Virginia Commonwealth University
VCU Scholars Compass

Theses and Dissertations | Graduate School

1996

A New Device for Performing Simultaneous Sterno-Thoracic Cardiopulmonary Resuscitation (SST-CPR)

Deepak S. Gupta

Follow this and additional works at: https://scholarscompass.vcu.edu/etd

 Part of the Biomedical Engineering and Bioengineering Commons

© The Author

Downloaded from
https://scholarscompass.vcu.edu/etd/4692

This Thesis is brought to you for free and open access by the Graduate School at VCU Scholars Compass. It has been accepted for inclusion in Theses and Dissertations by an authorized administrator of VCU Scholars Compass. For more information, please contact libcompass@vcu.edu.

Virginia Commonwealth University
School of Engineering

This is to certify that the thesis/dissertation prepared by Deepak S. Gupta entitled **"A New Device for Performing Simultaneous Sterno-Thoracic Cardiopulmonary Resuscitation (SST-CPR)"** has been approved by his committee as satisfactory completion of the thesis requirement for the degree of Master of Science.

[redacted]

Joseph P. Ornato M.D., Director of Thesis

[redacted]

Martin Lenhardt Ph.D., School of Engineering

[redacted]

Edgar R. Gonzalez Pharm.D., Department of Pharmacy and Pharmaceutics

[redacted]

Gerald E. Miller Ph.D., Department Chairman

[redacted]

Henry McGee Ph.D,, Dean, School of Engineering

[redacted]

Jack L. Haar, Ph.D., Dean, School of Graduate Studies

August 15, 1996

Date

©Deepak S. Gupta 1996
All Rights Reserved

A New Device for Performing Simultaneous Sterno-Thoracic Cardiopulmonary Resuscitation (SST-CPR)

A Thesis submitted in partial fulfillment of the requirements for the degree of Master of Science at Virginia Commonwealth University.

By

Deepak S. Gupta
B.S. Mechanical Engineering
Virginia Polytechnic Institute and State University
(Virginia Tech)

Director: Joseph P. Ornato, M.D.
Affiliated Professor of
Biomedical Engineering

Virginia Commonwealth University
Richmond, Virginia
August 1996

Acknowledgments

I would like to say a special thanks to Dr Hwang and Dr Ornato for their guidance and understanding in helping me to excel in my academic career. I would also like to thank Dr Gonzalez and Dr Lenhardt for serving on my research committee. Finally, I would like to thank my parents and my brother. I would have not gotten this far without them. Thank you!!

Table of Contents

Page

List of Figures v

List of Tables vii

List of Abbreviations viii

Abstract ix

Introduction 1

Literature Review 5
Mechanism of Blood Flow During Standard External Chest Compression (ECC) 5
CPR Devices And New Techniques to Augment Blood Flow During CPR 9
Devices 9
Automatic Resuscitator 9
Cardiac Press 10
Active Compression-Decompression Device 10
Weightlessness Device 12
Vest Device 12
Resuscitation Apparatus 13
New Techniques to Augment Blood Flow During CPR 14
Simultaneous Compression And Ventilation (SCV-CPR) 14
High-Impulse External Chest Compression (HI-CPR) 15
Interposed Abdominal Compression (IAC-CPR) 16

Method 17
Simultaneous Stemo-Thoracic (SST)-CPR 17
Device 17
General Description of The Device 17
Detail Description of The Device 20

Mechanical Study....30

Pilot Animal Study....32
Study Design....32
Preparation of the animals....32
Cardiopulmonary resuscitation(CPR)....34
Measurement....35
After study....35

Results....36
Mechanical Study....36
Pilot Animal Study....36
Aortic pressure....37
Coronary blood flow....37
Intrathoracic pressure....37

Interpretation....41
Limitations....44

Summary And Conclusion....46

References....47

Appendix
Dimensions of the SST-CPR Device....53

Vita....58

List of Figures

Page

1. "Cardiac Pump" Mechanism....6

2. "Thoracic Pump" Mechanism....6

3. Automatic Resuscitator....9

4. Active Compression-Decompression Device....11

5. Weightlessness Device....12

6. Vest Device....13

7. Resuscitation Apparatus....14

8. Interposed Abdominal Compression (IAC-CPR)....16

9. Simultaneous Stemo-Thoracic (SST)-CPR Device....18

10. Cross-sectional View (SST-CPR Device)....19

11. Systolic and Diastolic phase (SST-CPR Device)....21

12. Straps (SST-CPR Device)....23

13. Piston (SST-CPR Device)....25

14. Base-Supporting Structure (SST-CPR Device)....26

15. Table (SST-CPR Device)....28

16. Backboard (SST-CPR Device)....29

17. Plastic Barrel for Mechanical Testing 31

18. Aortic Pressure 38

19. Coronary Blood Flow 39

20. Intrathoracic Pressure.....40

21. Dimensions of the Base-Supporting Structure.....54

22. Cross-Sectional View of the Base-Supporting Structure 55

23. Base-Supporting Structure in Pig Device.....56

24. Dimensions of the Piston in the Base-Supporting Structure 57

25. Cross-Sectional View of the Backboard for the Device 58

List of Tables

Page

1. Size And Material 22

2. Experimental Procedure.....34

List of Abbreviations

* ACD - Active Compression Decompression
* CAD - Computer Aided Design
* CPR - Cardiopulmonary Resuscitation
* ECC - External Chest Compression
* HI - High Impulse
* IAC - Interposed Abdominal Compression
* IM - Intramuscular
* MI - Michigan
* MS - Microsoft
* NASA - National Aeronautics and Space Administration
* SCV - Simultaneous Compression and Ventilation
* SST - Simultaneous Stemo-Thoracic
* TEE - Transesophageal Echocardiography
* 3d - 3 Dimensional

A NEW DEVICE FOR PERFORMING SIMULTANEOUS STERNO-THORACIC CARDIOPULMONARY RESUSCITATION (SST-CPR)

ABSTRACT

A thesis submitted in partial fulfillment of the requirement for the degree of Master of Science at Virginia Commonwealth University.

Deepak S. Gupta, M.S.

Virginia Commonwealth University, 1996.

Major Director: Joseph P. Ornato, M.D.

Executive Summary

There are two different physiological mechanisms that cause blood to circulate around the human body during cardiopulmonary resuscitation. The "cardiac pump" generates blood flow by squeezing blood out of the heart as the sternum is depressed. The "thoracic pump" generates flow by forcing blood out of the heart and the great vessels as the intrathoracic pressure rises due to chest compression.

To date, all CPR techniques try to circulate blood during cardiac arrest by exploiting either the cardiac pump or the thoracic pump mechanism of blood flow. No mechanical CPR device thus far invented has tried to exploit both mechanisms at the same time. We hypothesize that a combination of the cardiac and thoracic pump mechanisms of blood flow should generate more blood flow than either alone.

We have thus invented a device that performs simultaneous sterno-thoracic cardiopulmonary resuscitation (SST-CPR).

Our SST-CPR device augments blood flow to the vital organs by performing cardiac and thoracic compression simultaneously using two components. A piston provides direct sternal compression, squeezing blood out of the heart directly ("cardiac pump"). A thoracic strap and back supporting structure create circumferential thoracic constriction ("thoracic pump"). Simultaneous compression and constriction are performed by pushing the compressing piston, which directly compresses the heart and increases intrathoracic pressure by constricting the thorax. Mechanical tests have been performed. Tests have also been performed to measure hemodynamic parameters in vivo. The device has been designed to allow variation in the relative contribution of either the cardiac or thoracic pump during CPR, thus allowing better understand of the relative importance of each mechanism during CPR.

INTRODUCTION

Deepak Gupta*, Sung-Oh Hwang, MD~, Edgar R. Gonzalez, Pharm D~,
Martin Lenhardt, PhD*, George W. Maier, MD^, Joseph P. Ornato, MD~*

Program of Biomedical Engineering*, Department of Surgery^,
and Department of Emergency Medicine~
Virginia Commonwealth University - Medical College of Virginia

In 1960, Kouwenhoven et al. {{1}} proposed precordial compression as a method of artificial circulation in the cardiac arrest patient. External cardiac compression rapidly became the standard technique {{2}} for rescuers to provide artificial circulation during cardiac arrest. Unfortunately, standard closed-chest cardiopulmonary resuscitation (CPR) generates only 15-30% of the normal cardiac output. This severe reduction in the blood flow to vital organs during CPR can harshly limit the likelihood that a patient will be resuscitated successfully. Coronary perfusion pressure above 20 mmHg is required to restore spontaneous circulation {{3}}, and carotid blood flow above 20% of cardiac output is needed to prevent the brain from developing cellular necrosis. Failure of the current CPR technique to maintain adequate vital organ perfusion has motivated many investigators to explore other promising new CPR techniques. However, most CPR techniques have not been shown to demonstrate increased survival in cardiac arrest patients.

There are two theoretical mechanisms of blood flow during CPR. The "cardiac pump" {{4,5}} generates blood flow by directly compressing the heart between the sternum and the vertebrae. At the time closed chest CPR as a method of artificial circulation was suggested, Kouwenhoven et al. {{1}} were credited with the idea that the heart, which is the largest organ between the sternum and the vertebrae, is "squeezed" by precordial compression. Closure of the mitral valve during "compression systole" {{6}} and deformation of the ventricular dimension according to the direction of the applied force {{7}} suggests that the heart acts as a pump during precordial compression.

In the 1970s, the cardiac pump theory was challenged by investigators who observed that increased intrathoracic pressure alone (without precordial compression) can generate blood flow. Criley et al {{8}} reported that increases in the intrathoracic pressure by repeated coughing could maintain adequate systemic blood pressure without precordial compression in a patient with ventricular fibrillation during cardiac catheterization. Halperin et al. {{9}} also demonstrated that fluctuation of the intrathoracic pressure using a pneumatic vest device could generate blood flow in dogs with cardiac arrest, thus proposing the "thoracic pump" as an alternate means for generating blood flow during cardiac arrest.

With the thoracic pump mechanism, pressures in the thorax are increased by air trapping in the alveoli and small bronchioles during chest

compression. Increase of intrathoracic pressure by chest compression creates a pressure gradient between the intrathoracic and extrathoracic cavities, which can generate forward blood flow {{10}}. In this context, the heart does not behave as a pump per se, but serves as a passive conduit for blood flow {{11,12}}.

Recently, transesophageal echocardiography (TEE) has been used to investigate CPR physiology{{6,13}}. TEE can provide quality images of the cardiac structures and shows the direction of blood flow during CPR in humans using Doppler imaging. Even though mitral valve closure has previously been considered to be evidence for left ventricular compression during precordial compression, TEE studies demonstrate that mitral valve closure during compression systole should not be considered as evidence of cardiac compression anymore{{14,15}} because the thoracic pump can also close the mitral valve. Thus TEE studies cannot resolve whether the dominant mechanism of blood flow during CPR in humans is the "cardiac" vs. "thoracic" pump.

While controversies about the mechanism of blood flow during CPR have continued, new CPR methods to augment blood flow have been proposed. For example, Halperin et al {{16}} invented "thoracic vest" CPR based on the thoracic pump mechanism. Maier et al {{17,18}} proposed "high impulse CPR" based on the cardiac pump mechanism. However, no

investigator has previously tried a CPR method using both mechanism simultaneously.

We hypothesize that combination of both the cardiac and thoracic pump mechanisms of blood flow during CPR simultaneously should generate better blood flow during CPR than either technique alone. To examine our hypothesis, we designed and built a new, manually powered, simple, inexpensive device that is designed to take advantage of both physiological mechanisms of blood flow during CPR by performing sternal and circumferential thoracic compression at the same time. We have tested the prototype mechanically, and we have also conducted an experiment in an animal model.

LITERATURE REVIEW

MECHANISM OF BLOOD FLOW DURING STANDARD EXTERNAL CHEST COMPRESSION(ECC)

A primary goal of CPR is to maintain and restore tissue perfusion to as normal a level as possible during resuscitation. Blood flow generated by current standard CPR technique is not enough to maintain viability of vital organs. This limitation of current CPR technique is largely responsible for the universally low resuscitation rates seen both in and out of the hospital. To increase blood flow to vital organs during cardiac arrest, many investigators have tried to develop better CPR techniques. To develop new CPR technique, it is essential to understand the mechanism of blood flow by external chest compression.

Even though the current technique of external chest compression remains relatively simple, the mechanism of blood flow remains controversial. Two different theories have been proposed to explain why blood flow is generated during external chest compression: the "cardiac pump" and "thoracic pump".

The heart acts as a mechanical pump in the "cardiac pump" mechanism. (Figure 1) During compression systole, both ventricles are squeezed between

the sternum and the spine. The aortic and pulmonic valve open by the increased ventricular pressure when the atrioventricular valves are closed, and then forward blood flow occurs.{{1}} During compression diastole, the heart expands and refills by it's own elasticity and blood enters the chambers. The mode of coronary flow and cerebral flow are similar to that of intact circulation. {{19}}

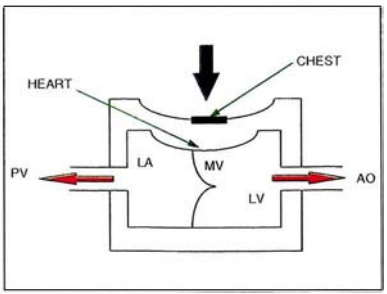

Figure 1. "Cardiac Pump" Mechanism

According to the "thoracic pump" mechanism, (Figure 2) increases in the intrathoracic pressure during external chest compression generates blood flow from the intrathoracic to the extrathoracic regions {{9}}{{15}}. Closure of the jugular vein valve at the thoracic outlet and the larger capacity of veins relative to arteries also facilitates generation of a pressure gradient between the intrathoracic and extrathoracic cavities {{20}}{{21}}{{22}}. During the relaxation phase of chest compression, venous return occurs because venous pressure is relatively higher than intrathoracic pressure. Some blood

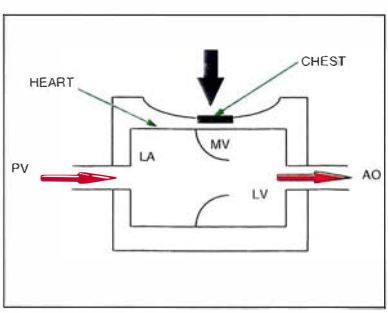

Figure 2. "Thoracic Pump" Mechanism

flows retrograde from extrathoracic to intrathoracic arteries perfusing the coronary arteries {{23}}{{24}}{{25}}. Therefore, the thorax itself behaves as a pump; the heart behaves as only a passive conduit in the "thoracic pump" mechanism. {{19}}

Controversy concerning the relative contribution of either mechanism of blood flow during CPR still remains unresolved. Several investigators have tried to delineate the major mechanism of blood flow during precordial compression. Using chronically instrumented dogs, Maier et al demonstrated that the magnitude of blood flow generated by external chest compression is determined by the compression rate, but not by the duration of compression. They interpreted these finding to indicate that direct cardiac compression is the cause of flow because stroke volume should be determined by the amount of cardiac deformation and prolonged compression after ejection will have no effect on stroke volume. On the other hand, Halperin et al demonstrated that prolongation of the compression duration increased blood flow during external chest compression in discrete species of animal model. They insisted that flow will be relatively constant due to decrease in the size of the vessels leaving the thorax by increased intrathoracic pressure, and it should be dependent on the duration of compression per cycle rather than the rate of compression.

Most CPR studies have been performed in animals. It is difficult to adapt the results of animal experiments to humans directly because animals have a

different chest shape and configuration compared to those in humans. Echocardiographic behavior of the mitral valve is not consistent with a pure cardiac or thoracic pump mechanism. During compression systole, some investigators report mitral valve closure while others observe persistent mitral valve opening. Mitral valve behavior changes over time during CPR in the same patient as time passes. These observations indicate that both mechanisms of blood flow might be operative during CPR in human being.

CPR Devices and New Techniques to Augment Blood Flow during CPR

DEVICES

Many CPR devices and techniques have been introduced since it was realized that standard CPR cannot generate adequate blood flow to vital organs. Advantages of using devices during CPR are improvement in blood flow to vital organs, standardization of CPR technique, and reduction in rescuer fatigue. Some of these devices are the Automatic Resuscitator, the Cardiac Press, the Active Compression-Decompression Device, Vest CPR, Resuscitation Apparatus, and the Weightlessness Device.

AUTOMATIC RESUSCITATOR

The most widely used device for performing CPR is the Thumper^{R} (Michigan Instruments, Grand Rapids, MI) or Automatic Resuscitator. This device can provide mechanical external chest compression or ventilations during CPR. The device is designed as a plunger mounted on a backboard. It is

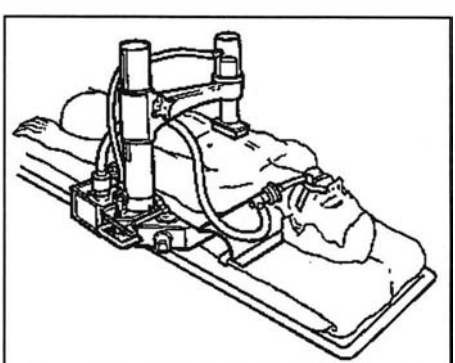

Figure 3. Automatic Resuscitator

powered by 100% compressed oxygen. (Figure 3) {{26}} A time pressure cycled ventilation is delivered every five compressions using a 50% cycle duration length. The plunger's depth of compression on the sternum can be adjusted by the rescuer from 1.5 to 2 inches (3.9 to 5 cm).{{26}}

The main disadvantage of Thumper^{R} is its costs. The advantages are its size, lightweight, and consistent performance of CPR for prolonged periods of time, making it particularly valuable during transport. Because the Thumper^{R} does standard CPR, the technique itself does not improve blood flow or survival compare to standard CPR. {{26}}

CARDIAC PRESS

The Cardiac Press is a simple, inexpensive device. The device has a hinged arm that makes chest compression easier. The downstroke of the device can be controlled to compress the sternum between 1.5 and 2 inches. The device can be applied very quickly. It is also lightweight, easy to assemble, and very compact for storage. The two disadvantages of this device are that the compressor head often changes position, and certain components of the device can become loose resulting in inadequate chest compression. Therefore, the device always needs monitoring to make sure compressions are adequate and the compressor head is centered. {{26}}

ACTIVE COMPRESSION-DECOMPRESSION DEVICE

Active Compression-Decompression CPR (ACD-CPR) is similar to

Standard CPR, but it also provides negative intrathoracic pressure during the relaxation phase. ACD CPR is performed with a device called the Ambu Cardio-Pump™ (Ambu Inc, Denmark). This device (Figure 4) consists of three important parts: "(1) a neoprene suction cup, (2) a plastic circular handle with an undercut hand grip, and (3) a force gauge." {{27}} The gauge can be calibrated to a fixed depth, which usually is similar to standard CPR (1.5 to 2 inches). To ensure proper function by the operator, the decompression force is also measured up to -30 lb by the gauge. The device is positioned at midsternum level in alignment with the nipples. {{27}}

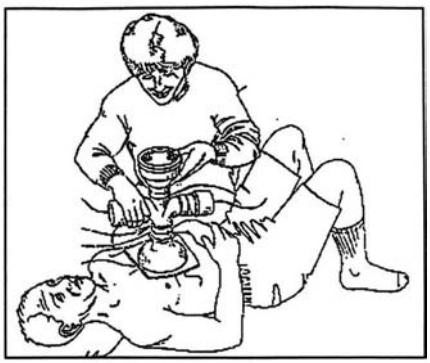

Figure 4. ACD-CPR

The mechanisms of ACD-CPR is not completely understood. Some believe that ACD-CPR might work by a mechanism similar to the thoracic pump. During active compression, air is fills the chest, causing it to increase the intrathoracic pressure. Negative intrathoracic pressure is produced by active decompression causing greater venous return.

This device is still being tested clinically, but preliminary results suggest no dramatic increase in blood flow. Further tests are needed.

WEIGHTLESSNESS DEVICE

NASA engineers invented this device to perform CPR in space. (Figure 5) The device is a belt which secures around the thorax, and a compression pad which go up and down on top of the sternum. The functions of this device are similar to Standard CPR. Force is applied only to the sternum. This device has not yet been tested clinically. {{28}}

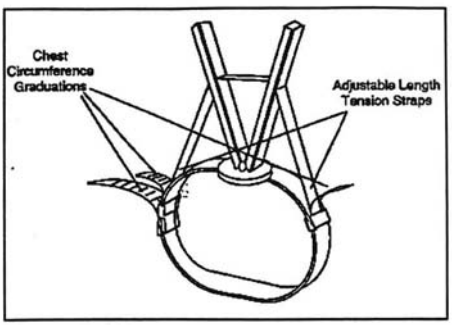

Figure 5. Weightlessness Device

VEST DEVICE

Vest CPR device is based on the thoracic pump. (Figure 6) The device is designed as a cuff containing a bladder that is wrapped around the patient's thorax. The bladder, which can be inflated and deflated in cycles, is attached to an automated pneumatic system to provide cyclical chest compression. Compression duration, inflation pressure, and rate can all be controlled with this device. Usually the compression duration is set to 40 to 50% of each cycle, inflation pressure is 250 mmHg, and the rate is 60 times/minute. A small positive pressure is always kept between the chest and the vest to provide a tight fit between the two, but the device totally deflates during ventilation to provide for chest expansion. The design of vest CPR causes a

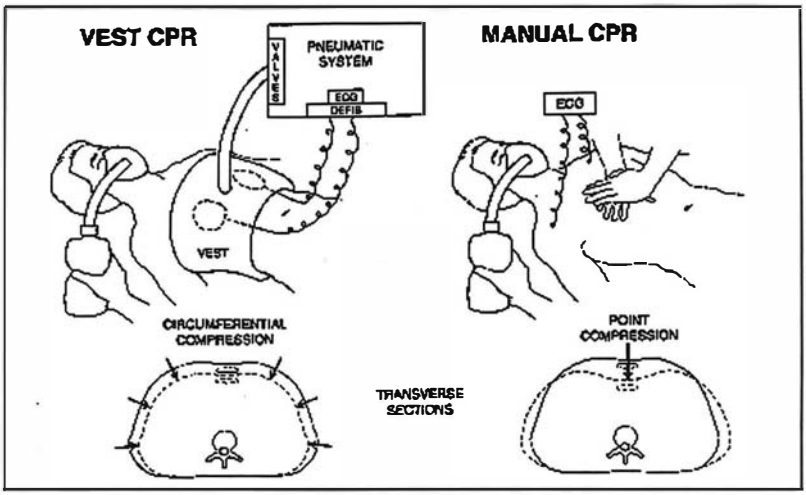

Figure 6. Vest CPR devices versus Manual CPR

large rise in intrathoracic pressure. The pressure is distributed evenly around the chest wall causing minimal displacement at any point on the thoracic surface. Therefore, traumatic injury is also reduced because no one point receives a high amount of pressure (14 to 15 psi). Preliminary results with this device seems to be very promising {{15,16}}

RESUSCITATION APPARATUS

The Resuscitation Apparatus is another device based on the thoracic pump. (Figure 7) There are no published reports on this device. One end of the device is attached to a roller; the other side is attached to a lever arm, which is pushed up and down to produced circumferential tightening and

relaxation. The device can be applied to the patient manually or mechanically. This device can also do sternum compression. A cylindrical rod is placed between the strap and the sternum. When the lever arm is pushed down, the strap applies pressure to the cylinder and pressure is transmitted to the sternum. {{29}}

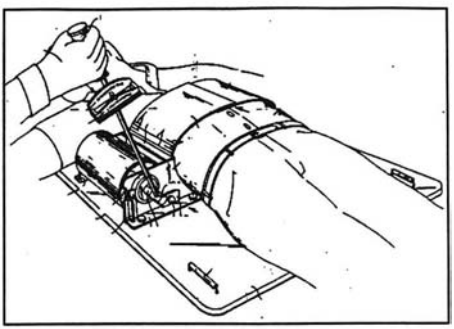

Figure 7. Resuscitation Device

NEW TECHNIQUES TO AUGMENT BLOOD FLOW DURING CPR

There have been many trials to develop new CPR techniques which can generate higher blood flow to vital organs by modifying standard CPR technique. Some of these techniques are Simultaneous Compression and Ventilation (SCV-CPR), High-Impulse External Chest Compression (HI-CPR), and Interposed Abdominal Compression (IAC-CPR). Currently, standard CPR has been used as a "measuring stick" for all the other techniques to compare. Problem with this is that survival rate with standard CPR is also to low that results from any other device initially seem to promising.

SIMULTANEOUS COMPRESSION AND VENTILATION (SCV-CPR)

Simultaneous chest compression and ventilation (SCV-CPR) is a

technique in which ventilation is performed simultaneously with every compression. This technique produces a forward blood flow by acting as a "thoracic pump" mechanism {{20}}{{30-39}} in which a pressure gradient is formed between the intrathoracic and extrathoracic structures. In animal models, SCV-CPR improves cerebral flow and systolic pressures {{20}}{{31}}{{32}}. A device which provides simultaneous chest compression and ventilation was introduced and clinically tested in the 1980s. An improvement in survival rate in one study was discovered with SCV-CPR in comparison to standard CPR, but the result could not be reproduced at other testing sites.{{30}}{{34}}{{35}}{{38}}{{39}}

HIGH-IMPULSE EXTERNAL CHEST COMPRESSION (HI-CPR)

This technique, which was introduced by Maier et al {{17}}, is very similar to standard CPR. The only difference is that the compression rate is performed at a higher rate. (120 to 150 compressions/minute) {{9}}{{18}}{40-42}} Ventilation with this technique remains at 12 breaths/minute the same as with Standard CPR.

Some investigators have reported that high-impulse CPR improves aortic pressure, cardiac output, and myocardial perfusion pressure in animal models {{9}}{{18}}, but clinical studies on this technique have yet to be done. These changes are only reported with manual, not mechanical, high impulse CPR {{37}}{{40-42}}. Further study is needed to determine whether high-

impulse CPR is better than standard manual CPR.

INTERPOSED ABDOMINAL COMPRESSION (IAC-CPR)

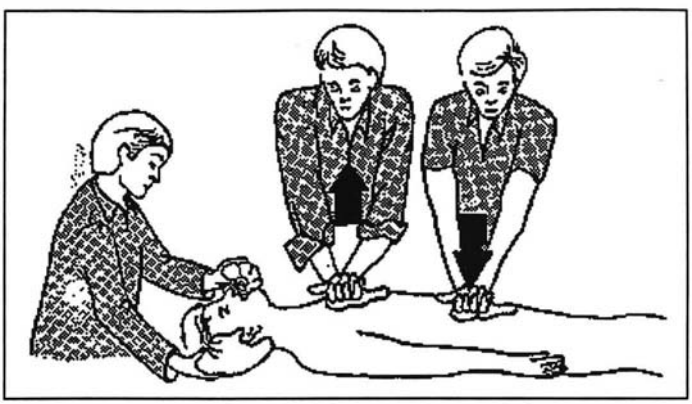

Figure 8. Interposed Abdominal Compression (IAC)-CPR

Another name for interposed abdominal compression (IAC)-CPR is abdominal counterpulsation CPR. This technique requires two more rescuers to provide compression to the chest and to the abdominal region close to the umbilicus. During the relaxation phase of chest compression in standard CPR, the second rescuer provides compression to the abdominal region. (Figure 8) The rate that the compression is applied is 80 to 100 compressions/minute, which is the same as for chest compression. The amount of compression force to apply is still in question. Some researchers have measured the abdominal compression pressure as 20 to 150 mmHg by different measuring gauges but there is as yet no established guideline.{{19}}{{26}}

METHOD

SIMULTANEOUS STERNO-THORACIC (SST)-CPR

None of the existing techniques or devices have tried to take an advantage of both CPR mechanisms simultaneously. Physiologically, SST-CPR should result in increased intrathoracic pressure with simultaneous compression of the heart producing a synergistic increase in blood flow and pressure.

DEVICE

The simultaneous Sterno-Thoracic (SST)-CPR device incorporates both components of the "cardiac" and "thoracic" pump. (Figure 9) In developing the device, many different aspects (dimensions, weights, materials and mechanics) had to be considered and understood. In describing this device, a general description and detail description are presented. The general description gives an overview of the device, while detail description actually describes each component of the device.

GENERAL DESCRIPTION OF THE DEVICE

The device consists of two main elements. (Figure 10) The first element is a piston which depresses the sternum. The second is a circumferential strap that compresses the thorax as the piston is pushed down on the sternum. The

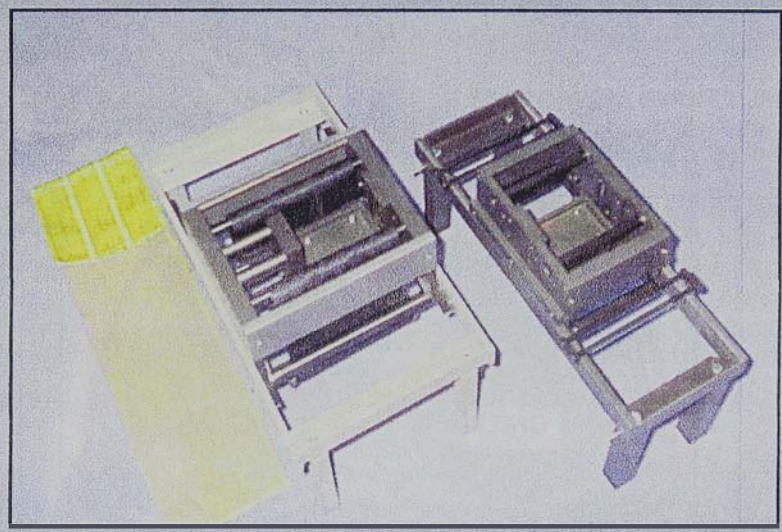

FIGURE 9. Simultaneous Sterno-Thoracic (SST)-CPR Device

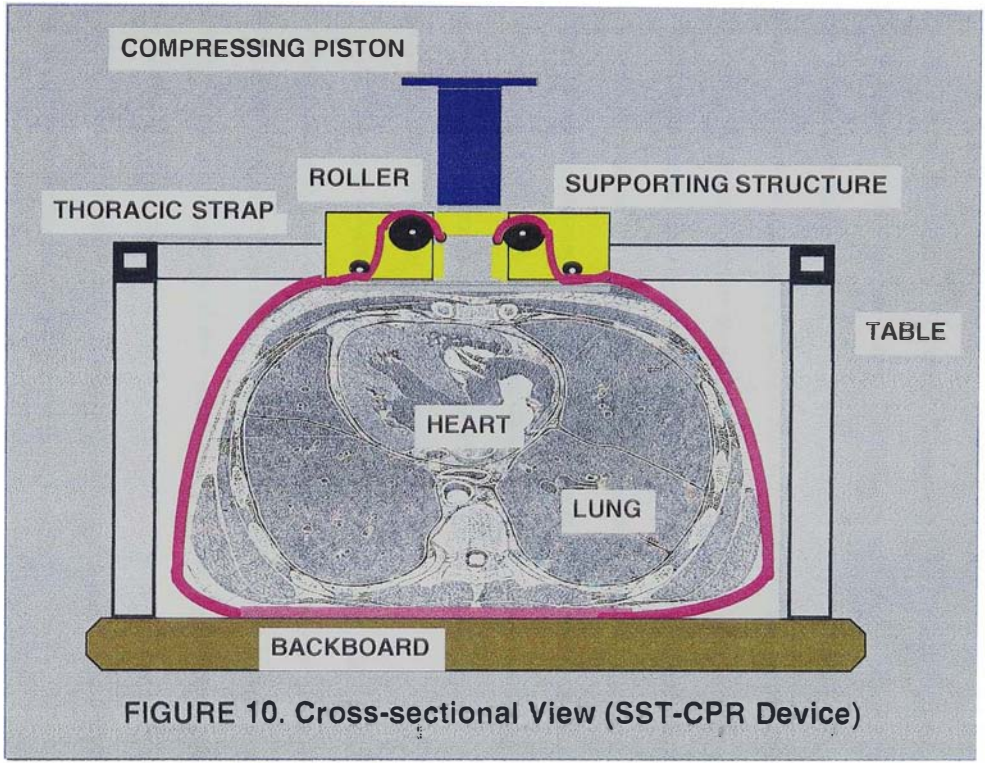

FIGURE 10. Cross-sectional View (SST-CPR Device)

sternal piston is a rectangular shaped solid prism that has broad top and handle. For clinical testing, Automatic Resuscitator was used to power the device instead of the sternal piston. The anterior end of the circumferential strap is attached to both sides of the compressing piston. The strap passes through rollers on a supporting structure that surrounds the sternal piston. The supporting structure is placed on top of a table which has a hole in the center. This table supports the weight of the supporting structure, and the adjustable legs are mounted on the backboard. When the sternal piston is pushed down, it depresses the sternum and pulls on the chest compression straps, thus applying direct force to the sternum (cardiac pump) and circumferential force to the thoracic cage (thoracic pump). (Figure 11)

DETAILED DESCRIPTION OF THE DEVICE

A Computer Aided Design software (SilverScreen^{R}, 3d CAD/Solid Modeling Software) was used to develop a preliminary concept of the SST-CPR device. This software helped in designing a solid model and in allowing us to create many upgrades of the model prior to developing the final design. The device is composed of five different components: strap, piston, supporting structure, table, and backboard. The general dimensions are shown in Table 1 and the design's dimensions are shown in the appendix.

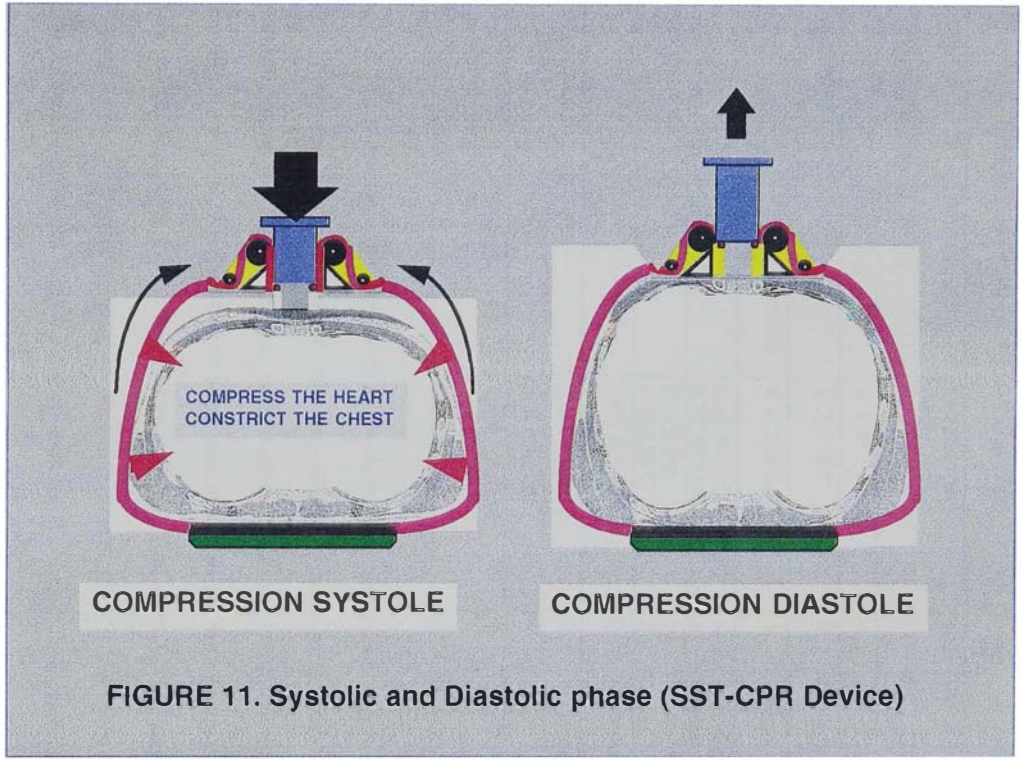

FIGURE 11. Systolic and Diastolic phase (SST-CPR Device)

TABLE 1. Size and Material for SST-CPR Device

STRUCTURE	COMPONENTS	DIMENSIONS (W X L X H)	NO	MATERIALS
COMPRESSING PISTON	PISTON	2" X 3" X 4"	1	Aluminum
	HANDLE	3" X 5" X 0.5"	1	Aluminum
SUPPORTING STRUCTURE	BASE (L-SHAPED)	2" X 3" X 3"	2	Aluminum
	INCORPORATED PISTON	2" X 3" X 2.5"	1	Aluminum
	ROLLERS (BEARING)	0.25"X0.5"IDX1.0"OD 0.25"X0.25"IDX0.5"OD	2 2	Steel Ball bearing
	ROD	6" X 0.5"D 6" X 0.25"D	2 2	Steel
	CONNECTING BRIDGE	2.25" X 1.5" X 2"	2	Aluminum
THORACIC STRAP	STRAP	5" X 50" X 0.1"	1	Polyester
	ROD	5" X 0.25"OD	3	Steel
BACK SUPPORTING STRUCTURE	BACK BOARD	15" X 25" X 0.5"	1	Wood

The purpose of the strap is to augment the "thoracic pump" mechanism. The strap can be broken down into two different components: the Velcro portion around the thorax, and the portion which connects the Velcro strap to the piston. (Figure 12) The Velcro strap was designed for the human thorax. The length was forty inches with a five and half inch width. This size was chosen to cover the thorax cage of an average man (5' 8"), but more study and testing need to be done to better optimize the length and width of the strap.

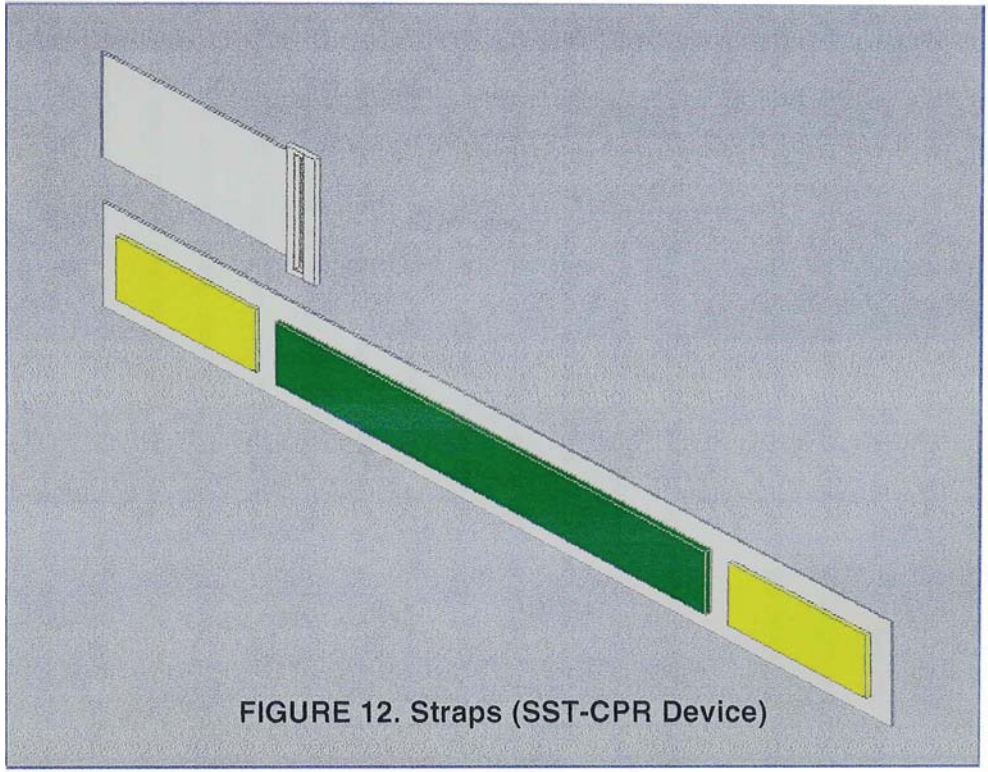

FIGURE 12. Straps (SST-CPR Device)

The strap connection between the Velcro and the piston is ten inches in length and two inches in width. This size was chosen to provide adequate movement of the strap with a two inch compression. The two inch width was designed so the strap would fit inside the piston. The length was chosen to allow travel of the Velcro strap without catching on the bearing in the base-supporting structure.

The piston, which simulates the "cardiac pump" mechanism, is designed to provide a two inch compression depth. (Figure 13) The pistons also anchors the straps. When the piston travels down two inches, the straps are pulled down two inches on each side providing a four inch reduction in circumferential length. To keep the piston aligned, there are grooves in the base-supporting structure to guide the piston perpendicularly. The piston is designed to provide compression at the sternum. The dimensions of the bottom half of the piston are designed to fit for sternal compression. The top half of the plunger is designed to fit Automatic Resuscitator plunger head.

The base-supporting structure is designed to house the piston and provide space for the strap to attach to the piston via rollers. (Figure 14) The lower roller is adjustable to provide a tight fit against the thorax. A tight fit is needed for the Velcro strap to encircle the greatest circumferential thoracic area. The top roller is fixed to provide the two inch height to the thorax. The structure is rectangular prism with a space on top designed to fit the Automatic

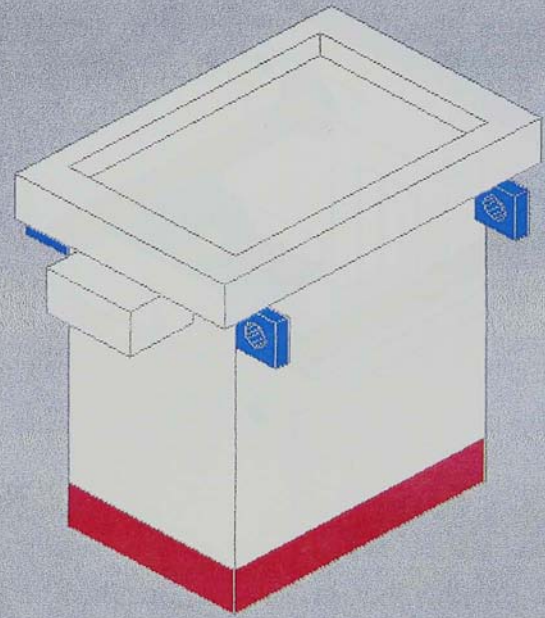

FIGURE 13. Piston (SST-CPR Device)

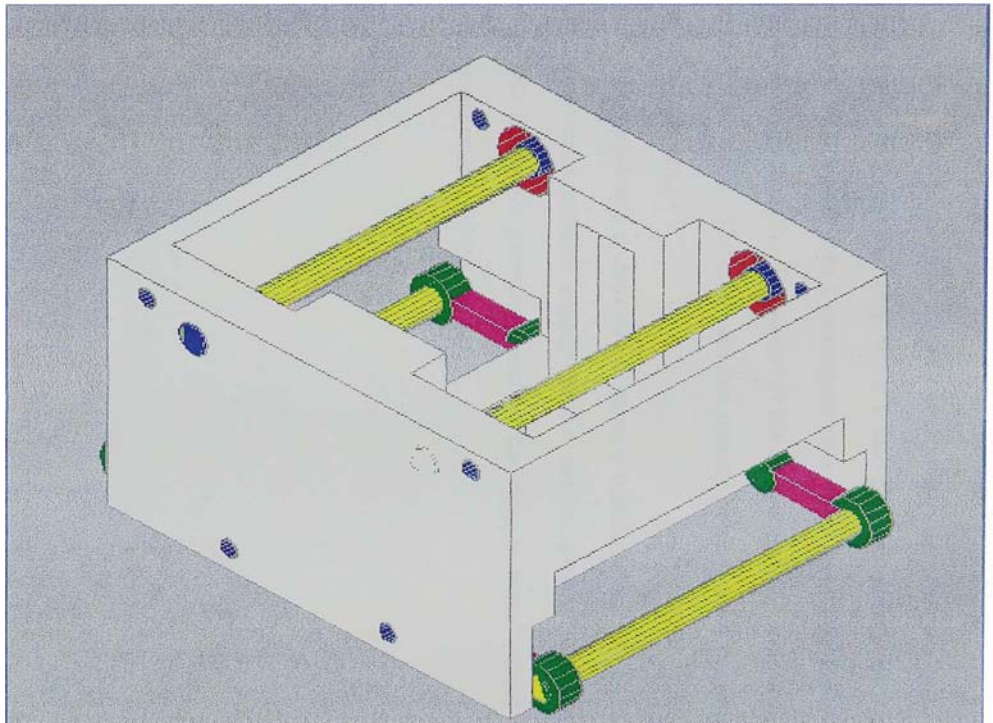

FIGURE 14. Base-supporting Structure (SST-CPR Device)

Resuscitator piston head.

The table is the part of the device, which supports the weight of the base-supporting structure. (Figure 15) It also provides stability for the device. The table is designed with a rectangular hole in the middle for the piston and the strap to go through. The weight of the base-supporting structure is on the edge of the device. The table also has multiple holes in each leg to provide a height adjustment.

The backboard is designed to place a patient on top of the board. Its angular structure provides support for the patient. (Figure 16) The groove within the backboard provides space for the strap to move without catching between the board and the thorax.

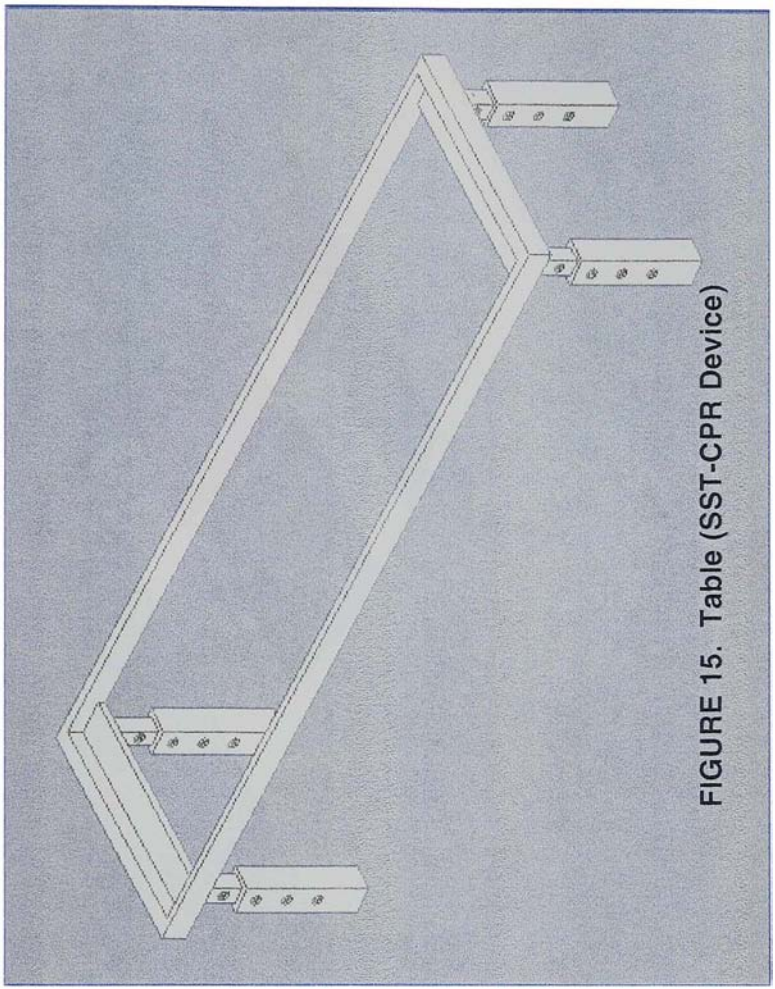

FIGURE 15. Table (SST-CPR Device)

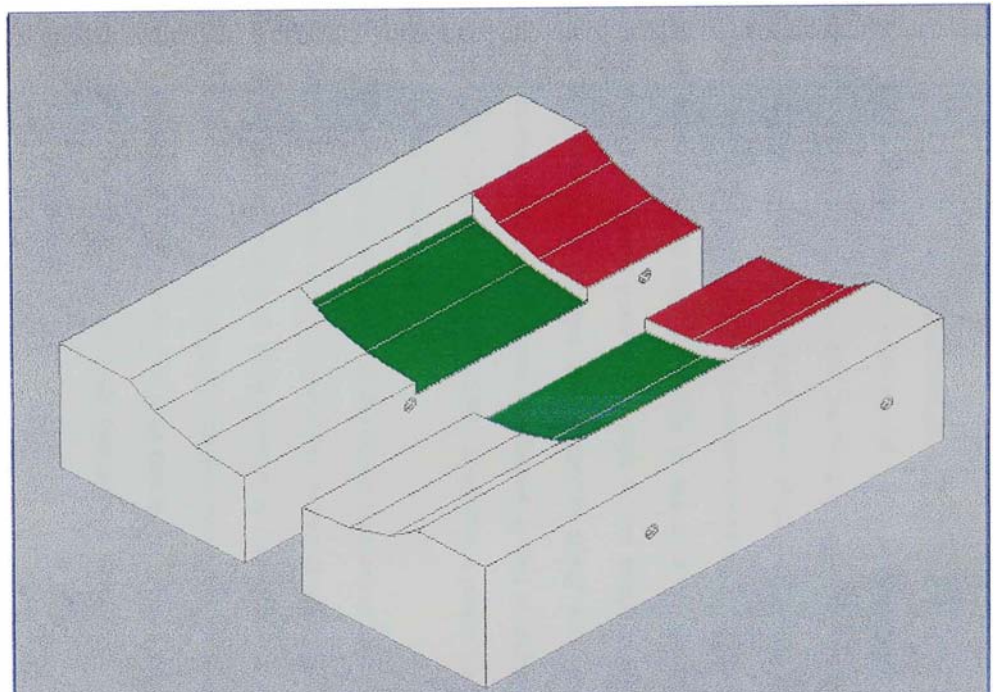

FIGURE 16. Backboard (SST-CPR Device)

MECHANICAL STUDY

Before performing an experimental study in an animal model, the prototype was tested on a model of the thoracic cage. The model was made out of a plastic barrel which is show below. (Figure 17) Initial testing of the device's mechanical properties was to be performed on a CPR manikin, which has physical properties that are similar to the adult human torso, but the compliance of the plastic chest was to high for the device to function. CPR manikin is designed to simulate only a "cardiac pump" and does not allow circumferential shortening as would be needed to test a "thoracic pump" or "combination" CPR device like SST-CPR. Therefore, a plastic barrel was used to simulate the thoracic cage.

Following things were investigated during mechanical testing: 1) whether the piston compression would constrict the thorax simultaneously, 2) if the strap would reduce the thoracic circumference by four inches, 3) whether, the piston would return to its starting (top) position during the relaxation phase of chest compression, 4) the amount of force needed to depress the sternum two inches, and 5) whether the Automatic Resuscitator will provide an adequate power source for the device.

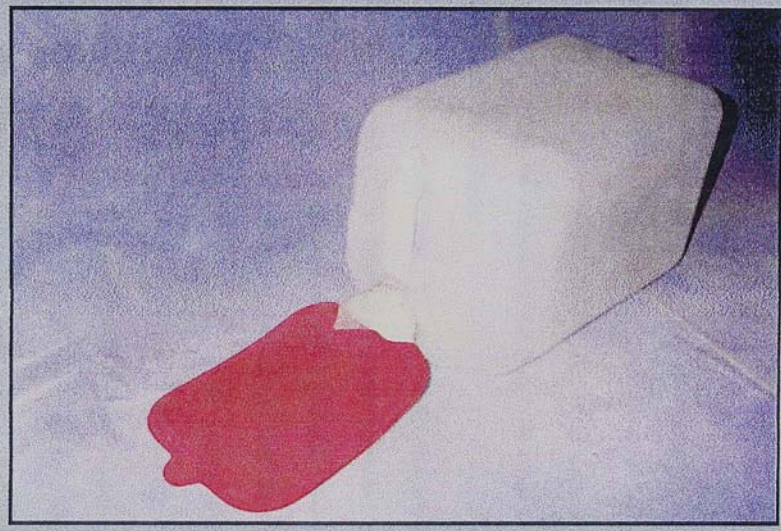

FIGURE 17. Plastic Barrel for Mechanical Testing

PILOT ANIMAL STUDY

STUDY DESIGN

After the prototype was tested mechanically, animal studies were performed to assess its hemodynamic performance. The model used to test the device was a chronically instrumented dog transferred from other experiment to determine myocardial oxygen consumption. The dog was instrumented 2 weeks before testing the device by other investigators.

1. Preparation of the animals

One healthy adult mongrel dog was instrumented under sterile conditions for studies. The animal was pre-medicated with Cefazolin 1 g, anesthetized with sodium pentothal, and ventilated. A left thoracotomy was performed through the fourth intercostal space and the heart was exposed. Ultrasonic crystals were positioned across the minor and major axis of the heart, and ultrasonic flow probes were placed around the ascending aorta and left circumflex coronary artery. Silicone rubber introducers were placed in the left atrium, aorta and mediastinum. A pacing wire was sewn to the right atrium and the left ventricle. All equipment was placed in a subcutaneous pocket dorsal to the incision. A chest tube was placed and the thoracostomy closed. The chest tube was removed one to two hours postoperatively. Each animal

will be allowed to recover for ten days to two weeks postoperatively, receiving postoperative antibiotics and wound care as needed. When the animal recovered, the instruments were externalized by sedating the dog with 0.5 mg/kg morphine sulfate IM, premedicating it with 1 g Ancef IM, and sterilely draping the area of the subcutaneous pocket. A 3 cm incision was made under local anesthesia with 1% lidocaine with epinephrine. Using only sterile instruments, the tubing and transducer wires were removed and the wound dressed and wrapped securely. Through cutdown on the external jugular vein, a catheter was introduced to the right atrium. Micromanometers were placed in the left atrium and thoracic cavity through the respective introducers, and fiberoptic catheter were placed in the aorta. The animal was watched closely post-procedure until the sedative has worn off. All transducers, catheters and flow probes were connected and calibrated with adequate data acquisition system respectively.

The dog was euthanized with potassium and pentobarbital. Immediately after euthanasia, the dog was transferred to us for testing new CPR device. There were no additional invasive procedures after euthanasia and throughout our experiment.

Table 2. Experimental Procedure during SST-CPR

Time(min.)	Procedure	Measurement(30 sec)	Equipment
0 - 2	Thumper^{R} CPR	Coronary blood flow Aortic pressure RA pressures Intrapleural pressure	Device Thumper Oxygen tank
3 - 5	SST-CPR		
6 - 8	Standard CPR		
9 - 11	SST-CPR		

2. Cardiopulmonary resuscitation(CPR) (Table 2)

To delineate the efficacy of SST-CPR, three different techniques of CPR were performed in a dog. Standard CPR was performed with AHA guidelines by one of our investigators.{{2}} Mechanical CPR was performed with the automatic resuscitator. Two types of CPR using our new device were performed. One method of CPR was performed by a compression piston with a thoracic strap(SST-CPR). The other method of CPR was performed by only using a compression piston simulating mechanical CPR. The same mode of compression and ventilation were provided for each CPR method: The point of the compression was centered at the lower 1/3rd of sternum. The depth of compression was 2 inches for each CPR method. The compression rate was 80 cycles per minutes for each method. The duration of compression systole and compression diastole was 50/50. After initiation of CPR, respirations were

delivered by ventilator.

Each intervention took two minutes and was followed by a minute for application or removal of the device. The complete study took less than 30 minutes after euthanasia.

3. Measurement

We measured the aortic pressure from the ascending aorta, the coronary blood flow of the left anterior descending coronary artery, and the intrapleural pressure from the mediastinum. Measurements was done before euthanasia and during each CPR techniques. To get a steady state, data aquisitions were performed one minute after the beginning of each CPR technique. We acquisited the data for a minute for each CPR technique using computerized data aquisition system. Data acquisition frequency was 200 per second. Stored data were analysed and graphed with MS Excel program. Each hemodynamic parameters were compared according to CPR methods.

4. After study

The dog was not defibrillated and resuscitated during the study. The skin and bony thorax were examined to determine the presence of complications from SST-CPR.

RESULTS

MECHANICAL STUDY

During mechanical testing, the device could provide simultaneous constriction to the mechanical model while piston was compressed. Compressions and constrictions were occurring in one phase. The strap was released immediately at the beginning of the compression diastole.

We noticed that in this mechanical model, the piston did not return to the starting position as the time passed. We found that the barrel continued to lose it's compliance and volume progressively so that the piston could not return to the normal position. However, we believe that the sternum would provide an adequate compliance and inertia pushing the piston to its starting position. The strap reduced circumferentially four inches at the beginning of the test, but since the piston was not returning to the top, the strap tightened and relaxed according to excursion of the piston.

The Automatic Resuscitator was an adequate power source for mechanical testing. Since the force required to compress the piston was only 80-90 psi during mechanical test, Automatic Resuscitator was adequate to depress the sternum two inches.

PILOT ANIMAL STUDY

AORTIC PRESSURE (Figure 18)

A relatively higher aortic systolic pressure during SST-CPR(statistically not significant) was generated. The aortic pressure curve simulated the combination of the pressure curve from the Thumper^{R} and manual CPR. The peak systolic aortic pressure during SST-CPR was higher than either manual or Thumper^{R} CPR. Systolic aortic pressure had two peaks and simulated an exaggerated aortic pressure curve from Thumper^{R} CPR. The diastolic aortic pressure during SST-CPR had a deep dip in early diastole.

CORONARY BLOOD FLOW (Figure 19)

A relatively higher diastolic coronary blood was generated during SST-CPR(statistically not tested). But total coronary blood flow was not different from the other techniques because SST-CPR also generated more negative coronary blood flow during the systolic phase. The coronary blood flow curve showed a sluggish increase in the early phase of diastolic flow.

INTRATHORACIC PRESSURE (Figure 20)

A higher intrathoracic pressure was generated during SST-CPR than with either of the other techniques(statistically significant). The intrathoracic pressure curve was very similar with Thumper^{R} CPR during compression systole, but the diastolic curve was different from that seen with Thumper^{R} CPR. SST-CPR created a much lower negative intrathoracic pressure with an early dip compared to that seen with Thumper^{R} CPR.

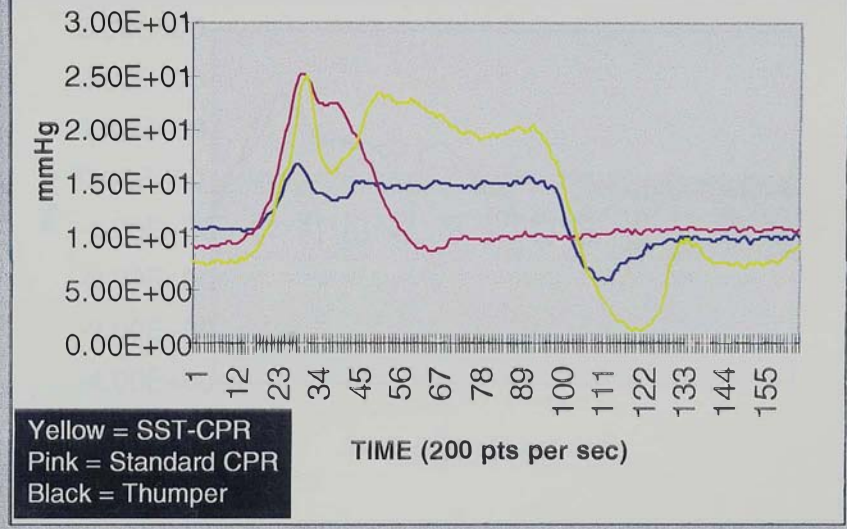

FIGURE 18. Aortic Pressure

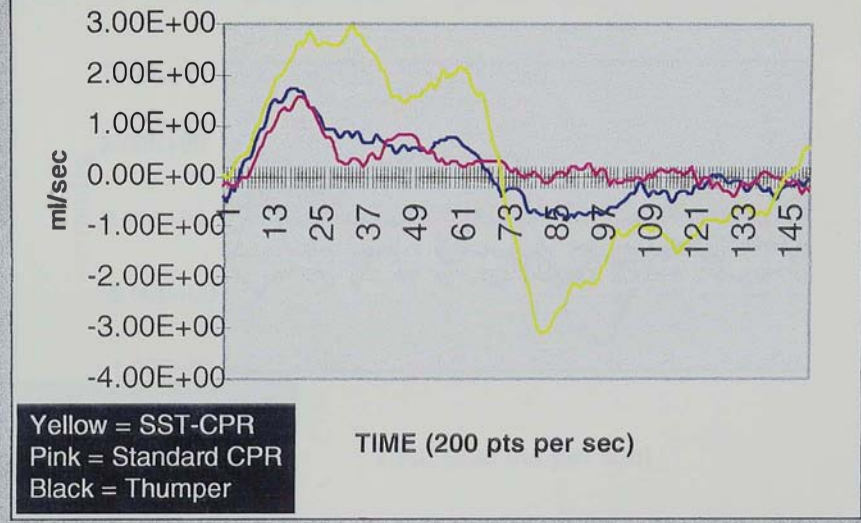

FIGURE 19. Coronary Blood Flow

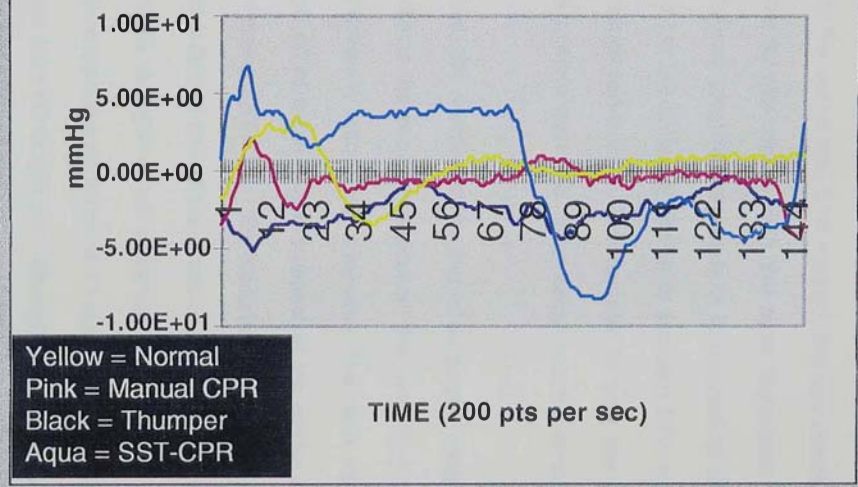

FIGURE 20. Intrathoracic Pressure

INTERPRETATION

To justify the use of new CPR method, the new device should generate more blood flow to vital organs than the other techniques. The SST-CPR device has produced promising results both in mechanical and animal model. The behavior of the our device is similiar to standard CPR in that the sternum is being compressed two inches. This can also be seen the pilot animal study that shows the compression and constriction occuring simultaneously during systolic.

Mechanical, the device has functioned to proper design configurations. The piston produces the two inch execution; the thoracic strap is shorten by four inches. Friction is reduced to a minimal; The table can be adjusted to different heights. All of these different aspect of the device make the design effective in performing SST-CPR. The materials, which are used in SST-CPR device, also make fatigue and stress a non-questionable issue. For example, the smallest tensile strength of an aluminum alloys, which makes the table, piston, and base supporting structure, is 13 kpsi. The forces which are used in this device range from 60 to 100 psi. Strength should not be a factor in the performance of SST-CPR device.

As hypothesized in designing this new device, we tried to test the effect of combination of cardiac and thoracic pump simultaneously. We could see a new type of the aortic pressure curve which is actually a peak and plateau pattern. The peak component was very similar to that from standard CPR, but plateau component was a new type of pressure curve. We interpreted that peak component of the aortic pressure in the early systole was generated by compressing the piston, and plateau component of the aortic pressure in the late systole by constricting the strap. Difference of phase in which patterns of the aortic pressure curve appear gives us a clue in assuming the mechanism of blood flow generated by this device. Appearance of peak component at the beginning of compression systole suggests that direct compression of the sternum by the piston plays a major role in this period. Late appearance of plateau pattern might be attributed from mechanism of constriction in this device. We found that the strap constriction began from piston compression and tension of strap was maximized by the full excursion of the piston. Therefore, plateau pattern of the aortic curve has occurred at the late systolic phase as intrathoracic pressure has increased progressively during piston compression. In other words, the plateau was caused by prolonged "thoracic pump" compression. We didnot measure cardiac output in pilot experiment, but the new curve produced a higher mean systolic pressure which was sustained for a longer period of time. This could result in better cerebral and myocardial blood flow than with standard CPR.

The diastolic aortic pressure during SST-CPR had a deep early dip reflecting a sudden decrease in the intrathoracic pressure during the early release phase. A very low diastolic pressure and intrathoracic pressure during SST-CPR should improve venous return from the extrathoracic to the intrathoracic space.

Higher coronary blood flow is important for sustaining survival of heart muscle during cardiac arrest. By producing a higher aortic pressure curve, SST-CPR should produce better coronary blood flow than with standard CPR. Results from our pilot experiment showed that SST-CPR could generate better coronary blood flow than with standard CPR. However, it was observed that more negative coronary blood flow occurred during the compression systole, which could be detrimental in restoring spontaneous circulation. Further study will be needed to determine the significance of this observation.

By increasing intrathoracic pressure with the strap in SST-CPR, a new type of intrathoracic pressure curve was generated. During compression systole, intrathoracic pressure curve composed of two components: a peak and plateau curve which reflects the effect of compression of the piston and constriction of the strap respectively. Another interesting observation was that intrathoracic pressure during SST-CPR was more constant and higher than with other techniques throughout respiration. This suggests that our device could compress the thorax cavity enough to generate high intrathoracic pressure irrespective of

the size of the thorax. At the very early phase of the compression diastole, intrathoracic pressure of SST-CPR decreased very rapidly and reached to more negative level than with other techniques. This phenomenon might be due to rapid release of the thoracic cavity after marked constriction. Diastolic negative pressure could help to increase venous return from extrathoracic to intrathoracic cavity.

As described above, this new device to perform SST-CPR could generate a new type of aortic pressure curve, higher systolic aortic pressure and coronary blood flow, and higher consistent intrathoracic pressure than with standard or mechanical CPR. We think that SST-CPR using this new device could give us a better understanding of the physiology of blood flow during CPR and a better method to resuscitate the patients with cardiac arrest.

LIMITATIONS

Few limitations of the device were detected during mechanical test. The alignment of the piston head to the center of the heart seem to be a problem. There need to be a better technique in centering the device, but the problem is also in the animal that is used for testing. The configuration of the thorax of the dog is not similar to the human's. Considering the function of our device using simultaneous compression and constriction of the thorax, the animal having flat chest is preferred to test the device. Another difficulty encountered during experiment is the tightening the strap. The strap seemed to loose it's

tension as SST-CPR progressed. We will modify the strap with one attachment to the piston and one adjustable portion instead of having two portion of the Velcro. The device can also be constructed with a lighter weight. To reduce the weight, Some kind of kelvar alloy can be used.

The main limitation in pilot study was the number of the dog used in experiment. Because the test of the device was performed in only one dog, hemodynamic results from SST-CPR could not be generalized. However, the aortic and intrathoracic pressure curves generated by SST-CPR looked like the curve hybridized by piston compression and strap constriction as we hypothesized.

The automatic mechanical compressor was used to compress the piston of the device. However, the force of the automatic mechanical compressor was not enough for the dog even though it was adequate for mechanical testing. A mechanical compressor with a larger strength is necessary to continue the animal study. Further animal study is needed to delineate the efficacy of this new device and the exact mechanism of blood flow of SST-CPR.

SUMMARY AND CONCLUSION

Cardiac arrests occurs once every two minutes somewhere in the United States alone. {{43}} The purpose of CPR is to facilitate blood movement to the vital organs and tissues. Without adequate amounts of oxygen and waste removal, vital organs and tissues do not survive. The first organ to fail in function is the brain. The brain survives only five to six minutes after a patient goes into cardiac arrest. This is why early access, early CPR, early defibrillation and early advanced care are so important.

Simultaneous Sterno-Thoracic(SST)-CPR Device is designed to provide a new mechanical technique for resuscitation, and it can be used to increase the understanding of the mechanism of the blood flow during CPR. Results from the in vivo study helps define the relative importance of both the "cardiac pump" and "thoracic pump" mechanisms during CPR, and explain the synergism between the two mechanisms when they are applied simultaneously. Further study is needed on this exciting new technique.

REFERENCES

REFERENCES

1. Kouwenhoven WB, Jude JR, Knickerbocker GG: Closed chest cardiac massage. JAMA 173:1064 1960

2. Guideline for cardiopulmonary resuscitation and emergency cardiac care: Recommendation of the 1992 National Conference, Emergency Cardiac Care Committee and Subcommittees, American Heart Association. JAMA 268:2171-2302, 1992

3. Paradies NA, Martin GB, Rivers EP, et al. Coronary Perfusion pressure and the return of spontaneous circulation in human cardiopulmonary resuscitation. JAMA 1990;263:1106-1113

4. Wolfe JA, Maier GW, Newton JR et al: Physiologic determinants of coronary blood flow during external cardiac massage. J Thorac Cardiovasc Surg 95:523-32 1988

5. Deshmukh HG, Weil MH, Gudipati CV et al: Mechanism of blood flow generated by precordial compression during CPR: I. Studied on closed chest precordial compression. Chest 95:1092-99 1989

6. Higano ST, Oh JK, Ewy GA et al: The mechanism of blood flow during closed chest cardiac massage in humans: transesophageal echocardiographic observations. Mayo Clin Proc 65:1432-40 1990

7. Feneley MP, Maier GW, Gaynor JW et al: Sequence of mitral valve motion and transmitral blood flow during manual cardiopulmonary resuscitation in dogs. Circulation 76:363-75 1987

8. Criley JM, Blaufuss AN, Kissel GL: Cough induced cardiac compression. JAMA 236:246-50 1976

9. 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; 73:539-550.

10. Halperin HR, Brower MC, Weisfeldt JE et al: Air trapping in the lungs during cardiopulmonary resuscitation in dogs. Cir Res 65:946-54 1989

11. Nieman JT, Rosborough JP, Hausknecht M et al: Pressure-synchronized cineangiography during experimental cardiopulmonary resuscitation. Circulation 64: 985-91 1981

12. Werner JA, Greene HL, Janko CL et al: Visualization of cardiac valve motion in man during extemal chest compression using two-dimensional echocardiography.: implications regarding the mechanism of blood flow. Circulation 63:1417-21 1981

13. Redberg RF, Tucker KJ, Cohen TJ et al: Physiology of blood flow during cardiopulmonary resuscitaiton: A transesophageal echocardiographic study. Circulation 88:534-42 1993

14. Ma MH, Hwang JJ, Lai LP et al: Transesophageal echocardiographic assessment of mitral valve position and pulmonary venous flow during cardiopulmonary resuscitation in humans. Circulation 92:854-61 1995

15. Halperin HR, Weiss JL, Guerci AD et al: Cyclic elevation of intrathoracic pressure can close the metral valve during cardiac arrest in dogs. Circulation 78:754-60 1988

16. 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; 329:762-768.

17. Maier GW, Newton JR, Wolfe JA et al: The influence of manual chest compression rate on hemodynamic support during cardiac arrest: high-impulse cardiopulmonary resuscitation. Circulation 74(suppl IV):IV51-59 1986,

18. Feneley MP, Maier GW, Kem KB et al: Influence of compression rate on initial success of resuscitation and 24 hour survival after prolonged manual cardiopulmonary resuscitation in dogs. Circualtion 77:240-50 1988

19. Paradis NA, Halperin HR, Norwak RM. Cardiac Arrest: The Science and Practice of Resuscitation Medicine. Baltimore: Williams & Wilkins, 1996.

20. Rudikoff MT, Maughan WL, Effron M, Freund P, Weisfeldt ML. Mechanisms of blood flow during cardiopulmonary resuscitation. Circulation 1980; 61:345-352.

21. Paradis NA, Martin GB, Goetting MG, Rosenberg JM, Rivers EP, Appleton TJ, Nowak RM. Simultaneous aortic, jugular bulb, and right atrial pressures during cardiopulmonary resuscitation in humans: insights into mechanisms. Circulation 1989;80:361-368.

22. Fisher J, Vaghaiwalla F, Tsitlik J, Levin H, Brinker J, Weisfeldt ML. Determinants and clinical significance of jugular venous valve competence. Circulation 1982;65:188.

23. Weisfeldt ML, Halperin HR: Cardiopulmonary resuscitation: beyond cardiac massage. Circulation 1986;74:443-448.

24. Halperin H, Guerci A, Tsitlik J, Chandra N, Weisfeldt ML. Peak Pressure, arterial resistance, and arterial compliance determine vital organ flow during cardiopulmonary resuscitation. Circulation. Ann Emerg Med 1984;70:II-233.

25. Halperin HR, Weisfeldt ML. New approaches to CPR. Four hands, a plunger, or a vest . JAMA 1992;267:2940-2941.

26. Cummins RO. Textbook of Advanced Cardiac Life Support. American Heart Association, 1994.

27. Shultz JJ, Coffeen P, Sweeney M, Detloff B, Kehler C, Pineda E, et al. Evaluation of standard and active compression-decompression CPR in an acute human model of ventricular fibrillation. Circulation 1994; 89:684-693.

28. Technical Support Package for Device Assists Cardiac Chest Compression. NASA Tech Briefs MSC -22148. National Aeronautics and Space Administration. Lyndon B. Johnson Space Center, Houston Texas 77058.

29. Lach RD, Lusk EC. Resuscitation Method and Apparatus. Columbus, Ohio. Patent Number: 4,770,164. Date issued: 9/13/88. Application Number: 197,670. Int'l - A61H 31/00. US - 128/28. Date Filed: 10/16/80.

30. Kern KB, Carter AB, Showen RL, Voorhees WD, 3d, Babbs CF, Tacker WA, et al. Twenty-four hour survival in a canine model of cardiac arrest comparing three methods of manual cardiopulmonary resuscitation. J Am Coll Cardiol 1986; 7:859-867.

31. Chandra N, Snyder LD, Weisfeldt ML. Abdominal binding during cardiopulmonary resuscitation in man. JAMA 1981; 246:351-353.

32. Chandra N, Rudikoff M, Weisfeldt ML. Simultaneous chest compression and ventilation at high airway pressure during cardiopulmonary resuscitation. Lancet 1980; 1:175-178.

33. Niemann JT, Rosborough JP, Hausknecht M, Garner D, Criley JM. Pressure-synchronized cineangiography during experimental cardiopulmonary resuscitation. Circulation 1981; 64:985-991.

34. Sanders AB, Ewy GA, Alfemess CA, Taft T, Zimmerman M. Failure of one method of simultaneous chest compression, ventilation, and abdominal binding during CPR. Crit Care Med 1982; 10:509-513.

35. Niemann JT, Rosborough JP, Niskanen RA, Alfemess C, Criley JM. Mechanical "cough" cardiopulmonary resuscitation during cardiac arrest in dogs. Am J Cardiol 1985; 55:199-204.

36. Kisher JP, Fine EG, Weisfeldt ML, Guerci AD, Nagel E, Chandra N. Comparison of prehospital conventional and simultaneous compression-ventilation cardiopulmonary resuscitation. Crit Care Med 1989; 17:1263-1269.

37. Swenson RD, Weaver WD, Niskanen RA, Martin J, Dahlberg S. Hemodynamics in humans during conventional and experimental methods of cardiopulmonary resuscitation. Circulation 1988; 78:630-639.

38. Kern KB, Carter AB, Showen RL, Voorhees WD, 3d, Babbs CF, Tacker WA, et al. Comparison of mechanical techniques of cardiopulmonary resuscitation: survival and neurologic outcome in dogs [published erratum appears in Am J Emerg Med 1987 Jul;5(4):304]. Am J Emerg Med 1987; 5:190-195.

39. Martin GB, Carden DL, Nowak Rm, Lewinter JR, Johnston W, Tomlanovich MC. Aortic and right atrial pressures during standard and simultaneous compression and ventilation CPR in human beings. Ann Emerg Med. 1986;15:125-130.

40. Kern KB, Sanders AB, Raife J, Milander MM, Otto CW, Ewy GA. A study of chest compression rates during cardiopulmonary resuscitation in humans. The importance of rate-directed chest compressions. Arch Intern Med 1992; 152:145-149.

41. Omato JP, Gonzalez ER, Garnett AR, Levine RL, McClung BK. Effect of cardiopulmonary resuscitation compression rate on end-tidal carbon dioxide concentration and arterial pressure in man. Crit Care Med 1988; 16:241-245.

42. Martin GB, Gokli A, Paradis NA, et al. Effect of high compression rates during mechanical CPR in humans beings: preliminary results. Ann Emerg Med 1990; 19:1223-1224.

43. Clinical perspective: defibrillation: ensuring success. Health Devices 1990: 19:32.

APPENDIX

Dimensions of the SST-CPR Device

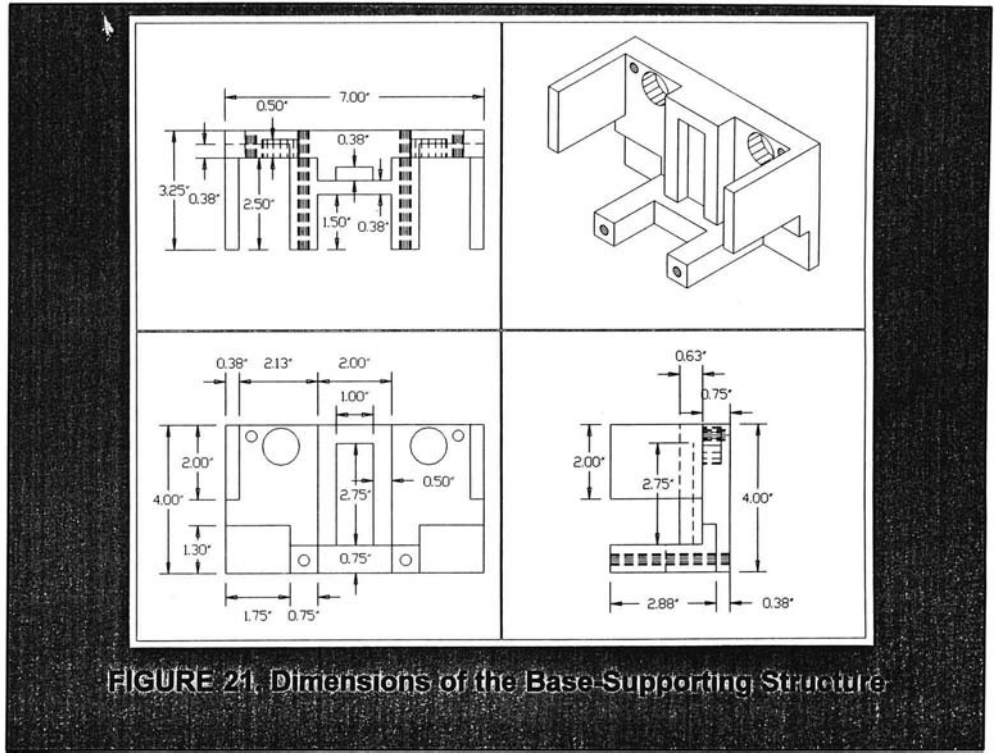

FIGURE 21. Dimensions of the Base Supporting Structure

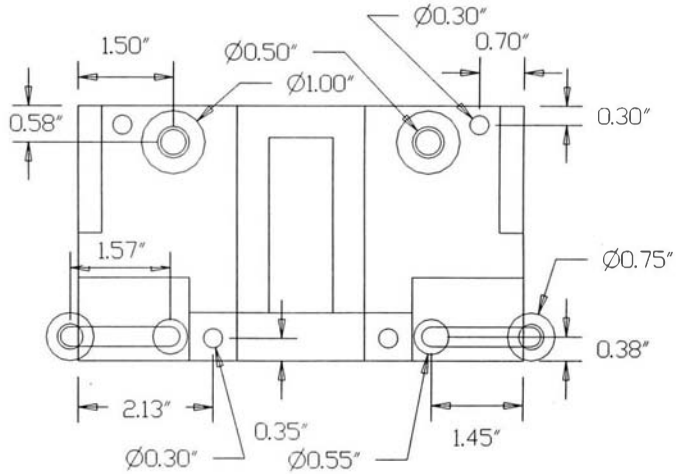

FIGURE 22. Cross-Sectional View of the Base Supporting Structure

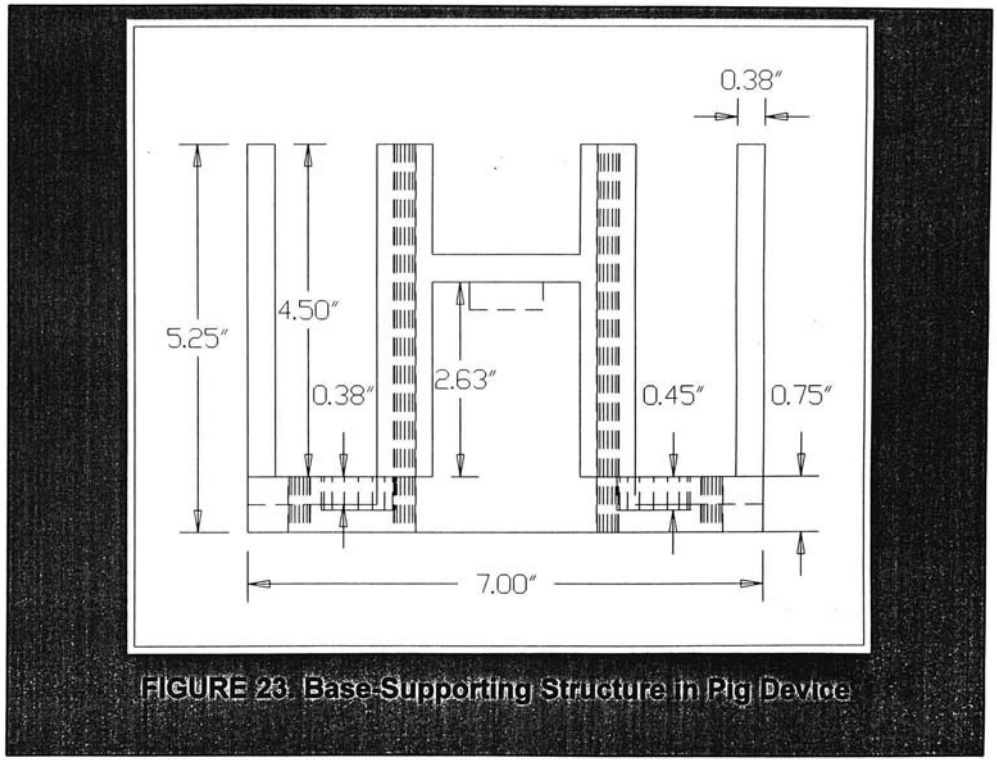

FIGURE 23. Base-Supporting Structure in Pig Device

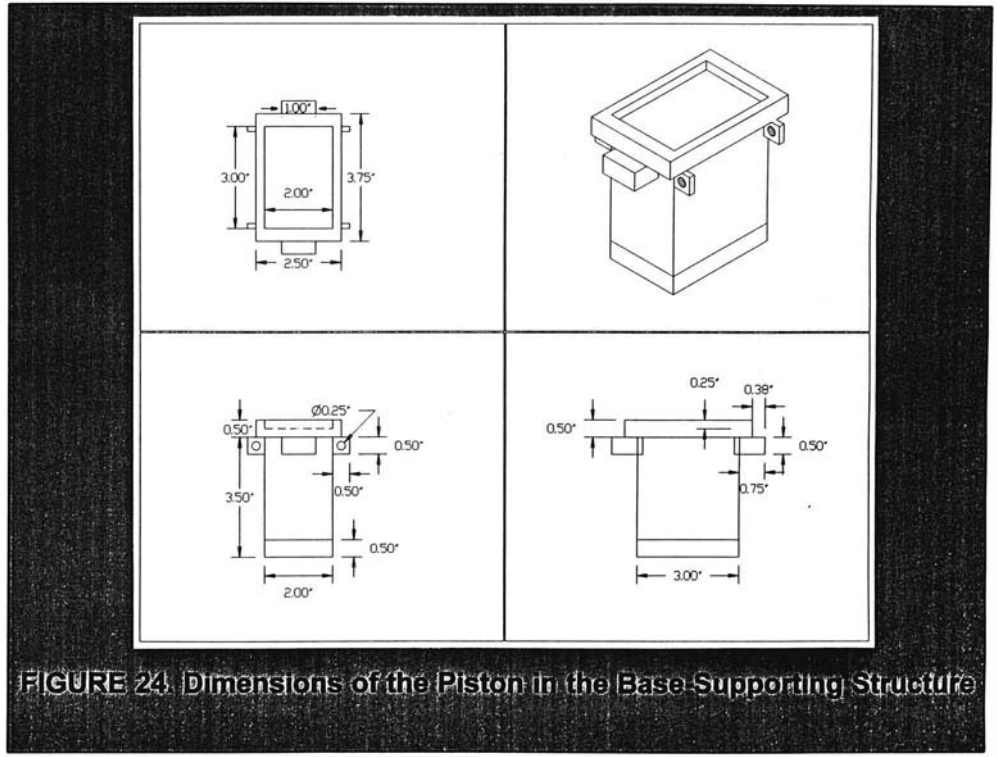

FIGURE 24. Dimensions of the Piston in the Base Supporting Structure

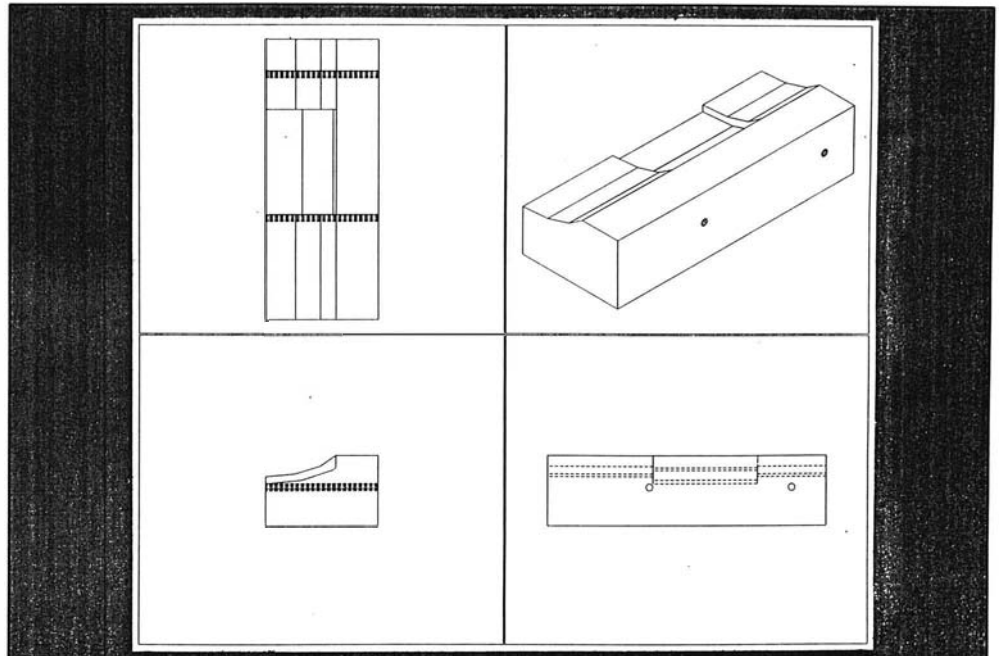

FIGURE 25. Cross-Sectional View of the Backboard for the Device

Vita