CPR-Dependent Consciousness: Evidence for Cardiac Compression Causing Forward Flow

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We present the case of a patient with cardiac arrest and resuscitation in whom, consistent with direct cardiac compression, large aortic-to-right atrial systolic pressure gradients occurred. Forward blood flow during CPR was sufficient for the patient to maintain consciousness. Although aortic-to-right atrial diastolic gradients adequate to maintain coronary perfusion in experimental models were generated, in our patient, cardiac function could not be restored and the resuscitation was ultimately unsuccessful. [Lewinter JR, Carden DL, Nowak RM, Enriquez E, Martin GB: CPR-dependent consciousness: Evidence for cardiac compression causing forward flow. Ann Emerg Med October 1989;18:1111-1115.]

INTRODUCTION

The mechanism of blood flow during closed-chest CPR has been the subject of controversy since the technique was introduced in 1960. Direct cardiac compression, providing selective increases in ventricular pressures, was initially believed to be the mechanism of forward flow (cardiac pump mechanism).[1] However, early investigations with invasive monitoring during CPR revealed equivalent increases in all vascular pressures.[2] It was shown that cardiac output could be generated by a pressure gradient between the intrathoracic and extrathoracic compartments (thoracic pump mechanism).[3] Current CPR research is defining the relative contributions of each mechanism with the aim of refining CPR techniques to optimize coronary and cerebral blood flow.

We present a case of a patient with cardiac arrest and resuscitation in whom large aortic (Ao)-to-right atrial (RA) pressure gradients were recorded during mechanical CPR. The patient maintained consciousness only when CPR was in progress. The Ao-RA systolic pressure gradients suggest that the cardiac pump mechanism was operative in this case and was responsible for sufficient forward flow to maintain consciousness.

CASE REPORT

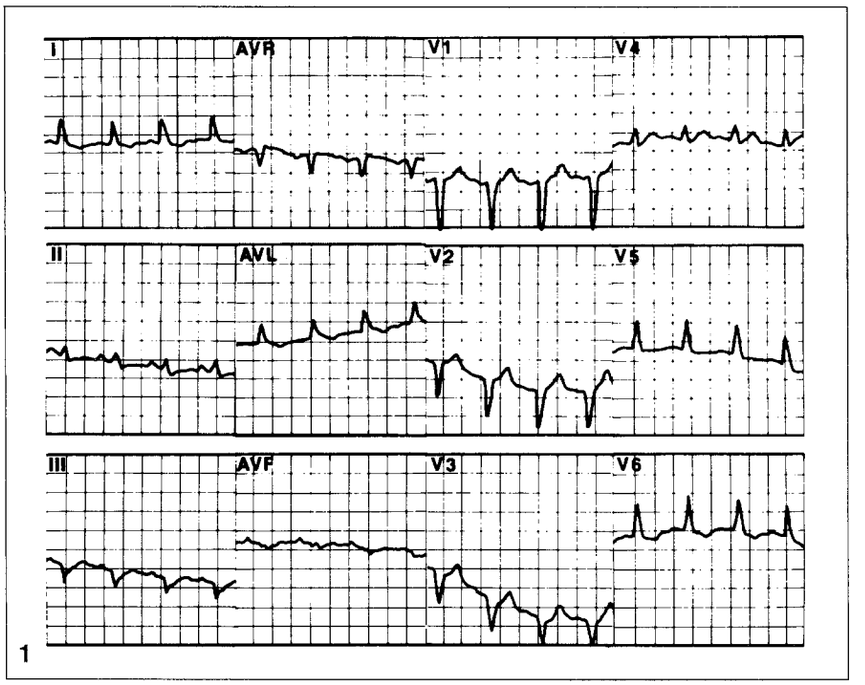
A 60-year-old woman presented to the emergency department with a three-hour history of retrosternal chest pain unrelieved by 12 sublingual nitroglycerin tablets. The pain radiated to both shoulders and was associated with marked shortness of breath and vomiting. Medical history was significant for a myocardial infarction two years before presentation. Current medications included topical nitrates, oral nifedipine, and sublingual nitrates as needed.

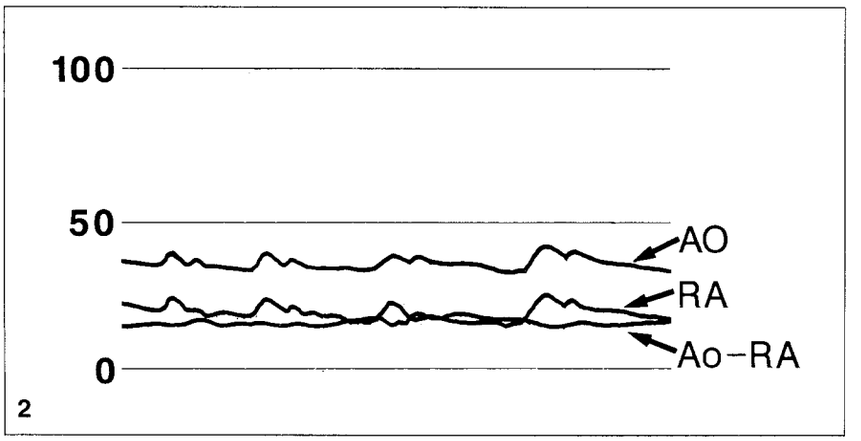
On admission, the patient's blood pressure was 71/55 mm Hg; pulse, 84; respirations, 40; and rectal temperature, 37.0 C. The patient was a thin, profusely diaphoretic woman who was alert but anxious. The cardiopulmonary examination revealed a thready, regular radial pulse, jugular venous distention to the angle of the mandible at 90 degrees, and rales to the midscapulae. Cardiac sounds were obscured by the pulmonary findings; however, no gross murmurs or rubs were appreciated. There was no peripheral edema.

The patient was placed on a cardiac monitor. A peripheral IV catheter was inserted, and a 5% dextrose in water infusion was begun. Oxygen was administered through a nonrebreather mask at 15 L/min, and a dopamine infusion at 5 μg/kg/min was started. An ECG was obtained (Figure 1), and

FIGURE 1. *Twelve-lead ECG obtained on admission to the ED.*

FIGURE 2. *Ao, RA, and Ao−RA pressure tracings, without CPR, obtained shortly after invasive monitoring was initiated.*





an upright portable chest radiograph revealed cardiomegaly and pulmonary edema. Initial laboratory values revealed potassium of 3.4 mmol/L (3.4 mEq/L); carbon dioxide, 11 mmol/L (11 mEq/L); hemoglobin, 114 g/L (11.4 g/dL); creatine kinase, 73 U/L; and lactate dehydrogenase, 131 U/L. Arterial blood gas values were pH, 7.23; oxygen pressure, 46 mm Hg; carbon dioxide pressure, 32 mm Hg; and bicarbonate ion, 13 mmol/L (13 mEq/L).

The patient's blood pressure increased transiently to 120/107 mm Hg in response to the dopamine infusion. Despite continuous increases in the rate of dopamine infusion and subsequent 50-mL fluid challenges to support any decreases in intravascular volume, the patient failed to maintain adequate arterial pressures and became increasingly lethargic. Ventricular ectopy developed 30 minutes after arrival and was not suppressed by an IV lidocaine bolus of 100 mg and a continuous infusion of 4 mg/min.

Thirty-three minutes after arrival at the ED, pulseless ventricular tachycardia developed. Immediate countershock with 300 J resulted in a wide complex pulseless rhythm. The patient was intubated, and mechanical ventilation and compression were initiated (Thumper®, Michigan Instruments, Grand Rapids, Michigan). Ventilatory pressure was 40 cm H_2O, and approximately 80 lb of force was used to compress the sternum 2 in.

In accordance with a protocol previously approved by the Human Rights Committee of Henry Ford Hospital, an arterial catheter (70 cm, 5.8F) was placed percutaneously in the right femoral artery by the Seldinger technique and advanced to the thoracic aorta distal to the subclavian artery. A central venous catheter (double lumen, 25 cm, 7F) was placed in the right subclavian vein and advanced to the right atrium. The catheters were connected to Gould Statham P-50 transducers, which were set to stretcher height and zeroed to atmosphere. Simultaneous pressure tracings were recorded (Figures 2 and 3) with Hewlett-Packard 78205D amplifiers and a Hewlett-Packard 7758 multichannel recorder (Hewlett-Packard, Sunnyvale, California). Continuous Ao, RA, and Ao−RA pressures were available throughout the resuscitation. Simultaneous arterial and central venous blood gases were obtained (Table 1).

When mechanical CPR was instituted, the patient became responsive and required repetitive small doses of IV morphine sulfate and diazepam for sedation. Whenever CPR was discontinued, the patient became unresponsive. Despite maximal pharmacologic intervention, it was not possible to maintain adequate systemic arterial blood pressure without mechanical chest compression. Ao and RA pressure tracings demonstrated deterioration over time (Table 2). Two hours and ten minutes after arrival at the ED, an Ao balloon pump was placed in an unsuccessful endeavor to stabilize the patient's cardiac status.

It was the consensus of the emergency physicians, cardiologists, and cardiothoracic surgeons that partial cardiopulmonary bypass, cardiac catheterization, or prolonged maintenance on mechanical CPR were not

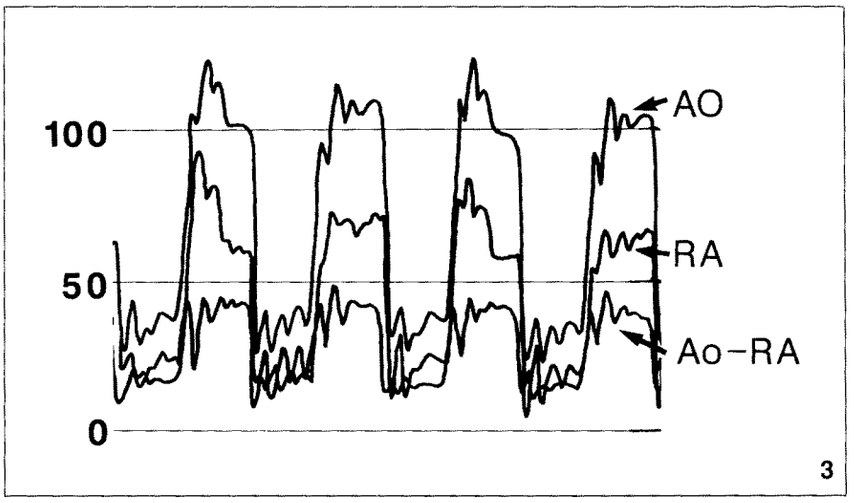


FIGURE 3. *Ao, RA, and Ao–RA pressure tracings, with CPR, obtained shortly after invasive monitoring was initiated.*

TABLE 1. *Simultaneous arterial and central venous blood gas results*

	Time From Initiation of Resuscitation			
	One-Half Hour		One and One-Half Hours	
	Arterial	Central Venous	Arterial	Central Venous
Partial pressure of oxygen (mm Hg)	205	32	108	28
Oxygen saturation (%)	99	47	96	32
Partial pressure of carbon dioxide (mm Hg)	20	33	25	40
pH	7.27	7.19	7.15	7.09

feasible. Three hours and fifteen minutes after presentation, resuscitative efforts were terminated and, in accordance with family wishes, an autopsy was not performed.

DISCUSSION

Kuowenhoven et al,[1] in their original description of the technique of closed-chest CPR, proposed that direct compression of the heart between the sternum and vertebral spine was responsible for blood flow during cardiac massage. Weale and Rothwell-Jackson questioned this theory after demonstrating equivalent elevations in arterial and venous pressures during external CPR and proposed that global increases in intrathoracic pressures and not cardiac compression were responsible for flow.[2] Thus, the mechanism of blood flow in closed-chest CPR has been subject to controversy since the introduction of the technique in 1960.

The thoracic pump theory of blood flow during CPR has been refined since the observations of Weale and Rothwell-Jackson. Rudikoff et al[3] demonstrated that an intrathoracic-to-extrathoracic pressure gradient was responsible for forward flow. Jugular venous valves were documented by cineangiography to close during artificial systole, preventing the retrograde transmission of flow to the extrathoracic veins.[4] Criley et al demonstrated that arterial pressures could be generated and consciousness could be maintained without chest compressions if patients in ventricular fibrillation were instructed to cough vigorously.[5] Further support for the thoracic pump theory was offered by echocardiographic studies that demonstrated no decrease in left ventricular size or closure of the mitral valve during chest compressions.[6,7]

The cardiac pump theory of blood flow assumes that preferential increases in ventricular pressures occur during chest compression. The cardiac pump mechanism is supported by cineangiograms that show direct ventricular compression during CPR.[8] Babbs et al, in a comparison of large and small dogs during CPR, demonstrated that techniques that increase intrathoracic pressures improve cardiac output in large dogs but have no significant effect in small dogs.[9] This suggests that the geometry of the chest may determine the relative contributions of the cardiac or thoracic pump to systemic blood flow. However, a recent study in human beings that demonstrated a significant role for the thoracic pump mechanism concluded that thoracic geometry and body weight did not correlate with Ao or coronary perfusion pressures during CPR.[10]

In experimental models, coronary perfusion pressure, as reflected by the diastolic Ao-RA pressure gradient, strongly correlates with coronary blood flow during CPR.[11] Successful resuscitation from cardiac arrest in animals has been shown to correlate with the maintenance of Ao diastolic pressures of more than 30 mm Hg[12-14] or with coronary perfusion pressures of more than 15 mm Hg.[15,16] Maier et al[17] demonstrated coronary blood flow 65% to 75% of baseline values during high-impulse manual compressions, while simultaneously recording significant differences between intracardiac and intrathoracic pressures.

Sanders et al[10,18-21] found that increasing intrathoracic pressures with simultaneous compression-ventilation CPR and abdominal binding was detrimental to Ao diastolic pressure and coronary perfusion pressure and that, compared with standard CPR, animals treated with these modified techniques could not be resuscitated.[13] Studies in human beings have failed to demonstrate adequate coronary perfusion pressures with conventional CPR or improve-

TABLE 2. *Aortic and right atrial pressures*

	Time From Initiation of Resuscitation					
	One-Half Hour		One and One-Half Hours		Two Hours	
	+ CPR	− CPR	+ CPR	− CPR	+ CPR	− CPR
Ao systolic pressure (mm Hg)	122	40	100	38	100	35
RA systolic pressure (mm Hg)	48	36	40	32	40	30
Ao−RA systolic gradient (mm Hg)	74	4	60	6	60	5
Ao diastolic pressure (mm Hg)	36	36	35	32	38	35
RA diastolic pressure (mm Hg)	15	17	12	17	15	20
Ao−RA diastolic gradient (mm Hg)	21	19	23	15	23	15

Ao, aortic; RA, right atrial; Ao−RA, aortic minus right atrial; +CPR, with CPR; −CPR, without CPR.
Systolic pressures were measured at peak systole, and diastolic pressures were measured at end diastole.

ments with modified CPR techniques.

Recently, venous-to-arterial carbon dioxide gradients have been identified as an indicator of the inadequacy of forward blood flow during cardiac arrest and resuscitation. Selective venous hypercarbia is believed to be associated with minimal forward blood flow, and successful resuscitation is associated with a precipitous decrease in the venous carbon dioxide tension and the venous-to-arterial carbon dioxide differences.[22-24]

In our patient, who had a small thoracic cage, cardiomegaly, and wide-complex pulseless rhythm, mechanical CPR produced large systolic Ao-RA pressure gradients, suggesting that direct cardiac compression occurred. The forward blood flow produced was sufficient to maintain consciousness. The low venous-to-arterial carbon dioxide gradients and normal central venous carbon dioxide pressures corroborate the adequacy of forward blood flow. Diastolic Ao-RA gradients of 15 to 23 mm Hg were achieved but proved inadequate to restore myocardial function in our patient. This was probably because of extensive myocardial infarction resulting in pump failure and/or fixed coronary obstructions preventing coronary flow in spite of these Ao-RA gradients.

SUMMARY

Progress in improving resuscitation from cardiac arrest is limited by the inability to evaluate the effectiveness of CPR. Arteriovenous pressure measurements and venoarterial carbon dioxide gradients provide physicians with measures of the adequacy of resuscitative efforts. If progress is monitored and found to be inadequate early in the resuscitation effort, alternative therapeutic maneuvers such as internal cardiac massage or cardiopulmonary bypass may be considered.

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REFERENCES

1. Kuowenhoven WB, Jude JR, Knickerbocker GG: Closed chest cardiac massage. *JAMA* 1960;173:1064-1067.

2. Weale FE, Rothwell-Jackson RL: The efficiency of cardiac massage. *Lancet* 1962;1: 990-992.

3. Rudikoff MT, Maughan WL, Effran M, et al: Mechanisms of blood flow during cardiopulmonary resuscitation. *Circulation* 1980;61:345-352.

4. Niemann JT, Rosborough J, Hausknecht M, et al: Blood flow without cardiac compression during chest CPR. *Crit Care Med* 1981;9: 380-381.

5. Criley JM, Blaufuss AH, Kissel GL: Cough-induced cardiac compression: Self-administered form of cardiopulmonary resuscitation. *JAMA* 1976:1246-1250.

6. Werner JA, Greene HL, Janko CL, et al: Visualization of cardiac valve motion in man during external chest compression using two-dimensional echocardiography: Implications regarding the mechanism of flow. *Circulation* 1981;63:1417-1421.

7. Rich S, Wix HL, Shapiro EP: Clinical assessment of heart chamber size and valve motion during cardiopulmonary resuscitation by two-dimensional echocardiography. *Am Heart J* 1981;102:368-373.

8. Babbs CF: Cardiac angiography during CPR. *Crit Care Med* 1980;8:189.

9. Babbs CF, Tacker WA, Paris RL, et al: CPR with simultaneous compression and ventilation at high airway pressure in four animal models. *Crit Care Med* 1982;10:501-504.

10. Swenson RD, Weaver WD, Niskanan RA, et al: Hemodynamics in humans during conventional and experimental methods of cardiopulmonary resuscitation. *Circulation* 1988;78: 630-639.

11. Ditchey RV, Winkler JV, Rhodes CA: Relative lack of coronary blood flow during closed-chest resuscitation in dogs. *Circulation* 1982;66: 297-302.

12. Redding JS: Abdominal compression in cardiopulmonary resuscitation. *Anesth Analg* 1971;50:668-675.

13. Sanders AB, Ewy GA, Alferness CA, et al: Failure of one method of simultaneous chest compression, ventilation and abdominal binding during CPR. *Crit Care Med* 1982;10:509-513.

14. Sanders AB, Ewy GA, Taft TV: Prognostic and therapeutic importance of the aortic diastolic pressure in resuscitation from cardiac arrest. *Crit Care Med* 1984;12:871-873.

15. Ralston SH, Voorhees WD, Babbs CF: Intrapulmonary epinephrine during prolonged cardiopulmonary resuscitation: Improved regional blood flow and resuscitation in dogs. *Ann Emerg Med* 1984;13:79-86.

16. Niemann JT, Criley JM, Rosborough J, et al: Predictive indices of successful cardiac resuscitation after prolonged arrest and experimental cardiopulmonary resuscitation. *Ann Emerg Med* 1985;14:521-528.

17. Maier GW, Tyson GS, Olsen CO, et al: The

physiology of external cardiac massage: High-impulse cardiopulmonary resuscitation. *Circulation* 1984;70:86-101.

18. Sanders AB, Ogle M, Ewy GA: Coronary perfusion pressure during cardiopulmonary resuscitation. *Am J Emerg Med* 1985;3:11-14.

19. McDonald JL: Effect of interposed abdominal compression during CPR on central arterial and venous pressures. *Am J Emerg Med* 1985; 3:156-159.

20. Martin GB, Carden DL, Nowak RM, et al: Aortic and right atrial pressures during standard and simultaneous compression and ventilation CPR in human beings. *Ann Emerg Med* 1986;15:125-130.

21. Howard M, Carrubba C, Foss F, et al: Interposed abdominal compression-CPR: Its effects on parameters of coronary perfusion in human subjects. *Ann Emerg Med* 1987;16:253-259.

22. McGill JW, Ruiz E: Central venous pH as a predictor of arterial pH in prolonged cardiac arrest. *Ann Emerg Med* 1984[Part I];13:684-687.

23. Weil MH, Rackow EC, Trevino R, et al: Difference in acid-base state between venous and arterial blood during cardiopulmonary resuscitation. *N Engl J Med* 1986;315:153-156.

24. Nowak RM, Martin GB, Carden DL, et al: Selective venous hypercarbia during human CPR: Implications regarding blood flow. *Ann Emerg Med* 1987;16:527-530.