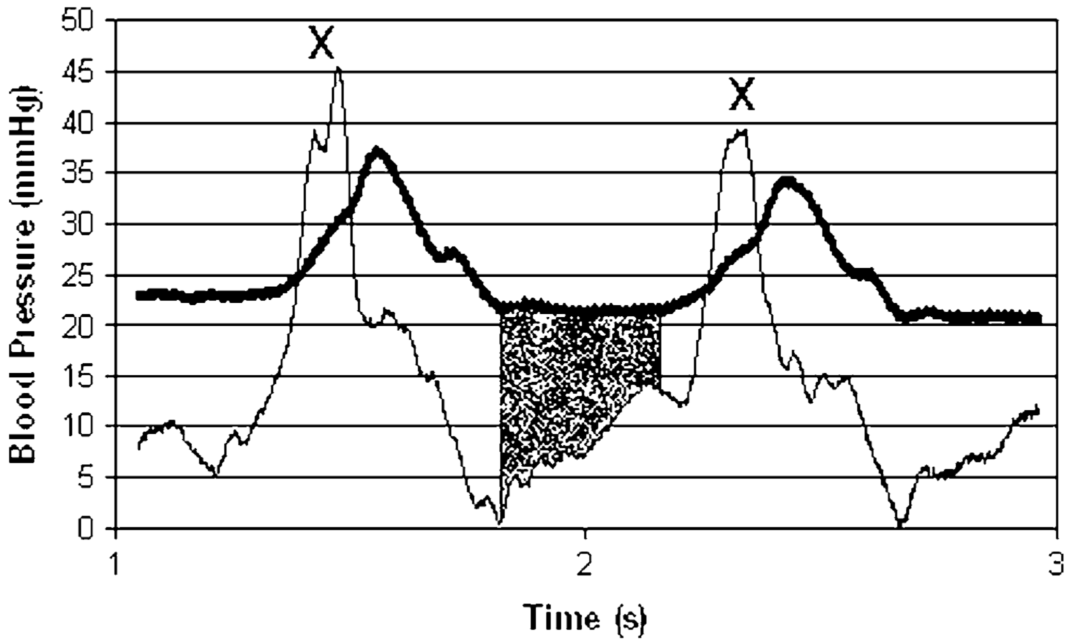


Fig. 6 The diastolic mean method, as described by Fenely et al. 1988, Cohen et al. 1992, and Wik et al. 1996

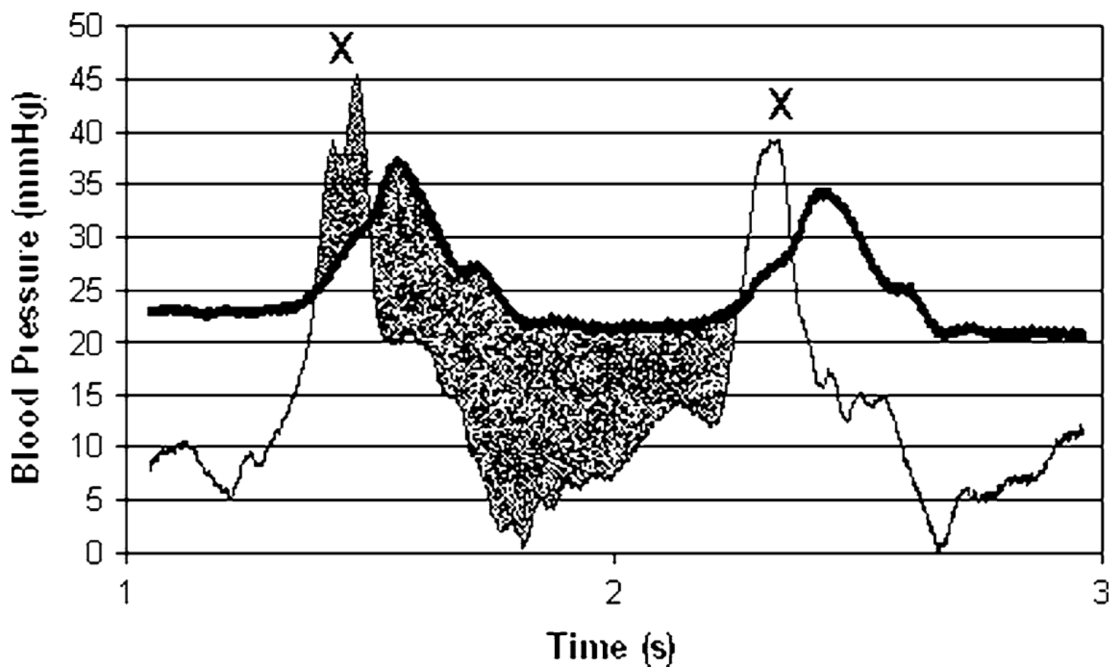


Fig. 7 The true mean method, as described by Geddes et al. 2007, Niemann et al. 1985, Babbs 2006, and Jung et al. 2006

## Results

Table 1 displays the CPP for the normally beating heart immediately before the episode of VF and the CPP provided by standard CPR (performed according to the 2005 AHA guidelines) during VF, as calculated by each of the CPP methods.

**Table 1** Coronary perfusion pressure as provided by the normally beating heart, standard CPR with VF

| CPP-Calculating method | CPP with beating heart (mmHg) | CPP with CPR (mmHg) |
|---|---|---|
| Peak diastolic | 55 | 26 |
| Mid-diastolic | 55 | 18 |
| End-diastolic | 55 | 9 |
| Peak systolic | 80 | −1 |
| Mean diastolic | 55 | 25 |
| True mean | 69 | 11 |
| Average ± SD | 61 ± 11 | 15 ± 11 |

**Table 2** True mean pressure compared with the CPPs of each of the other methods of CPP calculation

| Method | CPP | True mean | Ratio:CPP/(true mean) |
|---|---|---|---|
| Peak diastolic | 26 | 11 | 2.4 |
| Mid-diastolic | 18 | 11 | 1.6 |
| End-diastolic | 9 | 11 | 0.8 |
| Peak systolic | −1 | 11 | −0.1 |
| Mean diastolic | 25 | 11 | 2.3 |

For the beating heart control values, peak systolic measurement and true mean disagreed with all of the diastolic methods and with each other. None of the diastolic beating heart measurements from the same time intervals (individual measurements, prior to averaging) differed more than 2 mmHg. CPP for standard CPR ranged from -1 to 26 mmHg with the peak systolic and peak diastolic calculation methods, respectively. The standard deviation of the CPP results was 73% of the average.

Table 2 compares the ratio of calculated CPP to true mean CPP for each calculation method. Using a technique similar to the CPI method of Geddes et al. 2007, Niemann et al. 1985 recorded CPP by continuously subtracting right atrial from aortic pressure throughout the entire CPR cycle. Applying the method of Niemann et al. (1985) to this dataset, and then averaging the resulting CPP over the time interval (similar to the true mean method) provides a CPP of 11 mmHg.

## Discussion

Because the pig's normally beating heart rate was greater than 100 beats/min, the intervals between beats (the diastolic segments) were short. With a short segment, pressures do not change much when measuring from the beginning, middle, or end of diastole. Therefore, it is not surprising that the CPP methods measured during diastole did not disagree with each other. Systolic aortic pressure is usually much higher than diastolic (a difference of about 25 mmHg), while right atrial systolic pressure does not differ much from diastolic (about 5 mmHg). So, it is also not surprising that measuring CPP at the peak of systole provides a significantly different pressure than all of the diastolic measurement methods. The true mean method considers perfusion during systole and diastole; if the difference in pressures is great enough, it makes sense that true mean disagrees with both systolic and diastolic methods, because its results lie somewhere in between.

Coronary perfusion is known to occur during diastole, because mechanical pressure collapses the coronary vessels during systole. Since peak systolic and true mean methods consider pressures at times other than diastole, they should overestimate the true CPP of the normally beating heart. The diastolic measurement methods, which all agree very closely, should provide more trustworthy estimates of CPP during the normally beating heart.

For standard CPR, the methods of calculating CPP yielded differences of up to 27 mmHg. Considering CPP during standard CPR is usually between 10–20 mmHg, this is a considerable disagreement among the different methods. The peak diastolic method produced a CPP almost 3 times that of the end-diastolic, more than twice that of the true mean method, and almost half again greater than that of the mid-diastolic method. The peak systolic method returned a negative CPP value which, unlike all other methods, indicates net retrograde blood flow.

## Conclusion

Different methods of calculating coronary perfusion pressure (CPP) during cardiopulmonary resuscitation (CPR) with ventricular fibrillation (VF) produce considerably different results. The results are highly dependent on the blood pressure waveforms produced by CPR. When those methods are used to compare the efficacies of different types of CPR or CPR techniques, differences may be found or missed, depending on which CPP-calculating method is employed. To limit the variability in results due to the variability of the waveform, it seems that methods which take a mean of the pressure difference would produce a more reliable result. Since perfusion could occur at times other than decompression, logically, the true mean method should be the most reliable index of coronary flow (in the absence of direct flow measurement). This supposition cannot be confirmed by this study, and further exploration with a flowmeter could determine which method of calculating CPP is most strongly correlated with coronary perfusion.

Overall, these disagreements show that because of the use of different methods of computing CPP, values published in different studies cannot readily be compared across studies.

## References


Babbs CF. Interposed Abdominal Compression-CPR: a case study in cardiac arrest research. Ann Emerg Med. 1993;22:24-32.

Babbs CF. Design of near-optimal waveforms for chest and abdominal compression and decompression in CPR using a computer-simulated evolution. Resuscitation. 2006;68:277–93.

Cairns CB, Niemann JT. Hemodynamic effects of repeated doses of epinephrine after prolonged cardiac arrest and CPR: preliminary observations in an animal model. Resuscitation. 1998;36:181–5.

Cohen TJ, Tucker KJ, Redberg RF, Lurie KG, Chin MC, Dutton JP, et al. Active compression-decompression resuscitation: a novel method of cardiopulmonary resuscitation. Am Heart J. 1992;124:1145–50.

Criley JM, Blaufuss AH, Kissel GL. Cough-induced cardiac compression: self-administered form of cardiopulmonary resuscitation. JAMA. 1976;236:1246–50.

Fenely MP, Maier GW, Kern KB, Gaynor JW, Gall SA, Sanders AB, et al. Influence of compression rate on initial success of resuscitation and 24 h survival after prolonged manual cardiopulmonary resuscitation in dogs. Circulation. 1988;77:240–50.

Fries M, Tang W, Chang YT, Wang J, Castillo C, Weil MH. Microvascular blood flow during cardiopulmonary resuscitation is predictive of outcome. Resuscitation. 2006;71:248–53.

Geddes LA. Cardiovascular devices and their applications. New York: Wiley; 1984.

Geddes LA, Rundell A, Lottes A, Kemeny A, Otlewski M. A new cardiopulmonary resuscitation method employing only rhythmic abdominal compression. a preliminary report. Am J Emerg Med. 2007;25:786–90.

Halperin HR, Paradis N, Ornato JP, Zviman M, Lacorte J, Lardo A, et al. Cardiopulmonary resuscitation with a novel chest compression device in a porcine model of cardiac arrest: improved hemodynamics and mechanisms. J Am Coll Cardiol. 2004;44:2214–20.

Halperin HR, Tsitlik JE, Gelfand M, Weisfeldt ML, Gruben KG, Levin HR, et al. A preliminary study of cardiopulmonary resuscitation by circumferential compression of the chest with use of a pneumatic vest. N Engl J Med. 1993;329:762–8.

Halperin HR, Tsitlik JE, Guerci AD, Mellits ED, Levin HR, Shi AY, et al. Determinants of blood flow to vital organs during cardiopulmonary resuscitation in dogs. Circulation. 1986;73: 539–50.

Jung E, Babbs CF, Lenhart S, Protopopscu VA. Optimal strategy for cardiopulmonary resuscitation with continuous chest compression. Acad Emerg Med. 2006;13:715–21.

Kern KB. Coronary perfusion pressure during cardiopulmonary resuscitation. Bailliere's Clin Anesth. 2000;14:591–609.

Kouwenhoven WB, Jude JR, Knickerbocker GG. Closed-chest cardiac massage. JAMA. 1960;173:1064–7.

Lottes AE, Rundell AE, Geddes LA, Kemeny AE, Otlewski MP, Babbs CF. Sustained abdominal compression in standard CPR improves coronary perfusion pressure comparably to vasopressor drugs. Resuscitation. 2007;75:515–24.

Lurie KG, Lindo C, Chin J. CPR: the P stands for plumber's helper. JAMA. 1990;264:1661.

Lurie KG, Voelckel WG, Zielinski T, McKnite S, Lindstrom P, Peterson C, et al. Improving standard cardiopulmonary resuscitation with an inspiratory impedance threshold valve in a porcine model of cardiac arrest. Anesth Analg. 2001;93:649–55.

Lurie KG, Zielinski T, McKnite S, Aufderheide T, Voelckel W. Use of an inspiratory impedance valve improves neurologically intact survival in a porcine model of ventricular fibrillation. Circulation. 2002;105:124–9.

Maier GW, Tyson GS, Olsen CO, Kernstein KH, Davis JW, Conn EH, et al. The physiology of external cardiac massage: high-impulse cardiopulmonary resuscitation. Circulation. 1984;70: 86–101.

Mauer DK, Nolan J, Plaisance P, Sitter H, Benoit H, Stiell IG, et al. Effect of active compression-decompression resuscitation (ACD-CPR) on survival: a combined analysis using individual patient data. Resuscitation. 1999;41:249–56.

Niemann JT, Criley JM, Rosborough JP, Niskanen RA, Alferness C. Predictive indices of successful cardiac resuscitation after prolonged arrest and experimental cardiopulmonary resuscitation. Ann Emerg Med. 1985;14:521–8.

Niemann JT, Rosborough JP, Ung S, Criley JM. Coronary perfusion pressure during experimental cardiopulmonary resuscitation. Ann Emerg Med. 1982;11:127–31.

Paradis NA, Martin GB, Rivers EP, Goetting MG, Appleton TJ, Feingold M, et al. Coronary perfusion pressure and the return of spontaneous circulation in human cardiopulmonary resuscitation. JAMA. 1990;263:1106–13.

Raessler KL, Kern KB, Sanders AB, Tacker WA Jr, Ewy GA. Aortic and right atrial systolic pressures during cardiopulmonary resuscitation: a potential indicator of the mechanism of blood flow. Am Heart J. 1988;115:1021–9.

Ralston SH, Babbs CF, Niebauer MJ. Cardiopulmonary resuscitation with interposed abdominal compression in dogs. Anesth Analg. 1982;61:645–51.

Rubertsson S, Grenvik A, Zemgulis V, Wiklund L. Systemic perfusion pressure and blood flow before and after administration of epinephrine during experimental cardiopulmonary resuscitation. Crit Care Med. 1995;23:1984–96.

Sanders AB, Ogle M, Ewy GA. Coronary perfusion pressure during cardiopulmonary resuscitation. Am J Emerg Med. 1985;3:11–4.

Strohmenger HU, Lindner KH, Keller A, Lindner IM, Pfenninger E, Bothner U. Effects of graded doses of vasopressin on median fibrillation frequency in a porcine model of cardiopulmonary resuscitation: results of a prospective, randomized, controlled trial. Crit Care Med. 1996;24:1360–5.

Tang W, Weil MH, Schock RB, Sato Y, Jucas J, Sun S, et al. Phased chest and abdominal compression-decompression: a new option for cardiopulmonary resuscitation. Circulation. 1997;95:1335–40.

Voorhees WD, Babbs CF, Tacker WA. Regional blood flow during cardiopulmonary resuscitation in dogs. Crit Care Med. 1980; 8:134–6.

Wik L, Bircher NG, Safar P. A comparison of prolonged manual and mechanical external chest compression after cardiac arrest in dogs. Resuscitation. 1996;32:241–50.