

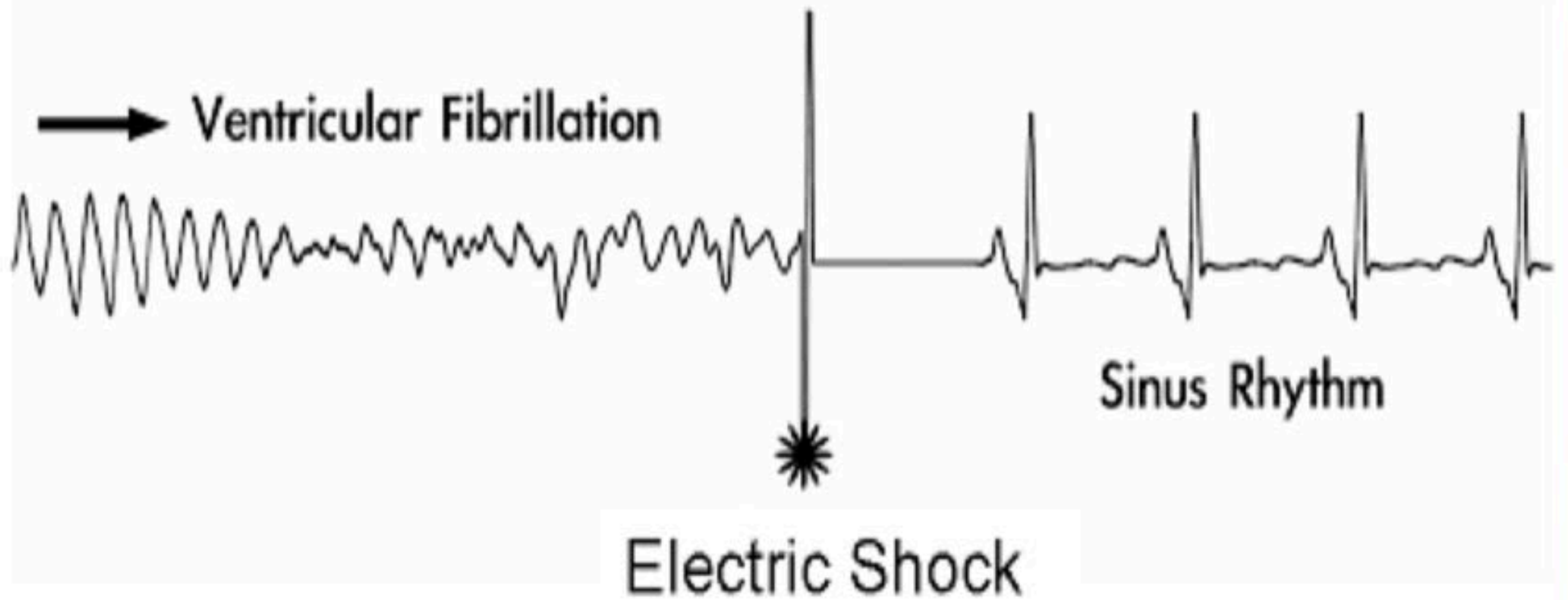


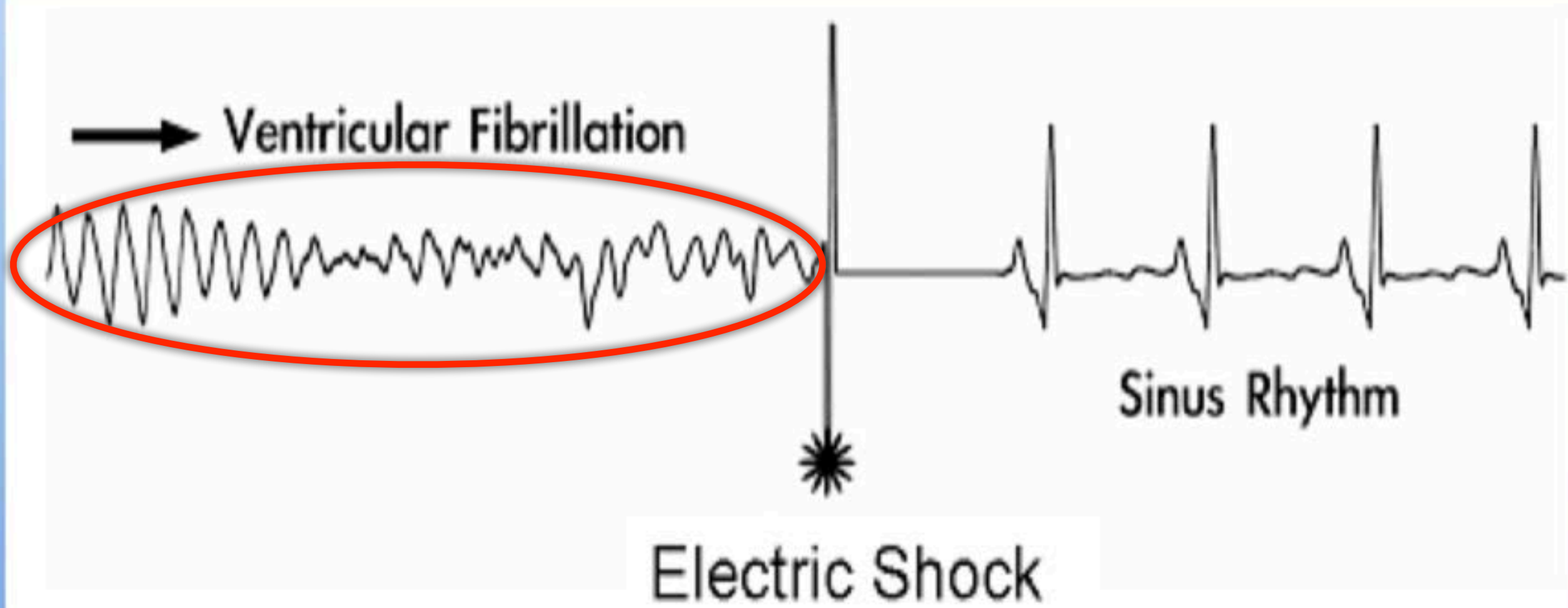
8th Annual Dr. Ed Waits Respiratory Care Conference
June 19, 2019

CQI for CPR

Providing
Real-Time Performance Feedback
During CPR

James R. Boogaerts, MD, PhD, FACC
UAB Division of Cardiovascular Disease





***Only* High Quality CPR
Can Provide**

**Adequate
Coronary and Cerebral Perfusion**

***After* Sudden Cardiac Arrest**

***Before* ROSC**



What will be covered:

- 1) **How to deliver quality to any process, including CPR**
- 2) **Normal physiological control of cardiac and circulatory function / limitations of using chest compressions to provide cardiac output from a fibrillating heart**
- 3) **Scale of the problem of Sudden Cardiac Arrest - an event most often fatal ... reflecting on our perception of an individual's mortality**
- 4) **What is happening when CPR is being performed? What are the important variables?**
- 5) **How can real-time CPR performance quality feedback be given - to optimize rate of compressions / depth of compressions / minimization of interruptions?**



Sudden Cardiac Arrest

Suddenly,
a Person becomes a Patient
... Now What?

Update on Treatment
Guidelines for SCA



Dr. W. Edwards Deming



W. Edwards Deming

Born on October 14, 1900, Dr. W. Edwards Deming was an eminent scholar and teacher in American academia for more than half a century. He published hundreds of original papers, articles and books covering a wide range of interrelated subjects—from statistical variance, to systems and systems thinking, to human psychology. He was a consultant to business leaders, major corporations, and governments around the world. His efforts lead to the transformation of management that has profoundly impacted manufacturing and service organizations around the world.

Considered by many to be the master of continual improvement of quality, as well as their overall operation, Deming is best known for his pioneering work in Japan. Beginning in the summer of 1950, he taught top managers and engineers the methods for improving how they worked and learned together. His focus was both internally, between departments, and externally,

with their suppliers and customers. As a trusted consultant, Deming significantly contributed to the dramatic turnaround of post-war Japanese industry, and their rise to a world economic power. Dr. Deming's role as the architect of Japan's post-World War II industrial transformation is regarded by many Western business schools and economists as one of the most significant achievements of the 20th century ([LA Times, 10/25/99.](#)) He is often called the “father of the third wave of the industrial revolution.”

W. Edwards Deming has taught us:

"Standard work is the most efficient method to produce a product (or perform a service) at a balanced flow to achieve a desired output rate. It breaks down the work into elements, which are sequenced, organized and repeatedly followed.

**** Each step in the process should be defined and must be performed repeatedly in the same manner. Any variations in the process will most likely increase cycle time and cause quality issues. ****

It typically describes how a process should consistently be executed and documents current 'best practices'."

Cardiac Physiology

Dynamics of Cardiopulmonary and Cardiocerebral Circulation

Quality CPR to Optimize Perfusion of the Heart and Brain



GUYTON AND HALL
TEXTBOOK OF **MEDICAL**
PHYSIOLOGY
THIRTEENTH EDITION

JOHN E. HALL

ELSEVIER

Physiology of the Heart

FIFTH EDITION

Arnold M. Katz, MD, D.Med (Hon), FACP, FACC

Professor of Medicine Emeritus
University of Connecticut School of Medicine
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 Wolters Kluwer | Lippincott Williams & Wilkins
Health
Philadelphia • Baltimore • New York • London
Buenos Aires • Hong Kong • Sydney • Tokyo

Arthur C. Guyton was born in Oxford, Mississippi, to Dr William S. Guyton, an ENT specialist and dean of the University of Mississippi Medical School, and Kate Smallwood Guyton, a math and physics teacher who had been a missionary in China before their marriage.

During his formative years, he enjoyed watching his father work at the Guyton Clinic, playing chess and swapping stories with William Faulkner,

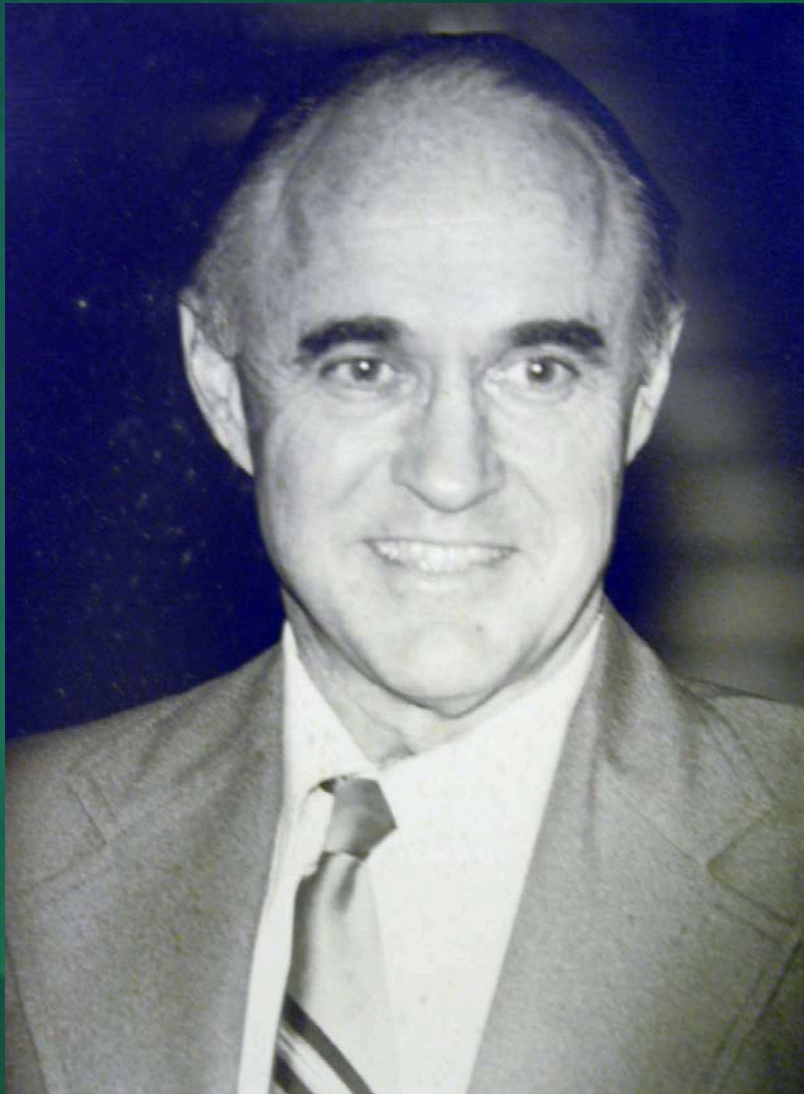
Arthur Guyton's brilliance shone early. He graduated top in his class at the University of Mississippi, distinguished himself at Harvard Medical School, and began his postgraduate surgical training at Massachusetts General Hospital.

His medical training was interrupted twice—once to serve in the US Navy during World War II and again in 1946 when he was stricken with poliomyelitis during his final year of residency training. Suffering paralysis in his right leg, left arm, and both shoulders, he spent nine months in Warm Springs, Georgia, recuperating and applying his inventive mind to building the first motorized wheelchair controlled by a “joy stick,” motorized hoists for lifting patients, special leg braces, and other devices to aid the handicapped. For those inventions he received a Presidential Citation.

He returned to Oxford where he devoted himself to teaching and research at the University of Mississippi School of Medicine and was named chair of the Department of Physiology in 1948. In 1951 he was named one of the 10 outstanding men in the nation. When the University of Mississippi moved its medical school to Jackson in 1955, he rapidly developed one of the world's premier cardiovascular research programs.

Perhaps his most important scientific contribution, however, was his unique quantitative approach to cardiovascular regulation through the application of principles of engineering and systems analysis.

Textbook of Medical Physiology is the world's best-selling physiology text.



Unit I Introduction to Physiology: The Cell and General Physiology

Chapter 1 Functional Organization of the Human Body and Control of the “Internal Environment”

Cells are the Living Units of the Body

Extracellular Fluid—the “Internal Environment”

Homeostasis—Maintenance of A Nearly Constant Internal Environment

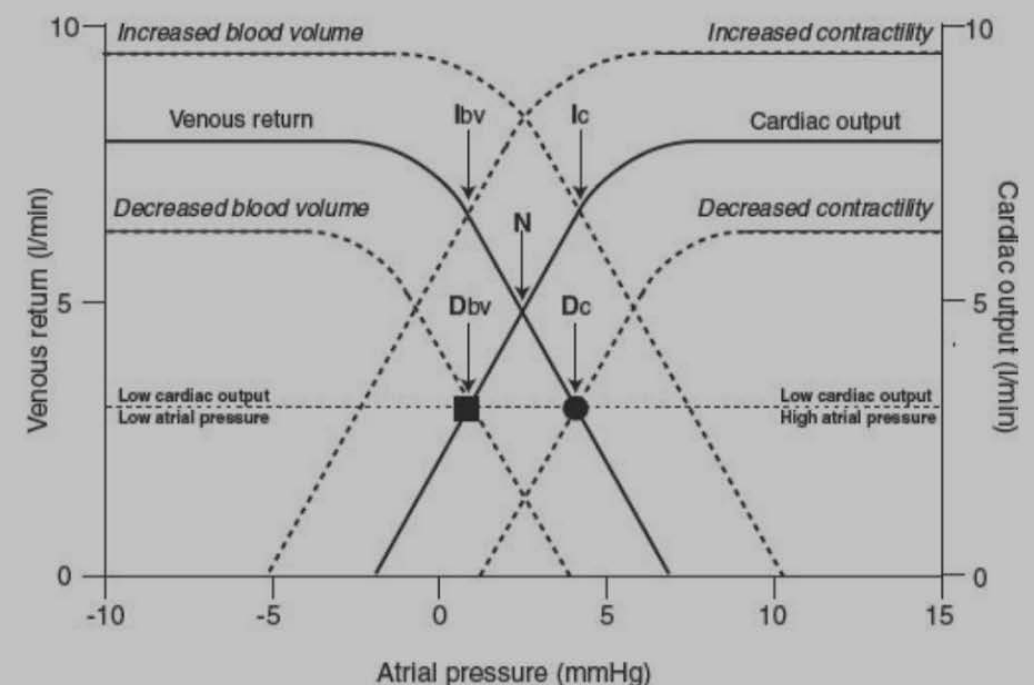
Control Systems of the Body

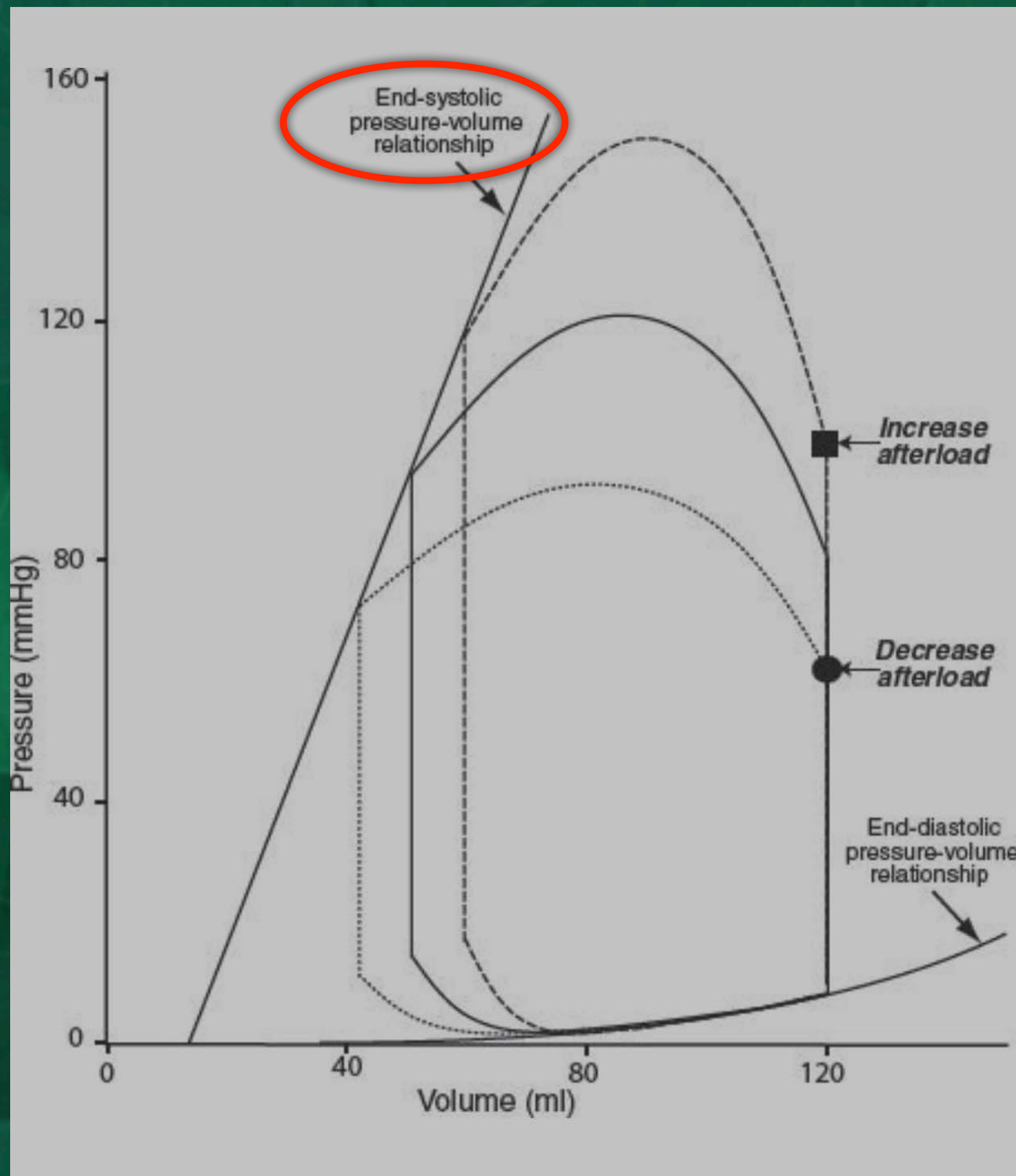
Summary—Automaticity of the Body

Bibliography

FIG. 12.29 Composite diagram, based on [Figures 12-27](#) and [12-28](#) showing the interactions between changes in blood volume and contractility. N: normal, lbv: increased blood volume, Dbv: decreased blood volume, Ic: increased contractility, Dc: decreased contractility. The dotted horizontal line shows how these curves can identify the cause of a fall in cardiac output: if atrial pressure is below normal (closed square), the low cardiac output can be attributed to decreased blood volume (Dbv). If, however, atrial pressure is above normal (closed circle), the low cardiac output can be attributed to decreased contractility (Dc).

Part Three • Normal Physiology

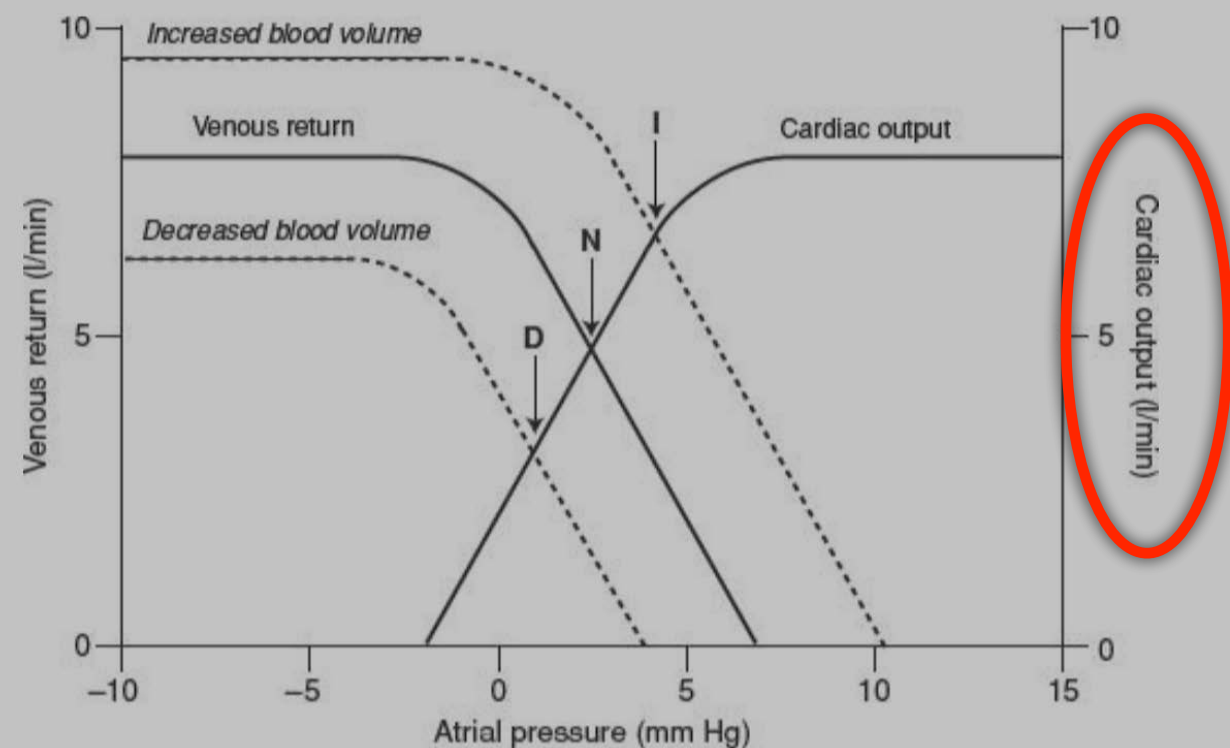




Regulation of Arterial Blood Pressure.

Several systems contribute to the regulation of arterial blood pressure. One of these, the *baroreceptor system*, is a simple and excellent example of a rapidly acting control mechanism (Figure 1-3). In the walls of the bifurcation region of the carotid arteries in the neck, and also in the arch of the aorta in the thorax, are many nerve receptors called *baroreceptors* that are stimulated by stretch of the arterial wall. When the arterial pressure rises too high, the baroreceptors send barrages of nerve impulses to the medulla of the brain. Here these impulses inhibit the *vasomotor center*, which in turn decreases the number of impulses transmitted from the vasomotor center through the sympathetic nervous system to the heart and blood vessels. Lack of these impulses causes diminished pumping activity by the heart and also dilation of the peripheral blood vessels, allowing increased blood flow through the vessels. Both of these effects decrease the arterial pressure, moving it back toward normal.

FIG. 12.27 Curves relating venous return and cardiac output showing effects of changing circulating blood volume. Increased blood volume increases venous return, whereas decreased blood volume reduces venous return. N, normal; I, increased blood volume; D, decreased blood volume.



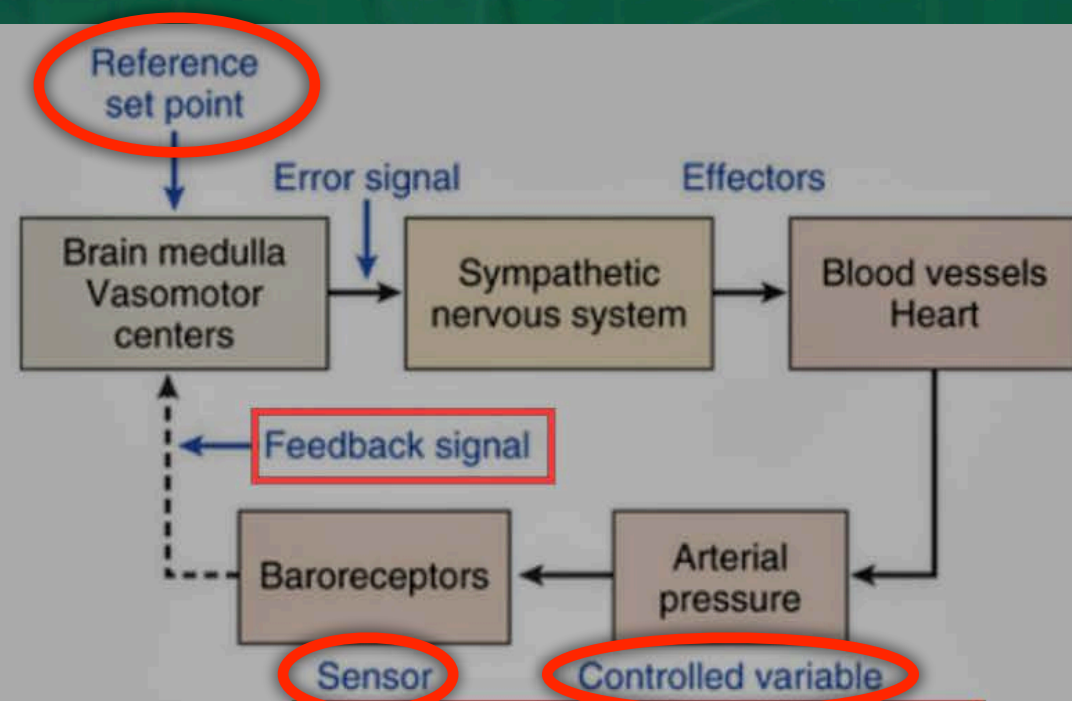


FIGURE 1-3 Negative feedback control of arterial pressure by the arterial baroreceptors. Signals from the sensor (baroreceptors) are sent to medulla of the brain, where they are compared with a reference set point. When arterial pressure increases above normal, this abnormal pressure increases nerve impulses from the baroreceptors to the medulla of the brain, where the input signals are compared with the set point, generating an error signal that leads to decreased sympathetic nervous system activity. Decreased sympathetic activity causes dilation of blood vessels and reduced pumping activity of the heart, which return arterial pressure toward normal.

Unit I Introduction to Physiology: The Cell and General Physiology

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Homeostasis—Maintenance of A Nearly Constant Internal Environment

Control Systems of the Body

Summary—Automaticity of the Body

Bibliography

Unit III The Heart

Chapter 9 Cardiac Muscle: The Heart as a Pump and Function of the Heart Valves

Physiology of Cardiac Muscle

Cardiac Cycle

Regulation of Heart Pumping

Bibliography

Unit IV The Circulation

Chapter 14 Overview of the Circulation; Biophysics of Pressure, Flow, and Resistance

Physical Characteristics of the Circulation

Basic Principles of Circulatory Function

Interrelationships of Pressure, Flow, and Resistance

Bibliography

Chapter 13 Cardiac Arrhythmias and Their Electrocardiographic Interpretation

Abnormal Sinus Rhythms

Abnormal Rhythms that Result from Block of Heart
Signals within the Intracardiac Conduction Pathways

Premature Contractions

Paroxysmal Tachycardia

Ventricular Fibrillation

Atrial Fibrillation

Atrial Flutter

Cardiac Arrest

Bibliography

A physiologist's view of homeostasis

Harold Modell,¹ William Cliff,² Joel Michael,³ Jenny McFarland,⁴ Mary Pat Wenderoth,⁵
and Ann Wright⁶

¹Physiology Educational Research Consortium, Seattle, Washington; ²Department of Biology, Niagara University, Niagara, New York; ³Department of Molecular Biophysics and Physiology, Rush Medical College, Chicago, Illinois; ⁴Department of Biology, Edmonds Community College, Lynnwood, Washington; ⁵Department of Biology, University of Washington, Seattle, Washington; and ⁶Department of Biology, Canisius College, Buffalo, New York

Submitted 20 July 2015; accepted in final form 4 August 2015

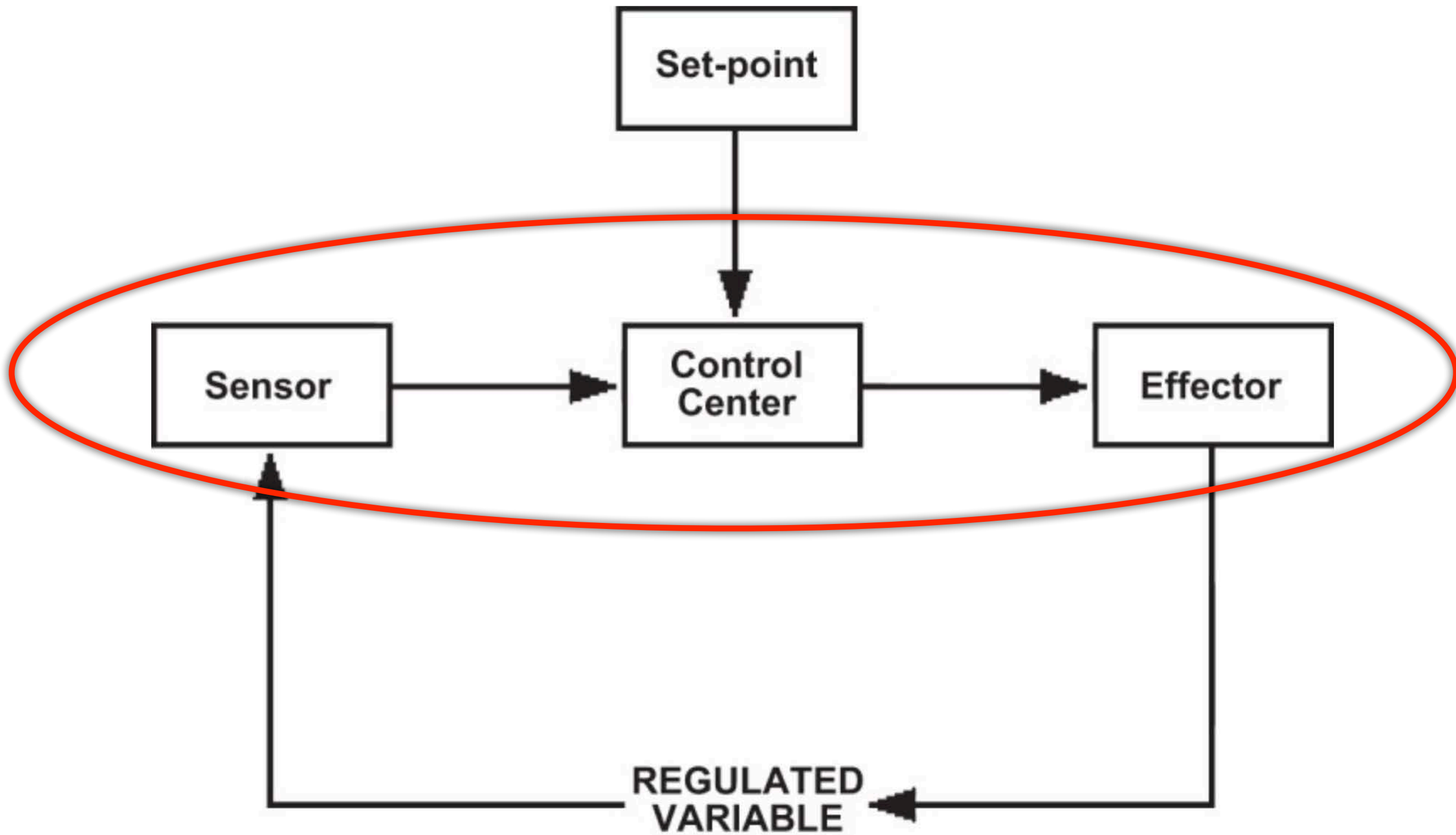
Arthur Guyton was the first major physiology textbook author to include a control systems theory approach in his textbook, and his book included detailed attention to the body's many regulatory mechanisms .

Guyton introduced many students to the concept of homeostasis as an active regulatory mechanism that tended to minimize disturbances to the internal environment.

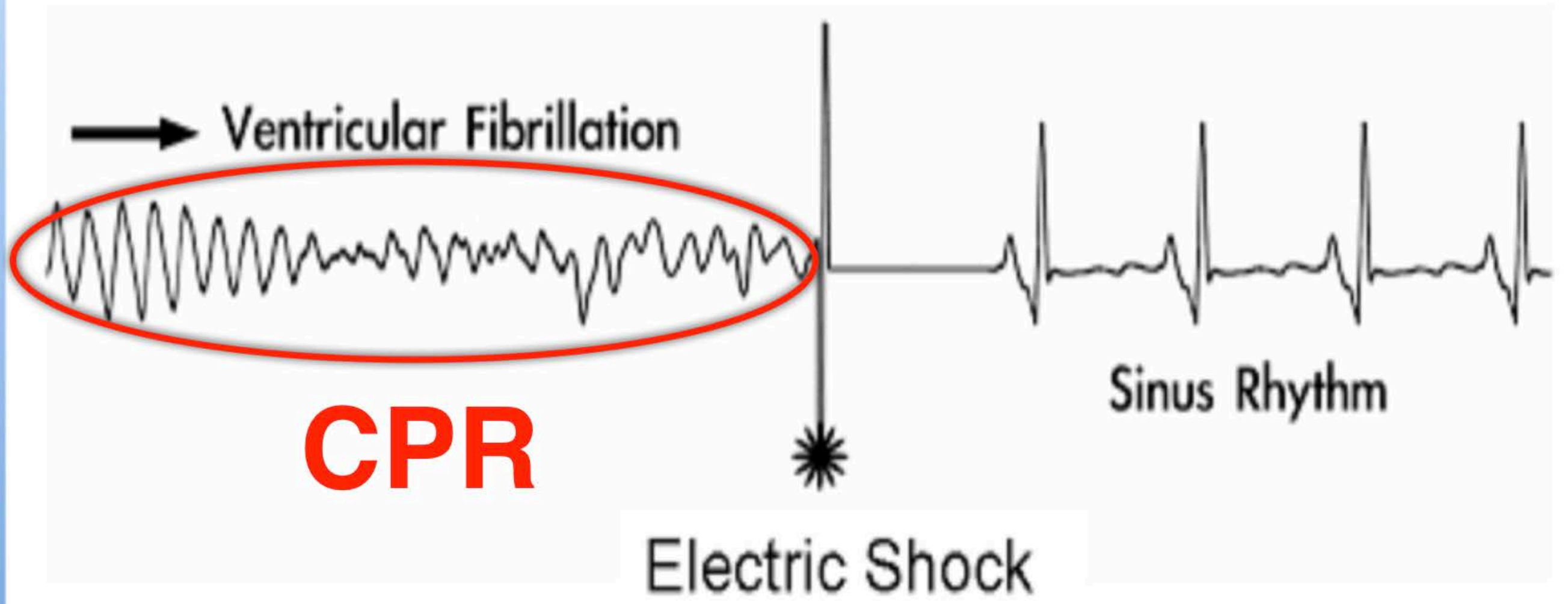
Engineering control systems theory describes a variety of other mechanisms to maintain the stability of a system. five critical components that a regulatory system must contain to maintain homeostasis:

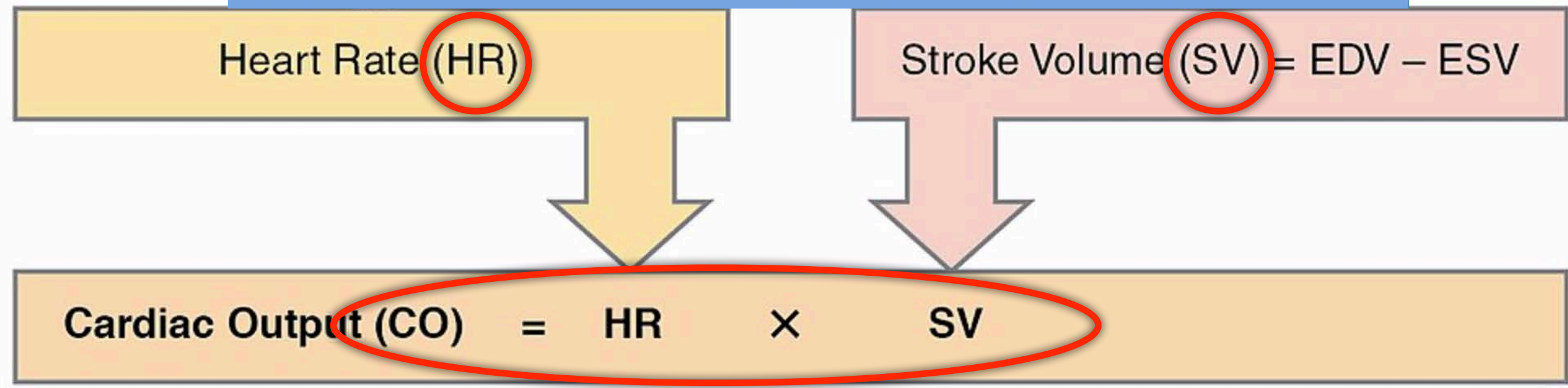
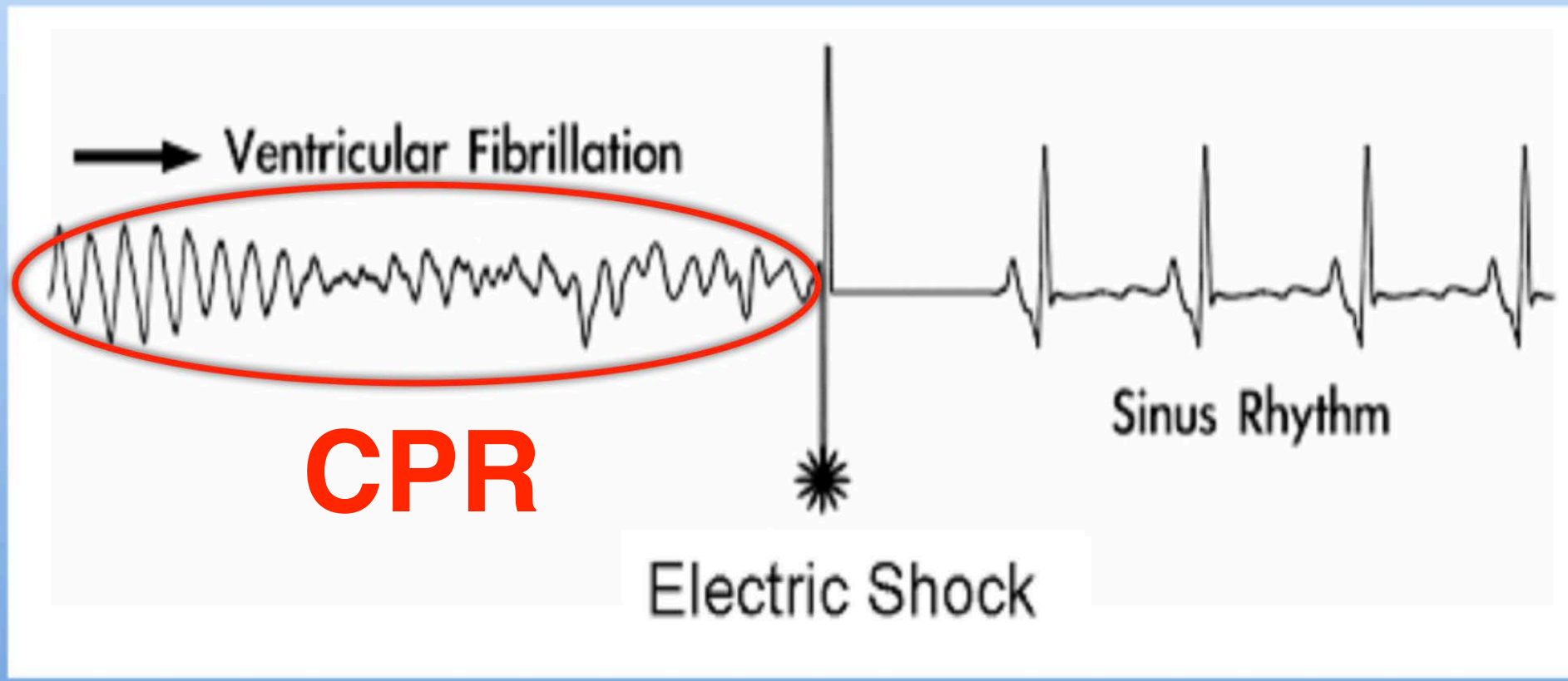
1. It must contain a sensor that measures the value of the regulated variable.
2. It must contain a mechanism for establishing the "normal range" of values for the regulated variable..
3. It must contain an "error detector" that compares the signal being transmitted by the sensor with the set point.
4. The controller interprets the error signal and determines the value of the outputs of the effectors.
5. The effectors are those elements that determine the value of the regulated variable.

Such a system operates in way that causes any change to the regulated variable, a disturbance, to be countered by a change in the effector output to restore the regulated variable toward its set point value.



Simplified representation of a homeostatic regulatory system. Several components shown in [Fig. 1](#) are combined in this representation.





Disclosures:

... only one:

(to explain use of metaphor, allegory, aphorisms, and dramatic video)

Disclosure:

In fall of 2010, I served as chair of an *ad hoc* committee at Birmingham's Trinity Medical Center, charged with developing an order set for therapeutic hypothermia (TH) for patients who were comatose following resuscitation from SCA.

On February 2, 2011, I sent physicians an email announcing availability of the TH protocol:

From: Jim Boogaerts
Sent: Wednesday, February 02, 2011 11:40 PM
To: Physicians All
Cc: NPs Montclair; NPs Brookwood
Subject: Therapeutic Hypothermia at Trinity

Therapeutic Hypothermia treatment for appropriately selected patients who survive cardiac arrest is now available at Trinity Medical Center.

Please take this opportunity to review the attachments and the recently published guidelines for post-resuscitative care. As with any newly-enacted protocol, it will be important for physicians to have understanding of the process, protocol, and order set before actual use.

Thanks,

Jim

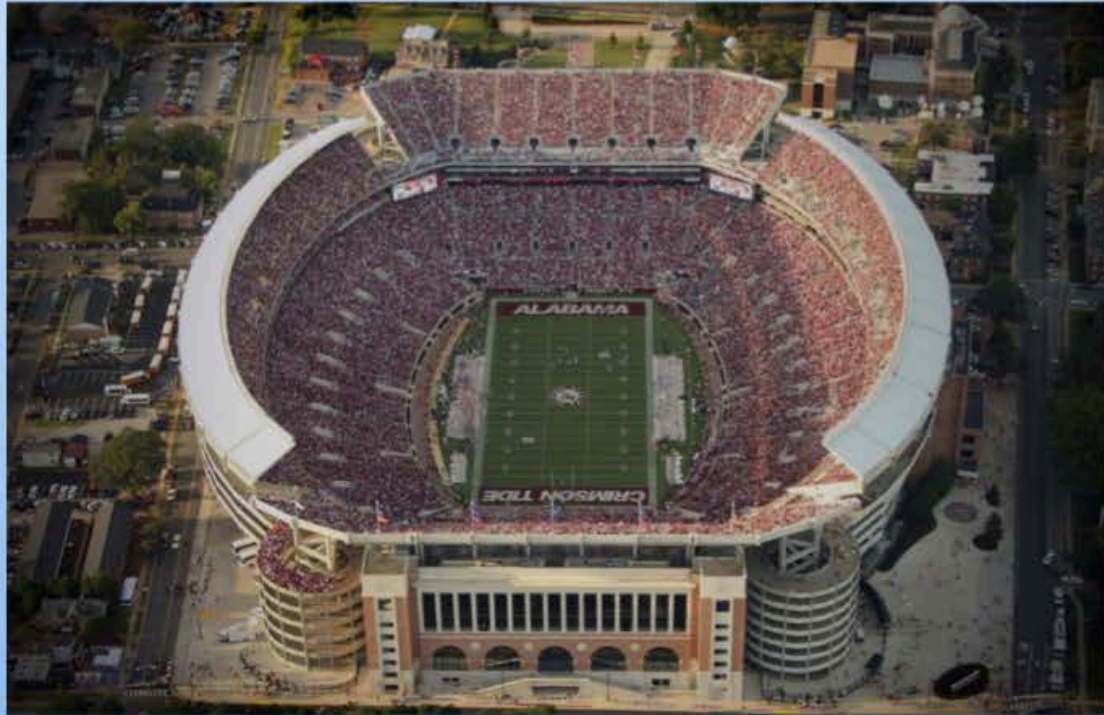
Six months later, on August 2, 2011, I suffered a VF cardiac arrest as I entered the hospital from the physicians' parking lot.

My body was discovered in the entrance hall after about 5 minutes (per security camera footage) and I received prolonged CPR (administered by an anesthesiologist).

With ongoing CPR, I was transported to the 2nd floor CCU. After multiple defibrillatory shocks, I had ROSC, ... and was the 5th patient at our hospital to receive therapeutic hypothermia.

My recovery was complete.

Sudden Cardiac Arrests in US every year (out-of-hospital & in-hospital): > 400,000



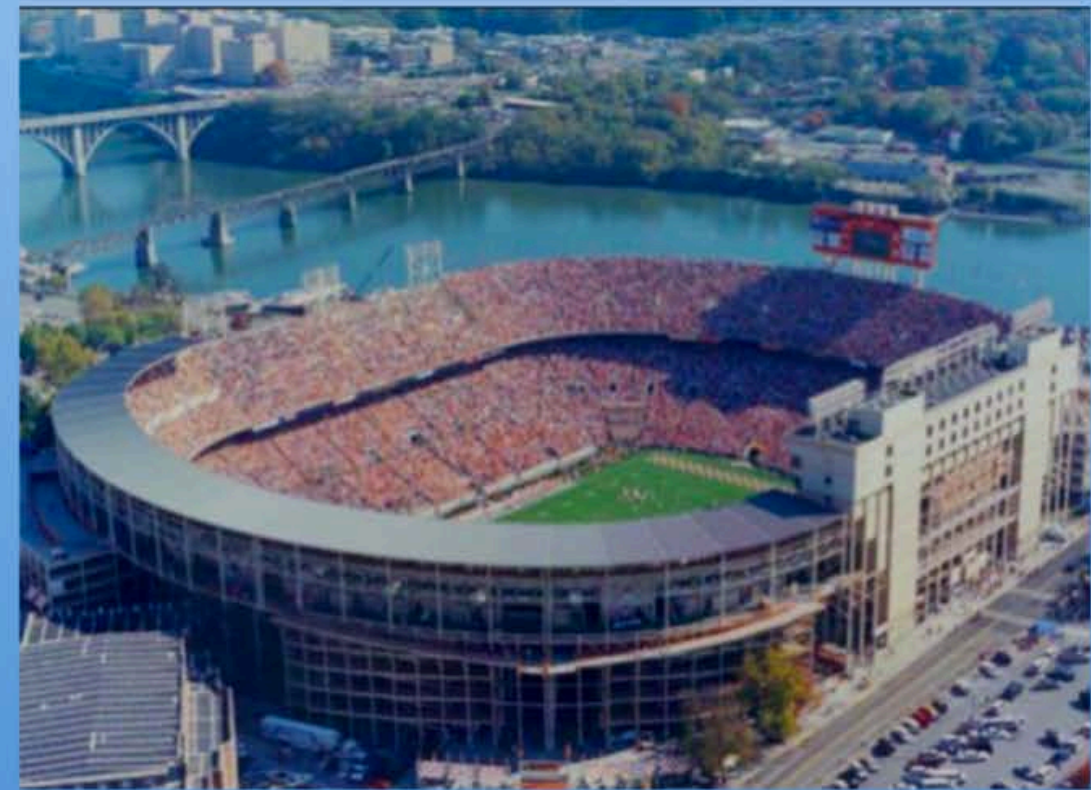
Bryant-Denny Stadium: 101,821



Jordan-Hare Stadium: 87,451



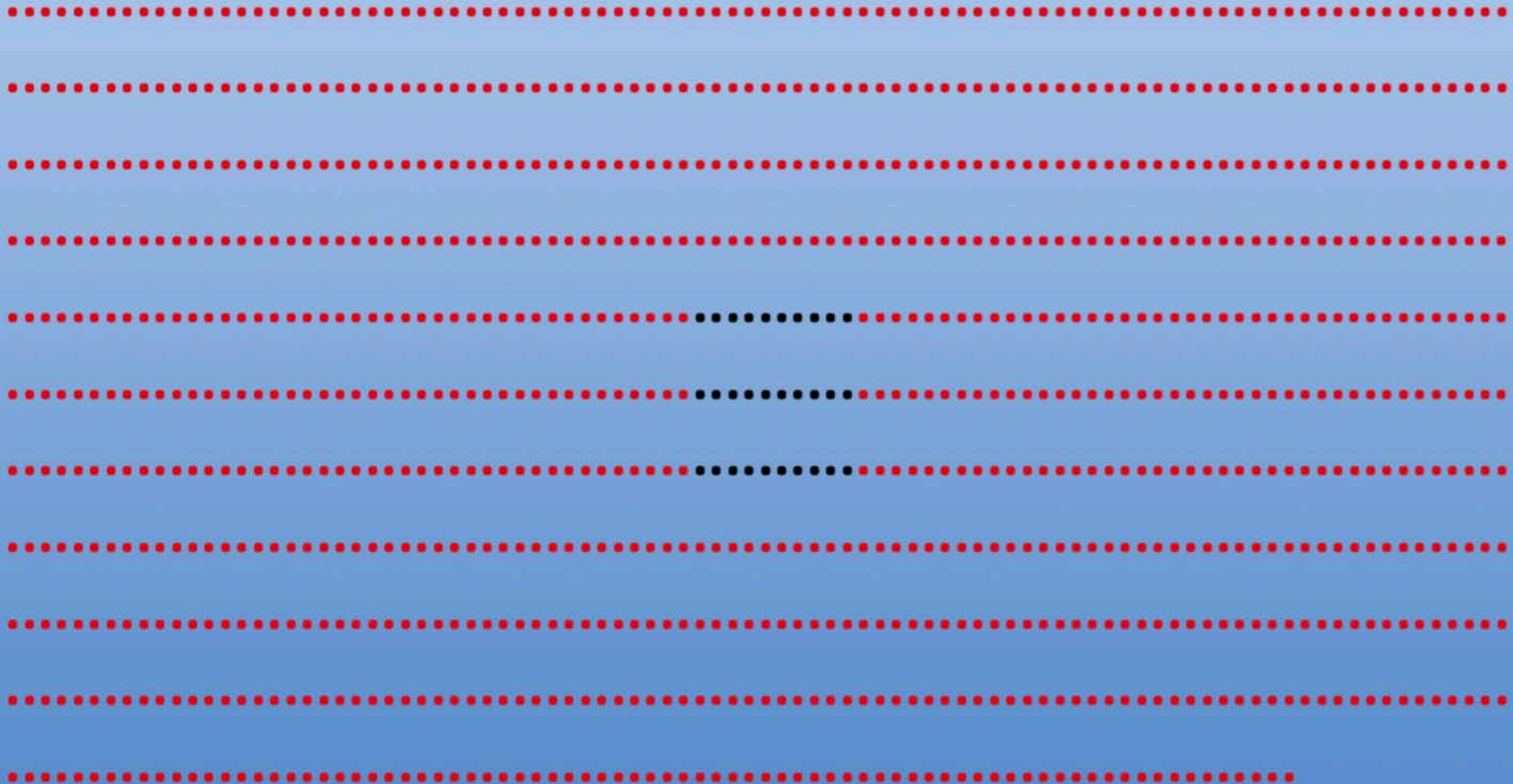
Tiger Stadium: 102,321



Neyland Stadium: 102,455

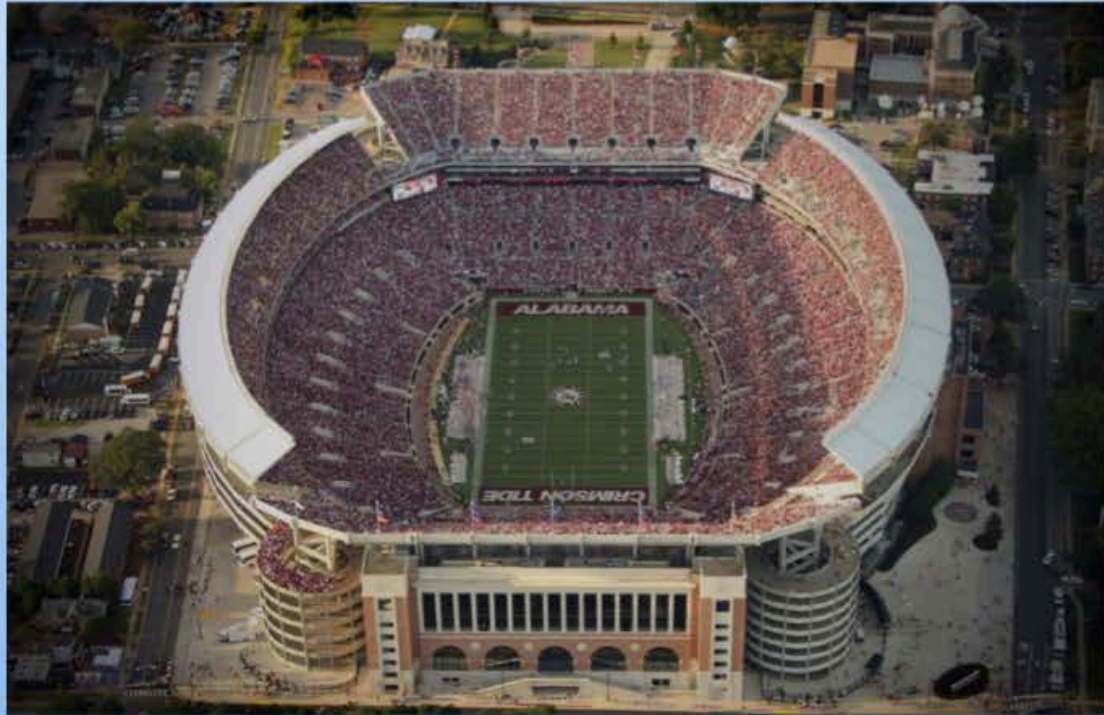
Here's the *status quo*:

3 % survival with good neuro status (.) :



[1,000 dots: each represents 400 individuals.]

Sudden Cardiac Arrests in US every year (out-of-hospital & in-hospital): > 400,000



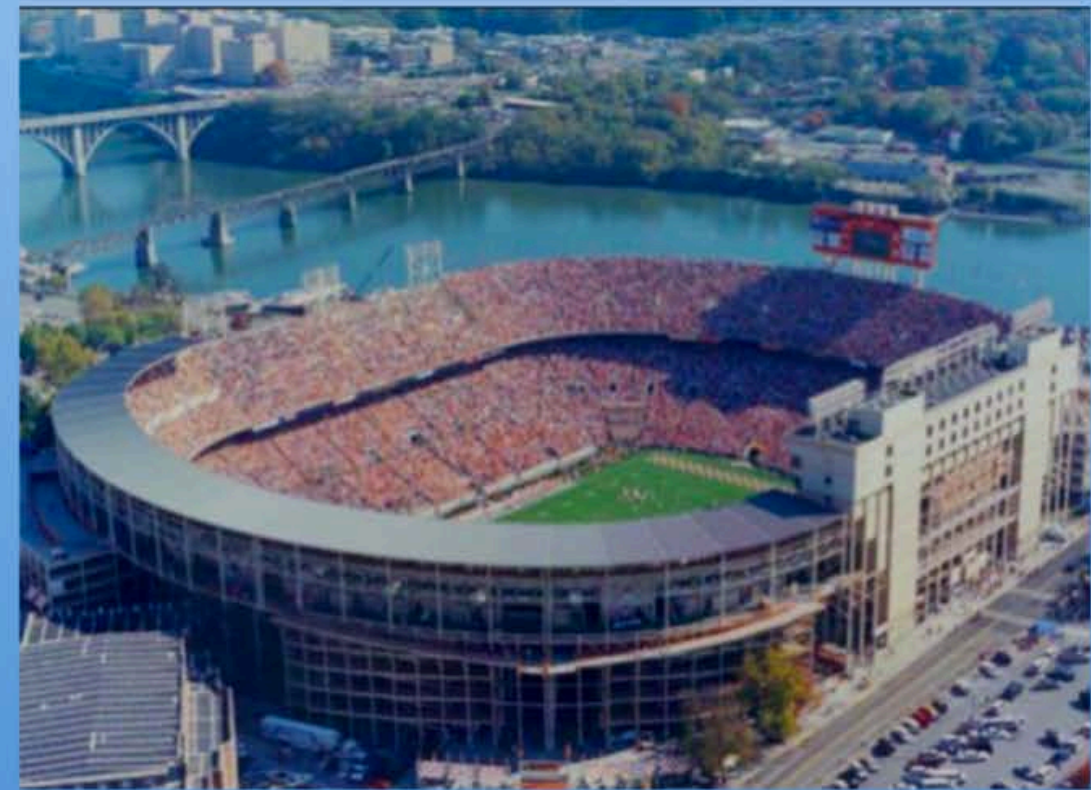
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Jordan-Hare Stadium: 87,451



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Neyland Stadium: 102,455

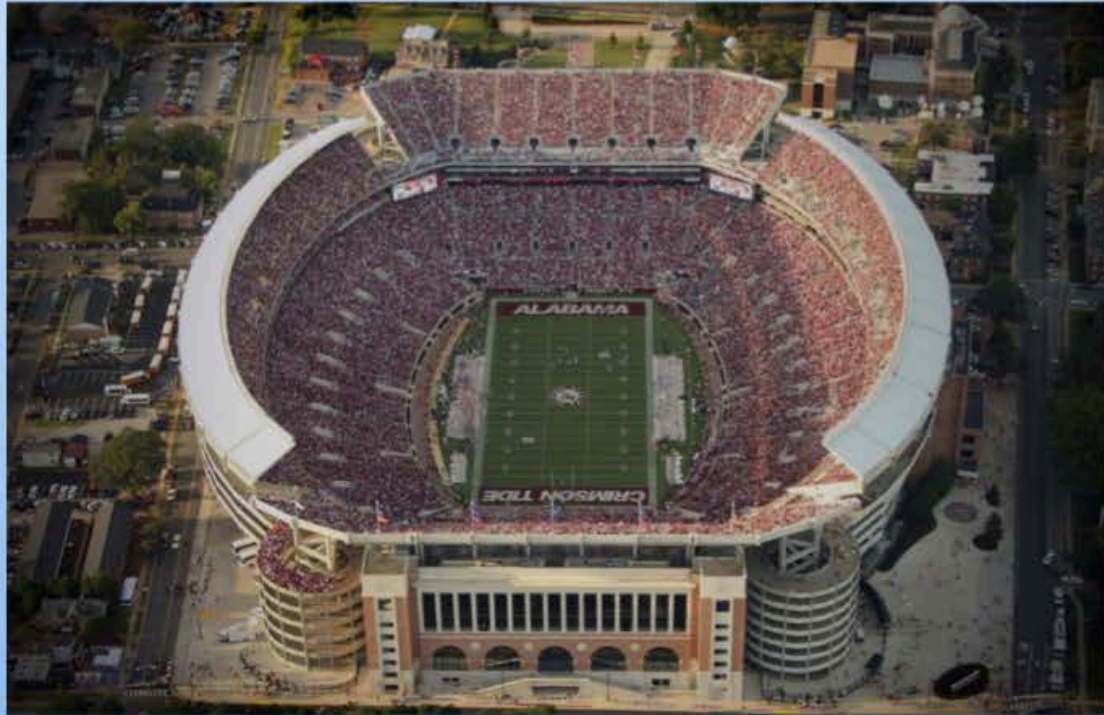
Here's an initial goal - currently achievable when ideal process is in place:

~ 30 % survival with good neuro status (.) :



[1,000 dots: each represents 400 individuals.]

Sudden Cardiac Arrests in US every year (out-of-hospital & in-hospital): > 400,000



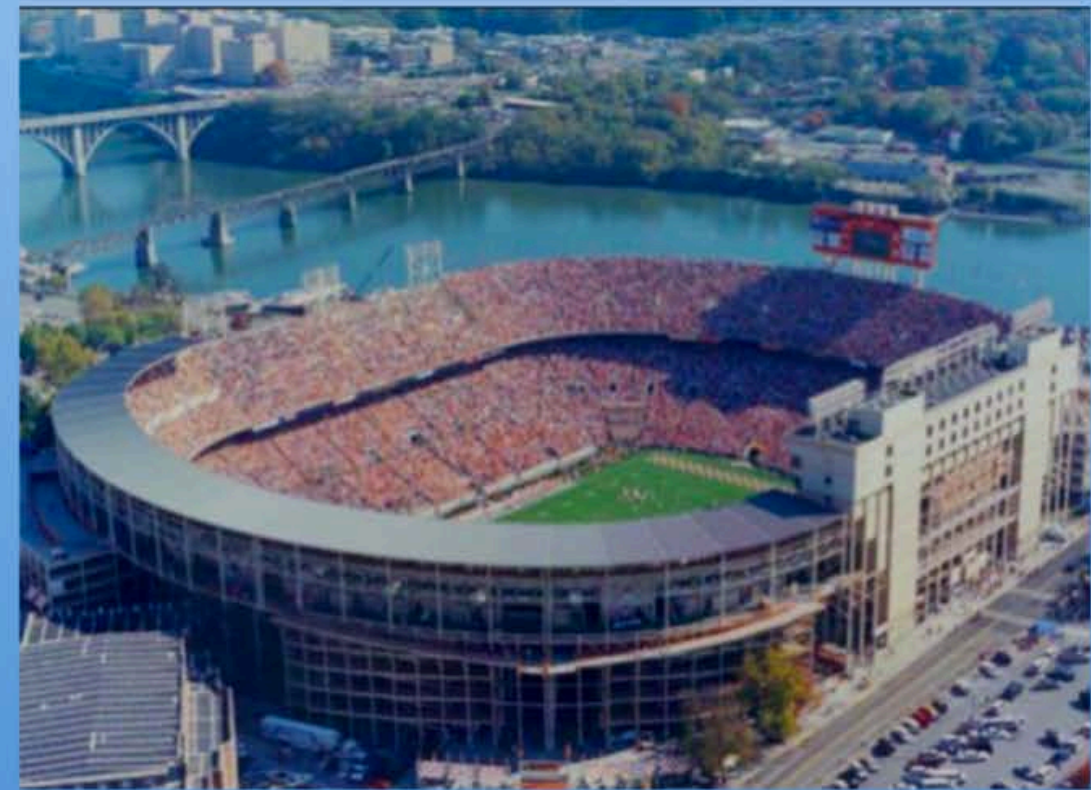
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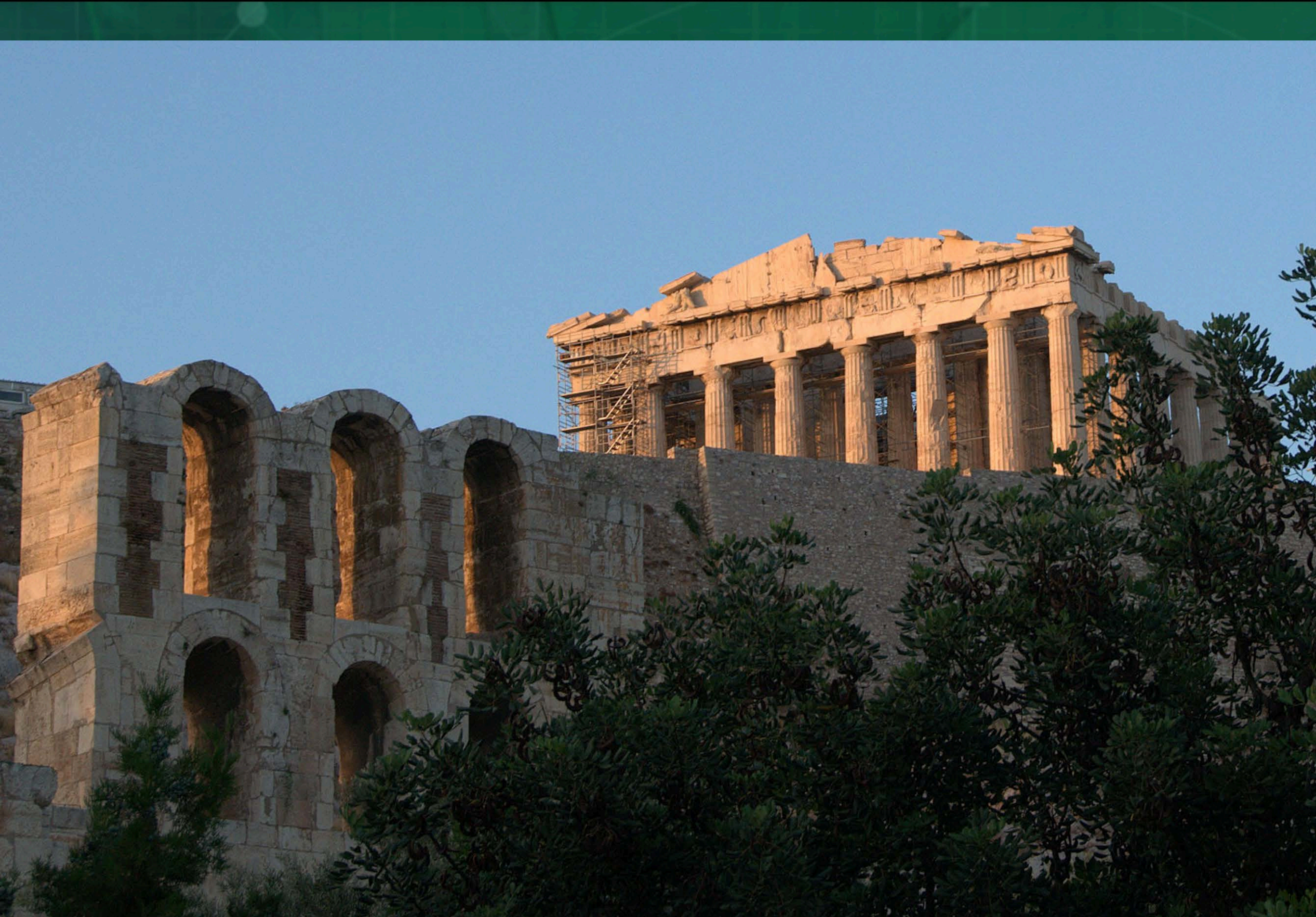
Jordan-Hare Stadium: 87,451



Tiger Stadium: 102,321



Neyland Stadium: 102,455





**In Greek mythology,
Prometheus**

**(Προμηθεύς - meaning "Forethought")
is a Titan,**

credited with the creation of man from clay.

**He defied the Olympian gods by
stealing fire and giving it to humanity,
an act that enabled progress and
civilization.**

Chorus

Did you perhaps go farther than you have told us?

Prometheus

Yes, I stopped mortals from foreseeing doom.

Chorus

What cure did you discover for that sickness?

Prometheus

I sowed in them blind hopes.

(Re: our perception of the mortal state)



Προμηθεύς Δεσμώτης
Prometheus Bound
Aeschylus - 430 BC

CHORUS: Whoever feels no pity for you, Prometheus, must be made of stone and he must have a heart of steel. I had no need to witness your pain, but now that I did, my heart breaks for you.

PROMETHEUS: Yes, I am a pitiful sight to my friends.

CHORUS: You haven't, by any chance, committed a further offense?

PROMETHEUS: Yes, I've made it so that humans cannot foresee their own death.

CHORUS: What sort of medicine did you use for this?

PROMETHEUS: I have filled their hearts with blind hopes.

CHORUS: That is a great gift you have given them, Prometheus.

PROMETHEUS: And even more than that, I have given them the gift of fire.

CHORUS: So those ephemeral creatures now possess the bright-faced fire?

PROMETHEUS: Yes and with it they will learn many crafts.



Προμηθεύς Δεσμώτης
Prometheus Bound
Aeschylus - 430 BC

The Olympian god Hephaestus (Vulcan) forged the steel chain and bound Prometheus to the mountain peak.

**“Here we are,
Hephaestus [*Vulcan*],
at the very limits of the
Earth. Now it’s your job
to do what Father (Zeus)
has ordered to be done
to this terrible rebel. Use
these unbreakable steel
chains and shackle him
to this high peak.**

**He stole the very
blossom of your craft,
*the blazing flame, the
very spark of every art,*
and gave it to the
mortals.”**





“Prometheus, teacher in every art, brought the fire that hath proved to mortals a means to mighty ends.”



The NEW ENGLAND
JOURNAL of MEDICINE

SPECIALTIES

TOPICS

MULTIMEDIA

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◀ PREVIOUS ISSUE

ARCHIVE

April 19, 1956

VOL. 254 NO. 16

ORIGINAL ARTICLES

Termination of Ventricular Fibrillation in Man by Externally Applied Electric Countershock

P.M. Zoll, A.J. Linenthal, W. Gibson, M.H. Paul, and L.R. Norman

VENTRICULAR fibrillation is usually a rapidly fatal arrhythmia that may occur in cardiac patients, in any patient under anesthesia and in drowning and electrocution. In cardiac patients it is a frequent cause of sudden death in the course of coronary...

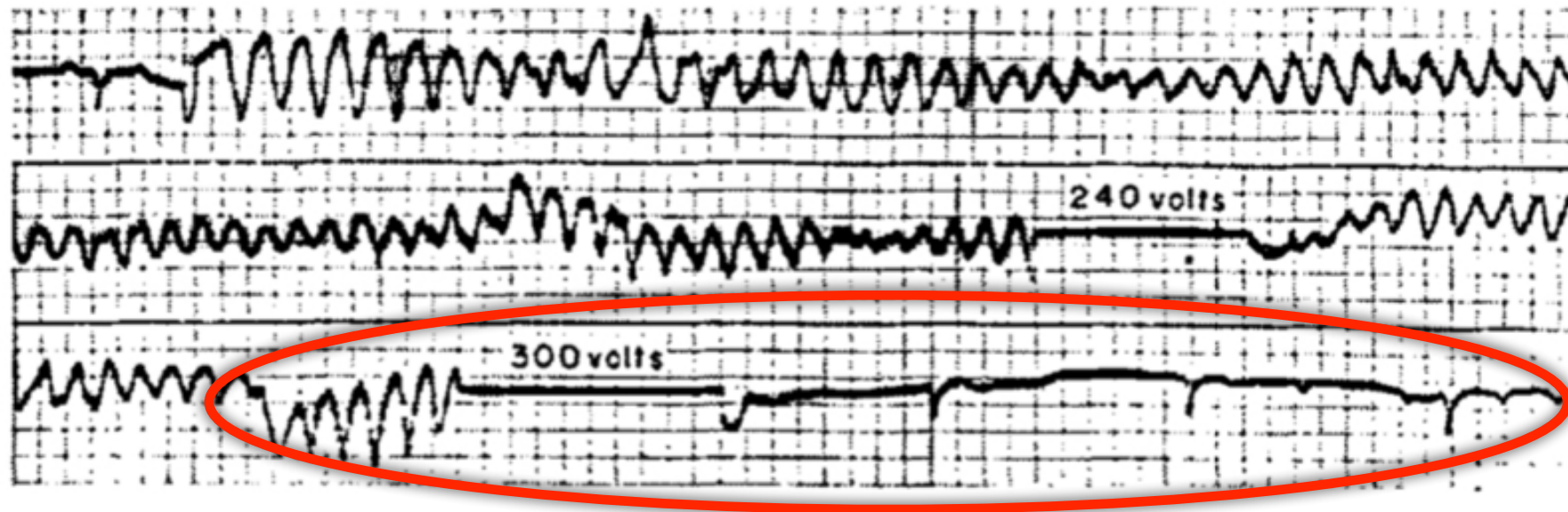


FIG. 4.—Continuous tracing showing the usual idioventricular pacemaker, ventricular fibrillation, ineffective AC countershock of 240 volts and successful countershock of 300 volts with return of idioventricular rhythm. (Reproduced by permission of the New England J. Med. 262:108, 1960.)

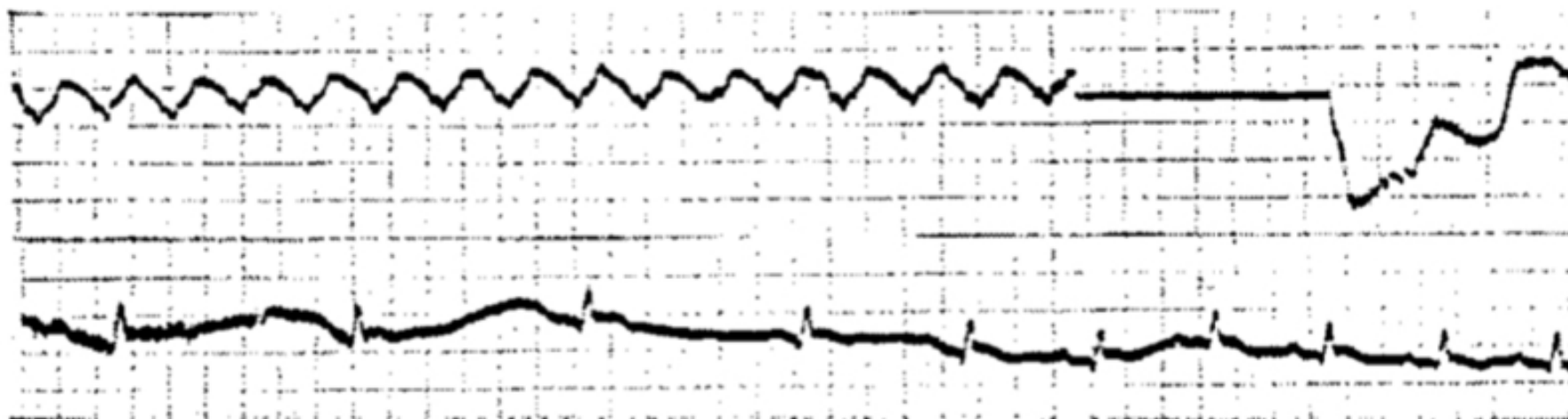


FIG. 5.—Continuous tracing showing successful termination of ventricular tachycardia (167 per min.) by 450-volt AC countershock followed by a few A-V nodal beats and then normal sinus rhythm.

PROGRAM FOR CARDIAC ARREST

Equally important to the success of resuscitation is the prompt institution of a pre-arranged and well rehearsed program of action (Table 2). The emergency restoration of circulation may involve external electric stimulation for ventricular standstill, external electric countershock for fibrillation, manual blows to the precordium, external cardiac massage, cardiac puncture and, in appropriate circumstances, even thoracotomy with direct cardiac massage. The sequence of

TABLE 2. PROGRAM FOR CARDIAC ARREST

PROGRAM FOR CARDIAC ARREST

1. *Emergency restoration of circulation*

Precordial blow

External electric stimulation or countershock

External cardiac massage

Cardiac puncture and intracardiac epinephrine

Thoracotomy and massage

if other methods fail

if adequate help is available

if prognosis is favorable:

no irreversible cerebral damage

general condition good

Artificial respiration

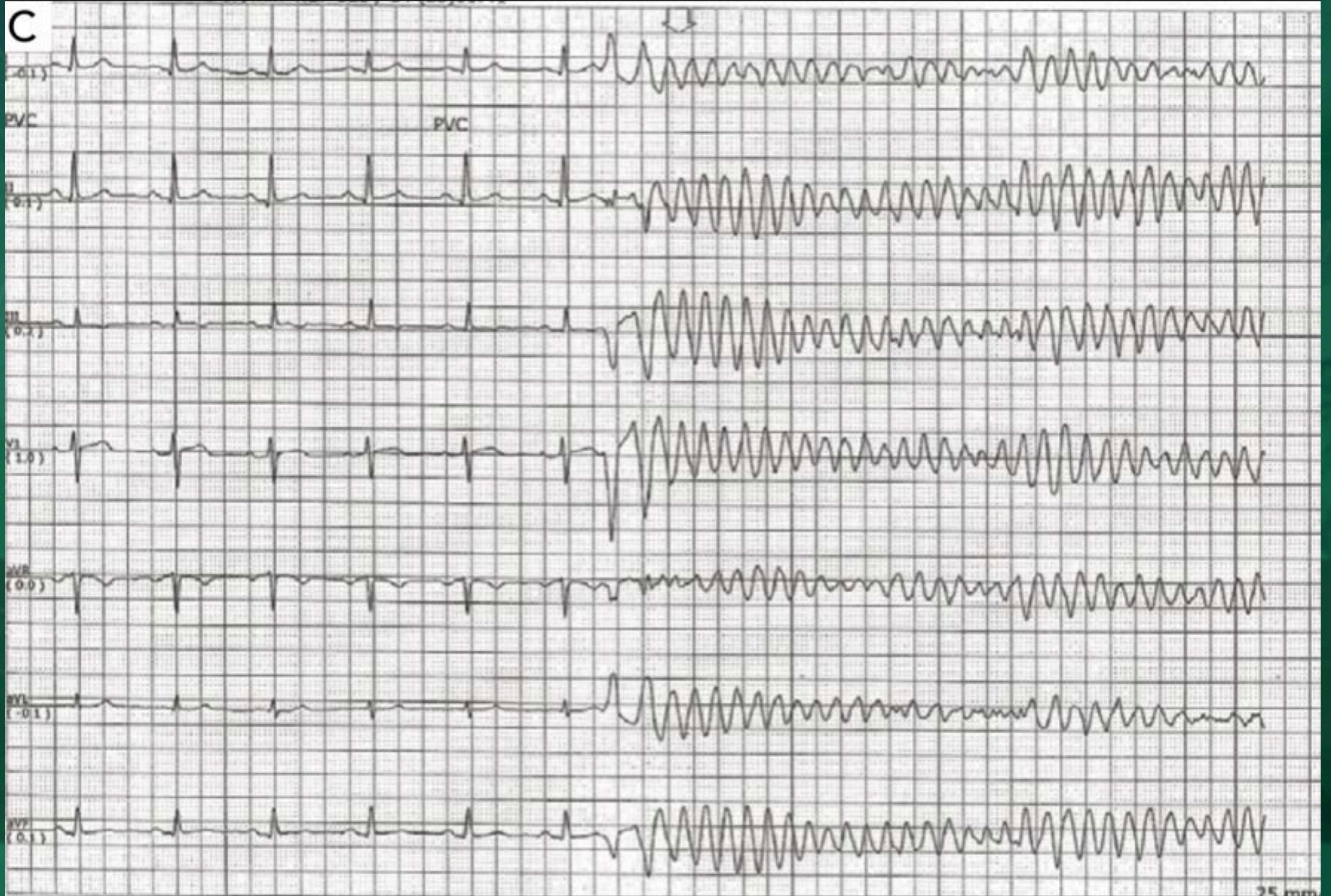
2. *Restoration of intrinsic cardiac rhythm*

Electrocardiograph or monitor

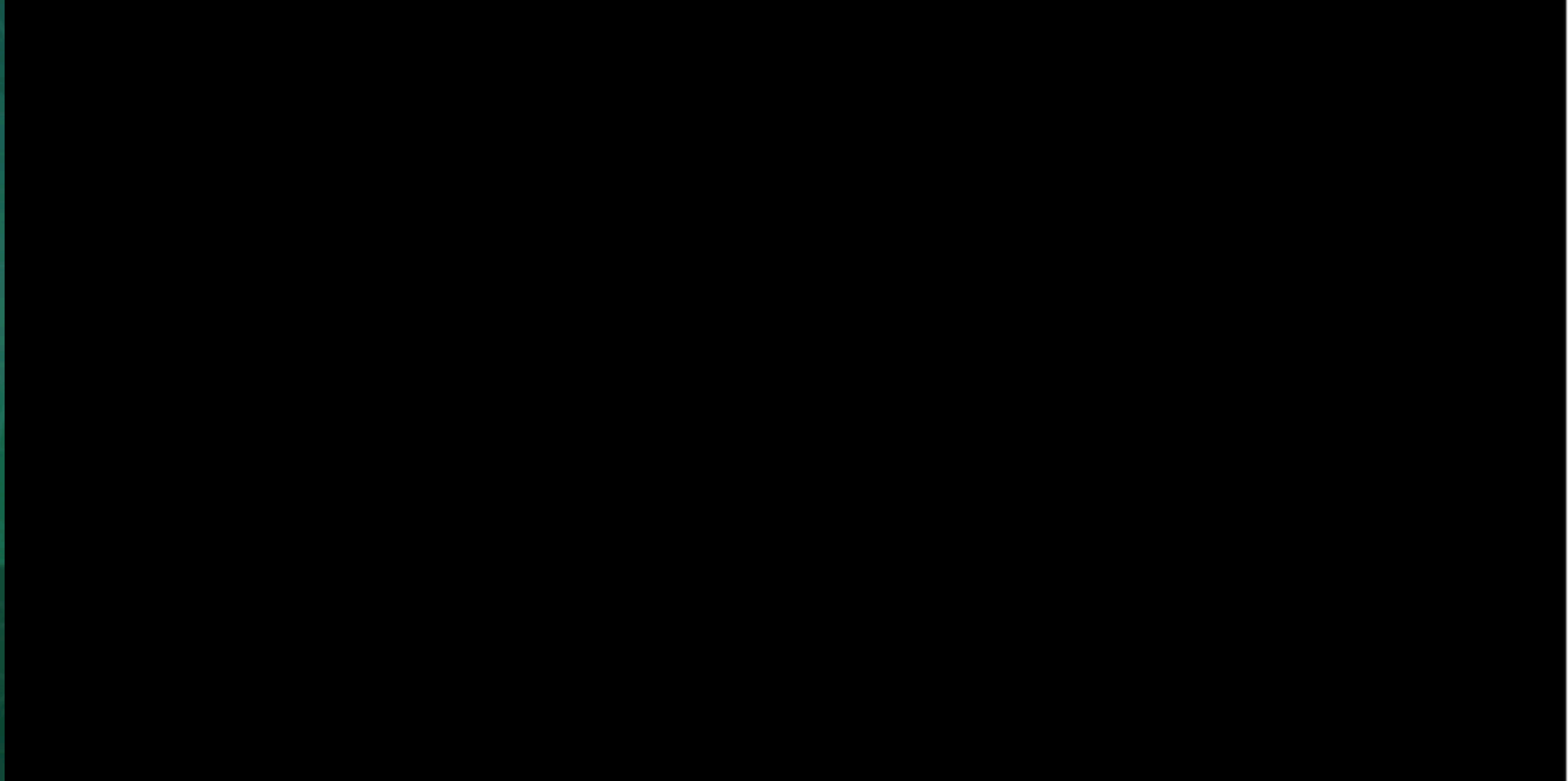
Electric stimulation or countershock

Drugs: epinephrine, isoproterenol, procainamide, calcium salts,

norepinephrine, sodium bicarbonate







0:00 / 14:24



*Control of Heart Action
by Electrical
and Mechanical Means*

PAUL M. ZOLL
ARTHUR J. LINENTHAL

1966

Paul M. Zoll

is Clinical Professor of Medicine, Harvard Medical School, from which institution he received his M.D., and Visiting Physician, Beth Israel Hospital, Boston. He is also Consultant in Cardiology at the Children's Hospital Medical Center in Boston. Dr. Zoll has been a frequent contributor to the literature, having published more than 60 articles on cardiovascular disease. He is an Associate Editor of the journal *Circulation*.

Arthur J. Linenthal

is Associate Clinical Professor of Medicine, Harvard Medical School, having also received his M.D. from that institution, and Visiting Physician, Beth Israel Hospital, Boston. He is also Consultant in Medicine at the Children's Hospital Medical Center in Boston. Dr. Linenthal's contributions to the literature have also been extensive, with more than 50 articles on cardiovascular disease to his credit. He is a member of the Editorial Board of *Circulation*.

A close-up portrait of Michael Kuster, a paramedic, wearing a red jacket. He is speaking and looking slightly off-camera. The background is a blurred hospital setting with other people in white coats.

MICHAEL KUSTER

PARAMEDIC AIR ZERMATT

Chest compressions are what save their lives.





**Dr. Axel Mann,
anesthesiologist
Emergency Medicine
Senior Medic, *Air Zermatt***



**Dr. Axel Mann,
anesthesiologist
Emergency Medicine
Senior Medic, *Air Zermatt***





AL - 74 Female

- OOHVF Arrest 10:23
- Bystander CPR
- 2 shocks from AED with ROSC
- *Pre-hospital ECG: STEMI 1045*
- PEA arrest in ambulance
- Autopulse



0:00 / 1:25



ANATOMICAL STUDIES
ON
THE MOTION
OF THE
HEART AND BLOOD

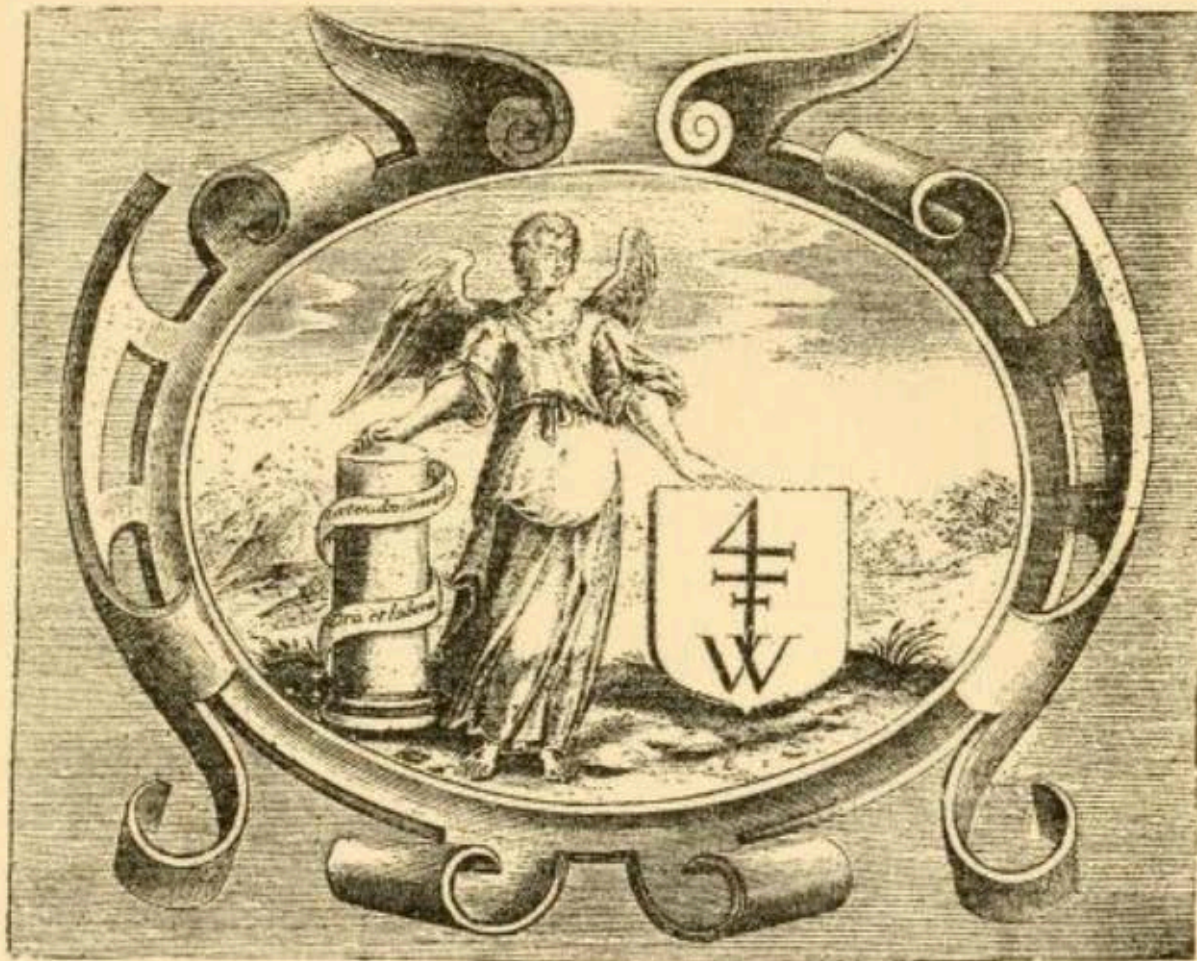


WILLIAM HARVEY, M.D.

1628

EXERCITATIO,
ANATOMICA DE
MOTV CORDIS ET SAN-
GVINIS IN ANIMALI.

BVS,
GVILIELMI HARVEI ANGLI,
*Medici Regii, & Professoris Anatomie in Col-
legio Medicorum Londinensi.*



FRANCOFVRTI,
Sumptibus GVILIELMI FITZERI.
ANNO M. DC. XXVIII.



*To The Most Illustrious and Indomitable
Prince CHARLES, KING of GREAT BRIT-
AIN, FRANCE and IRELAND, DEFENDER of
the FAITH*

MOST ILLUSTRIOUS PRINCE!

The heart of animals is the foundation of their life, the sovereign of everything within them, the sun of their microcosm, that upon which all growth depends, from which all power proceeds. The King, in like manner, is the foundation of his kingdom, the sun of the world around him, the heart of the republic, the fountain whence all power, all grace doth flow. What I have here written of the motions of the heart I am the more emboldened to present to your Majesty, according to the custom of the present age, because almost all things human are done after human examples, and many things in a King are after the pattern of the heart. The knowledge of his heart, therefore, will not be useless to a Prince, as embracing a kind of Divine example of his functions,—and it has still been usual with men to compare small things with great. Here, at all

To Build a Fire



DAY HAD DAWNED COLD AND GRAY WHEN

the man turned aside from the main Yukon trail. He climbed the high earth-bank where a little-traveled trail led east through the pine forest. It was a high bank, and he paused to breathe at the top. He excused the act to himself by looking at his watch. It was nine o'clock in the morning. There was no sun or promise of sun, although there was not a cloud in the sky. It was a clear day. However, there seemed to be an indescribable darkness over the face of things. That was because the sun was absent from the sky. This fact did not worry the man. He was not alarmed by the lack of sun. It had been days since he had seen the sun.

Jack London
1908

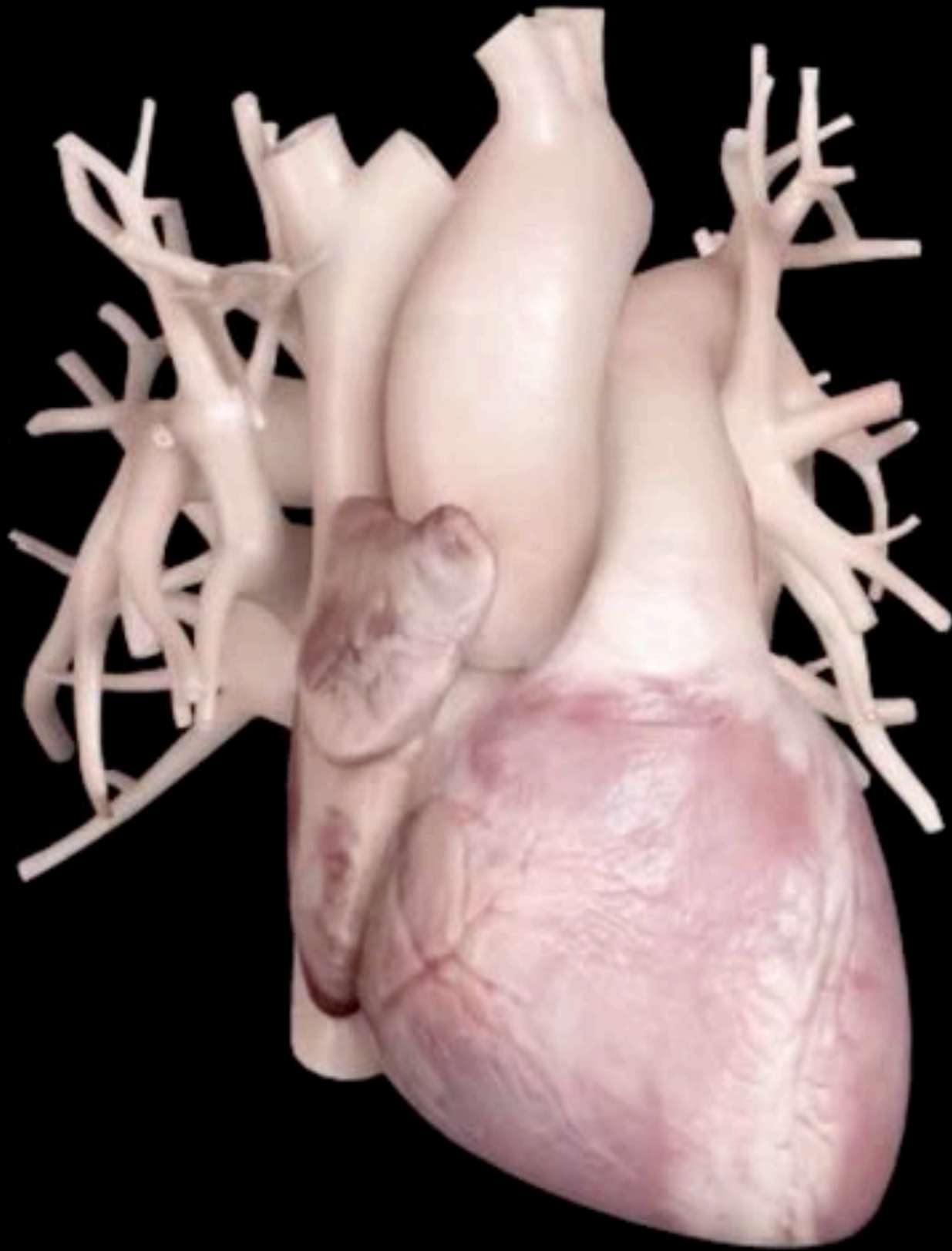


“ When it is seventy five below zero, a man must not fail in his first attempt to build a fire – that is, if his feet are wet. ” - Jack London, *To Build a Fire* - full text

(Re: delivery of resuscitative efforts in sudden cardiac arrest)

W. Edwards Deming has taught us:

*The real secret of improving quality is to enlist the efforts of willing workers to do things properly the first time and to **give them the right tools.***



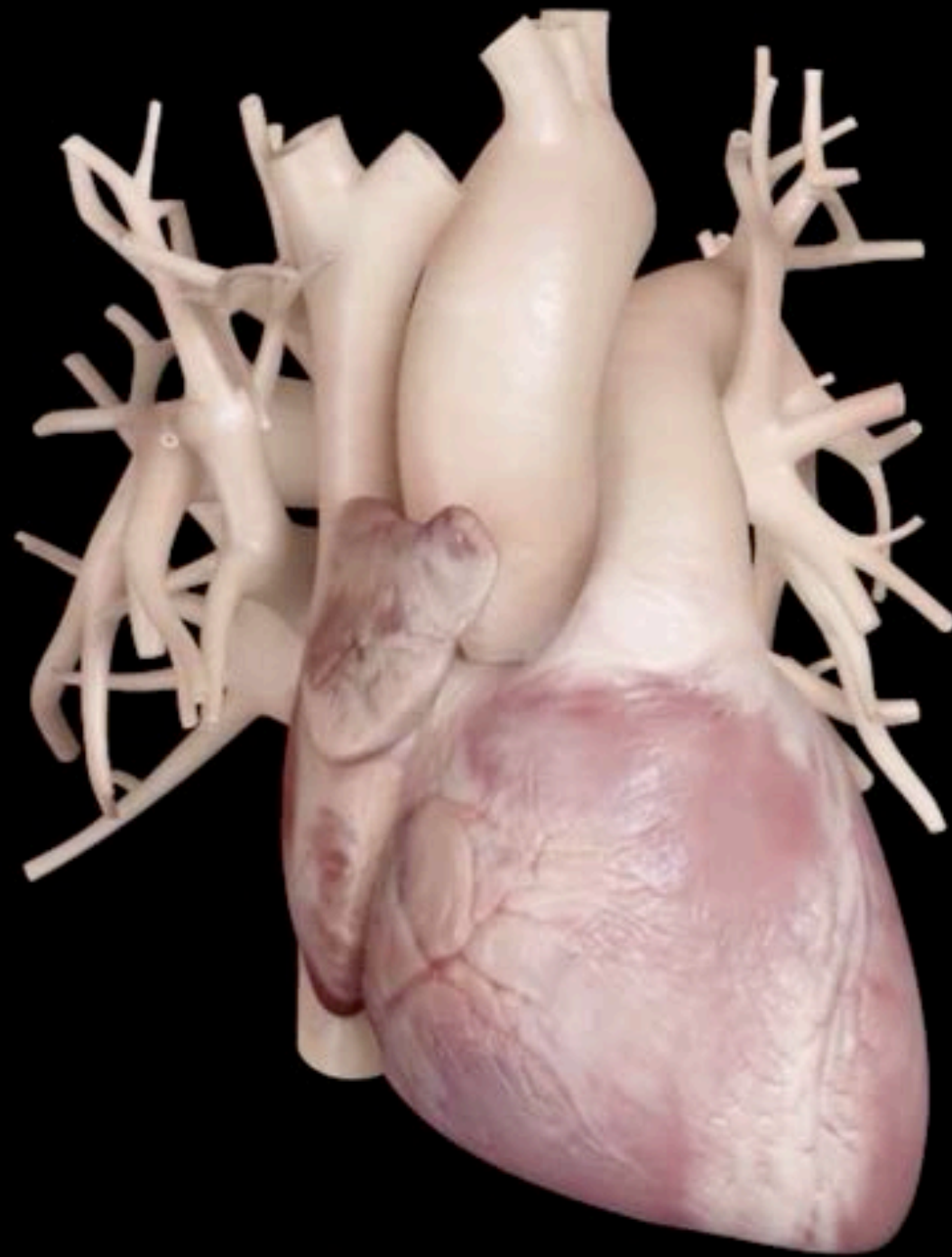
“*patient*”

from Latin verb: *patior, pati, passus sum*:

to suffer

“*one who suffers*”





3D4MEDICAL
HEART PRO III

UAB THE UNIVERSITY OF
ALABAMA AT BIRMINGHAM

When you encounter difficulties, you need to be optimistic.

The pessimists tend to die.

... quote from **Zhou Yougang** - father of **Pinyin** writing,
who died at 111 (from 1/14/17 **NYT** obit)

What is cardiac arrest?

Cardiac arrest is the abrupt loss of heart function in a person who may or may not have been diagnosed with heart disease. It can come on suddenly, or in the wake of other symptoms. Cardiac arrest is often fatal, if appropriate steps aren't taken immediately.

Each year in the United States, more than 350,000 cardiac arrests occur outside of a hospital setting.

Is a heart attack the same as cardiac arrest?

No. The term "heart attack" is often mistakenly used to describe cardiac arrest. While a heart attack may cause cardiac arrest, the two terms don't mean the same thing.

Heart attacks are caused by a blockage that stops blood flow to the heart. A heart attack (or myocardial infarction) refers to death of heart muscle tissue due to the loss of blood supply. Heart attack can be understood as a "circulation" problem. A heart attack is quite serious, sometimes fatal.

By contrast, cardiac arrest is caused when the heart's electrical system malfunctions. The heart stops beating properly. Hence the name: The heart's pumping function is "arrested," or stopped.

In cardiac arrest, death can result quickly if proper steps aren't taken immediately. Cardiac arrest may be reversed if CPR (cardiopulmonary resuscitation) is performed and a defibrillator is used to shock the heart and restore a normal heart rhythm within a few minutes.

Cardiac arrest may be caused by irregular heart rhythms, called arrhythmias. A common arrhythmia associated with cardiac arrest is ventricular fibrillation. Ventricular fibrillation means that the heart's lower chambers suddenly start beating chaotically and don't pump blood.



WHY CPR?

When cardiac arrest strikes, the heart stops. Completely. Hard and fast chest compressions can move some blood through the heart into the rest of the body. This can save a life.

How CPR Works

Cardiac Pump:

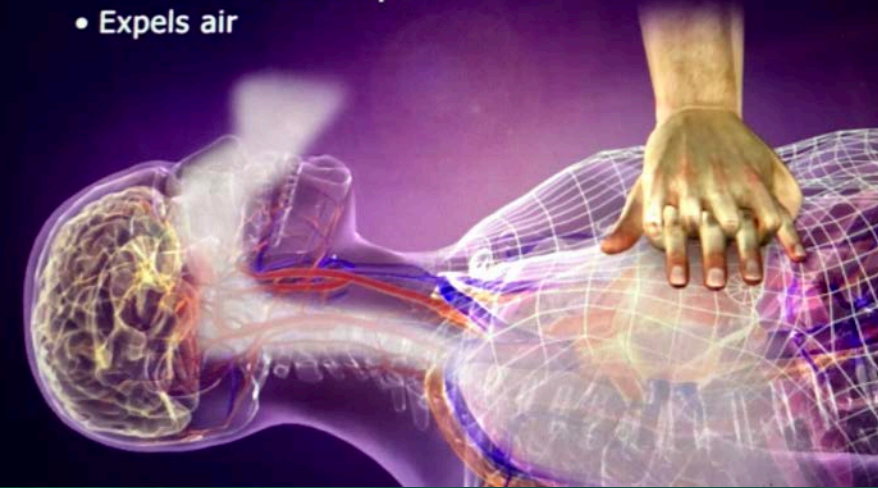
Direct compression forces blood out



Thoracic Pump - Compression

POSITIVE pressure:

- Creates cardiac output
- Expels air



Thoracic Pump - Decompression

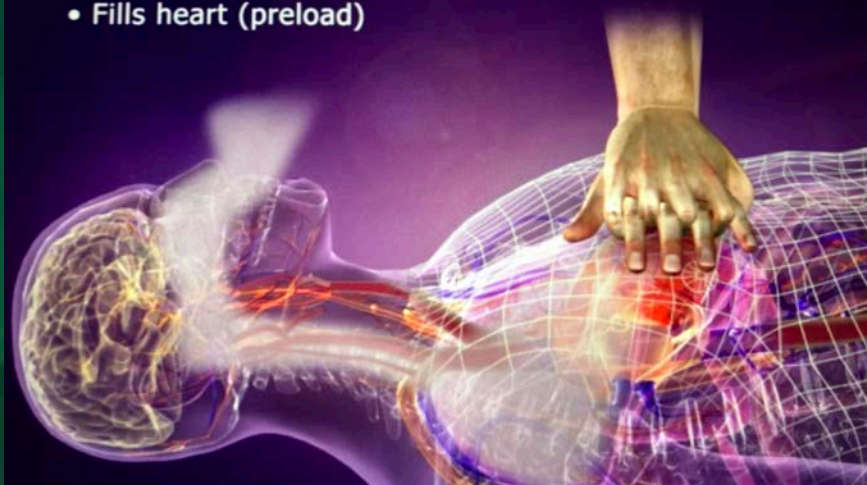
NEGATIVE pressure (vacuum):



Thoracic Pump - Decompression

NEGATIVE pressure (vacuum):

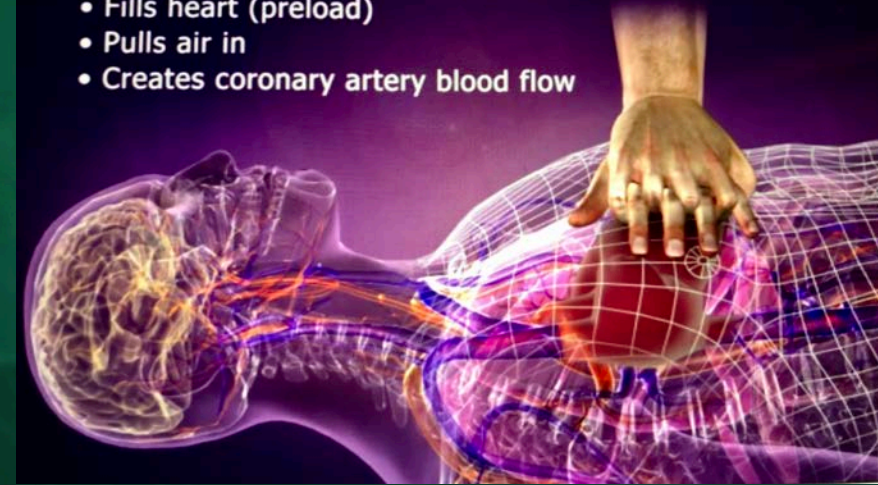
- Fills heart (preload)



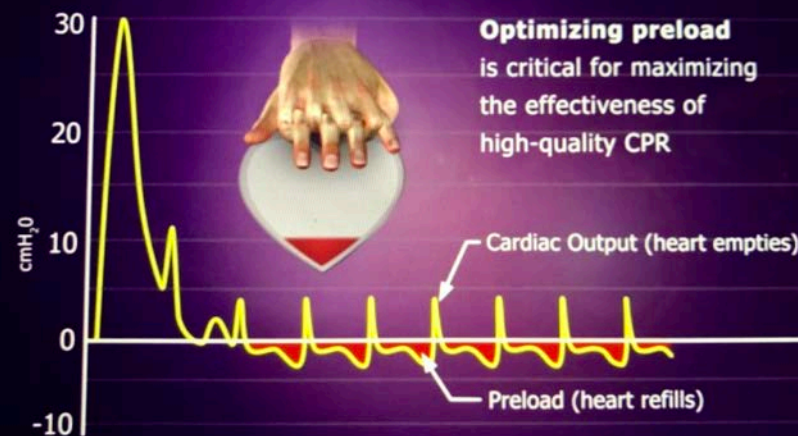
Thoracic Pump - Decompression

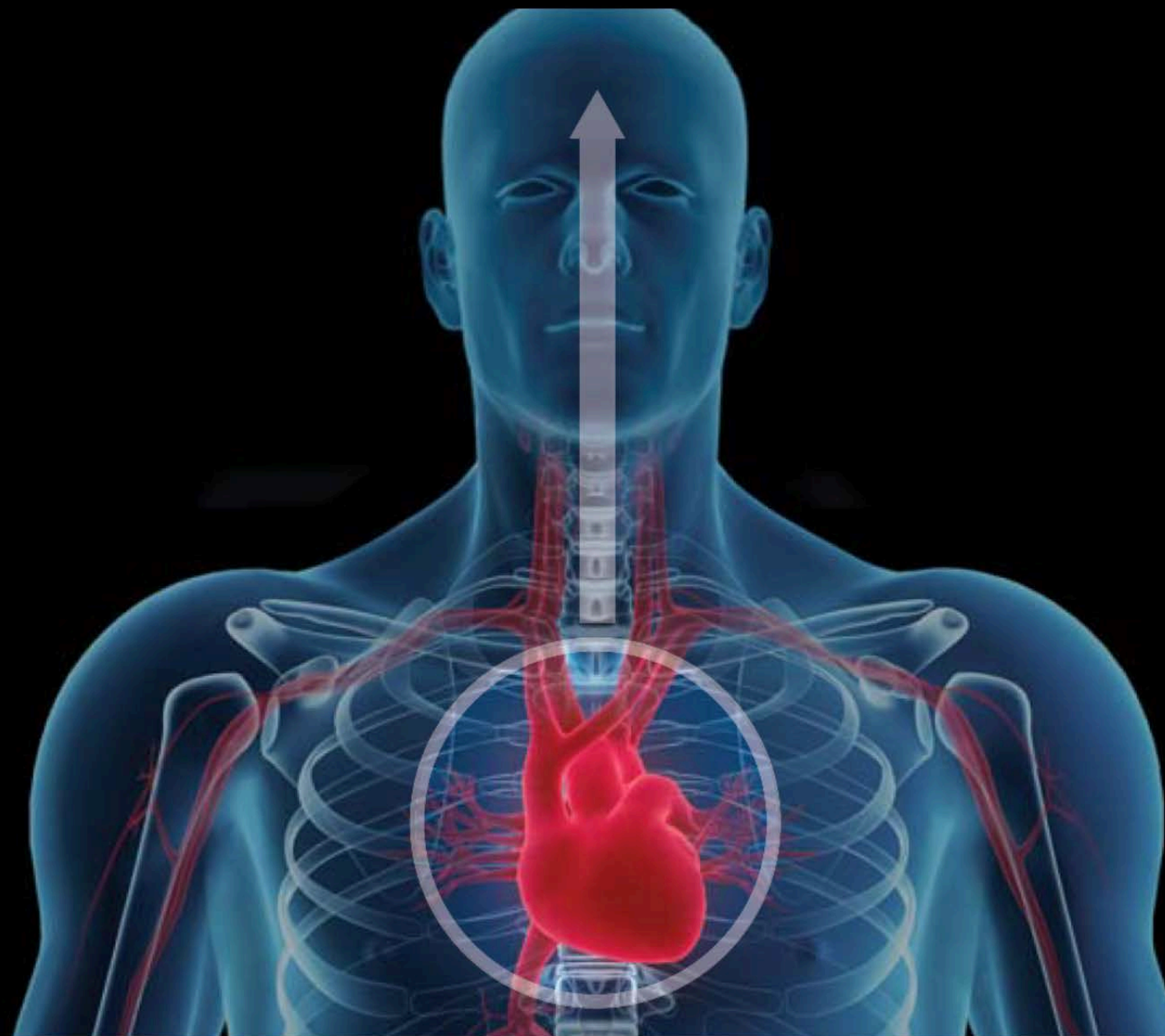
NEGATIVE pressure (vacuum):

- Fills heart (preload)
- Pulls air in
- Creates coronary artery blood flow



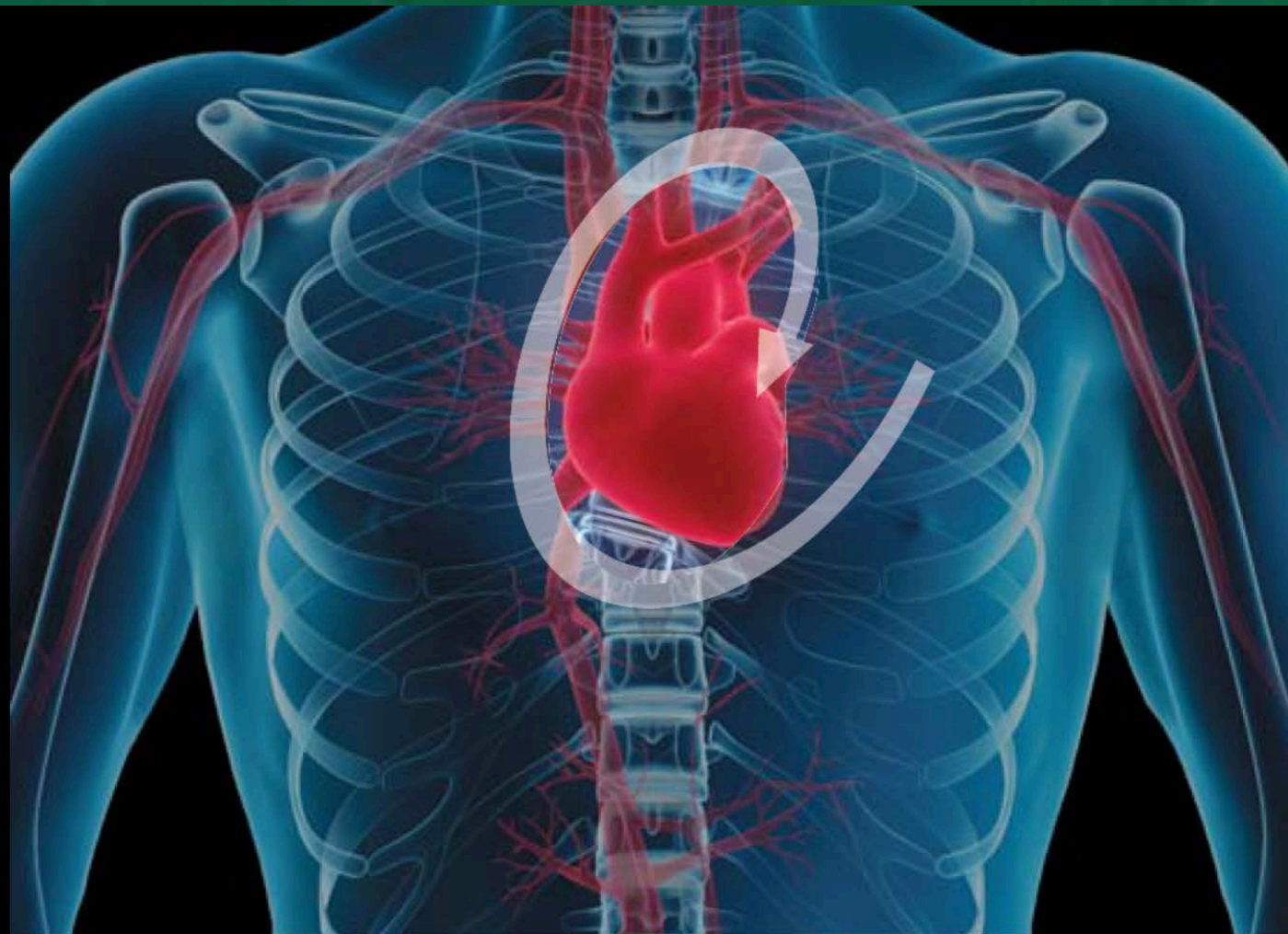
Intrathoracic Pressure





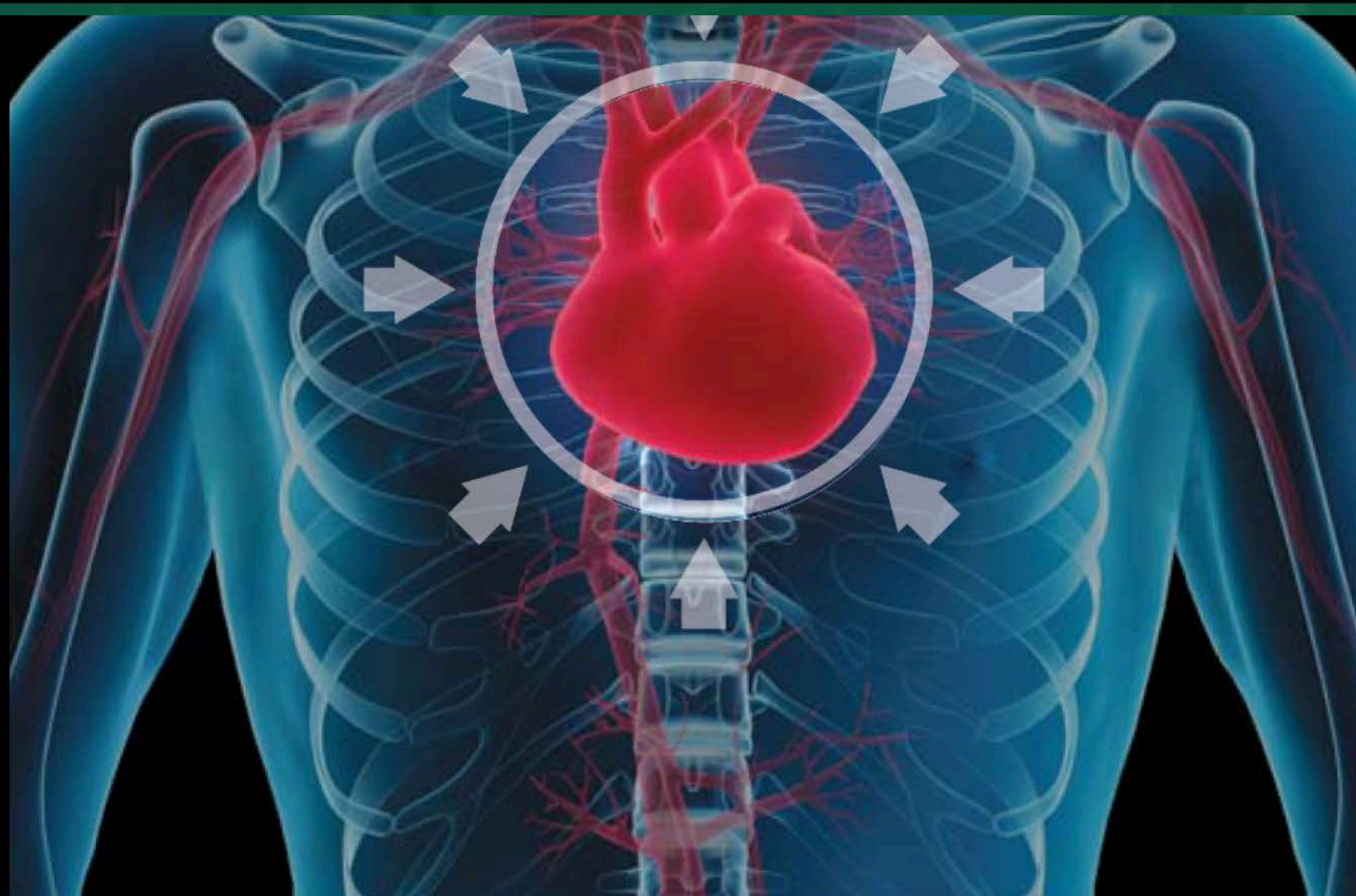
1. CPR PROVIDES BLOOD TO THE BRAIN

By moving some blood to the brain, CPR helps to preserve it. It can also play a significant role in the survivor's quality of life.



2. CPR MOVES BLOOD THROUGH THE HEART, TO THE HEART

CPR moves blood to the heart itself. This is critical right after a defibrillating shock. A shock cannot “restart” the heart. *It stuns it.* Then the heart’s own electrical pacemakers can reorganize and restore a natural heartbeat. As it reorganizes, the heart struggles for blood for several minutes. By moving blood through the heart back to the heart itself, CPR provides critical help to a struggling heart.



3. EVACUATES BLOOD FROM THE ENLARGED HEART

After four minutes without CPR, the arteries have quit, but the veins have continued delivering blood to the heart. This enlarges the heart's volume by about 50%, and prevents a shock from working (even if the heart rhythm is shockable) because it is too full of blood to resume pumping. *Chest compressions are now more critical than the shock itself* because they can restore the heart's normal volume and allow the shock to work. A victim down longer than four minutes always needs CPR prior to delivering a shock.

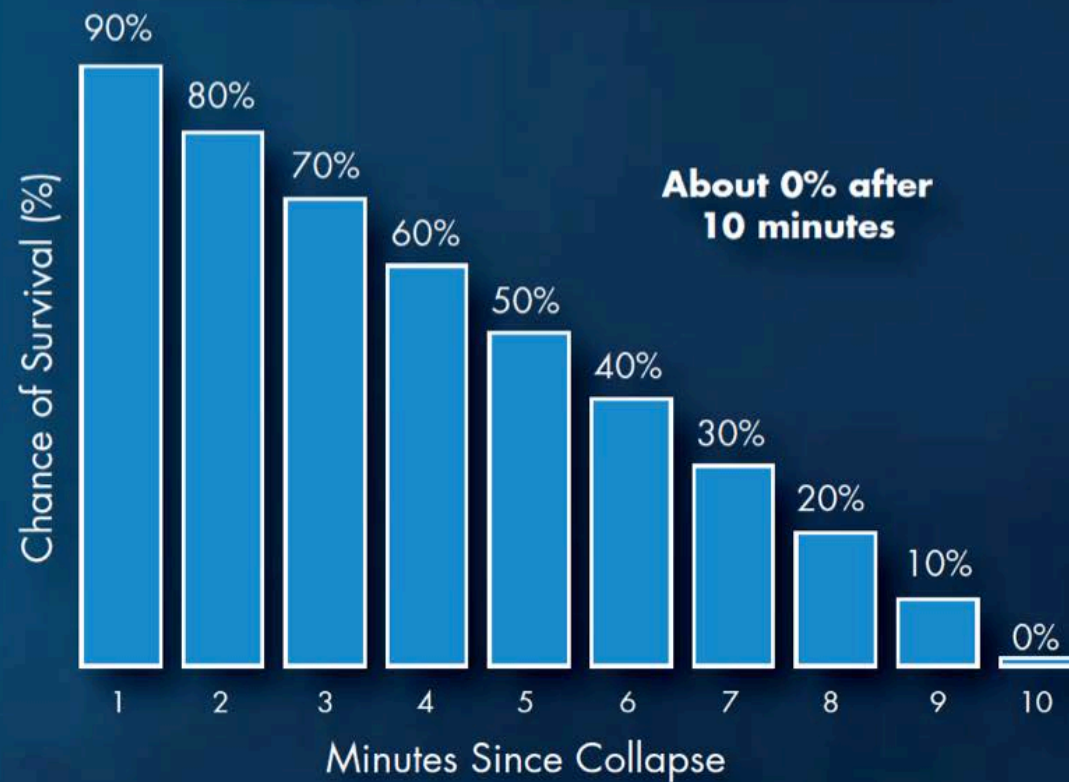
Speed is Critical...



For every minute that passes without any CPR or a defibrillating shock, the probability a shock will save the victim drops by about 10%.

So go get the AED as quickly as possible!

Probability of Survival (No CPR)



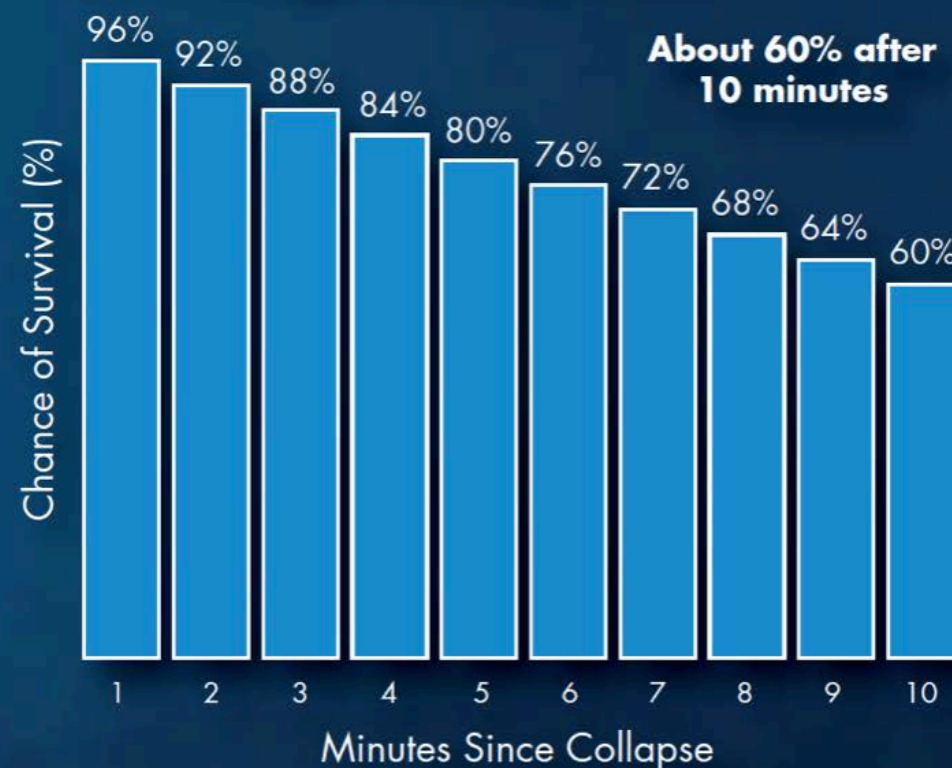
Source: 2010 AHA Guidelines for CPR and ECC. Circulation. 2010;122:S706.

Even CPR Alone can Make a Difference

Start chest compressions immediately. Even without an AED, good CPR alone radically improves the chances of survival. The probability of success only drops by 4% every minute if you continue doing good CPR.

If you do good CPR, after 10 full minutes, **the probability of success remains at 60%!** Even without an AED.

Probability of Survival (with High-Quality CPR)



Source: 2010 AHA Guidelines for CPR and ECC. *Circulation*. 2010;122:S706.

TIME

CALIFORNIA

Their 'First Kiss' Came When He Collapsed and She Performed CPR. Now They're Teaching Others to Save Lives

Gina Martinez
Aug 29, 2018



Max Montgomery, 56, survived, thanks in part to the lifesaving efforts of his date, Dr. Andi Traynor. The pair had just finished stand-up paddle board surfing in October 2017 in Capitola, California when Montgomery collapsed on the beach. Traynor immediately began performing CPR on Montgomery while passers-by called 911.

(Dr. Andrea Traynor
Clinical Associate Professor
Department of Anesthesiology
Stanford U. Medical Center
Palo Alto, CA)

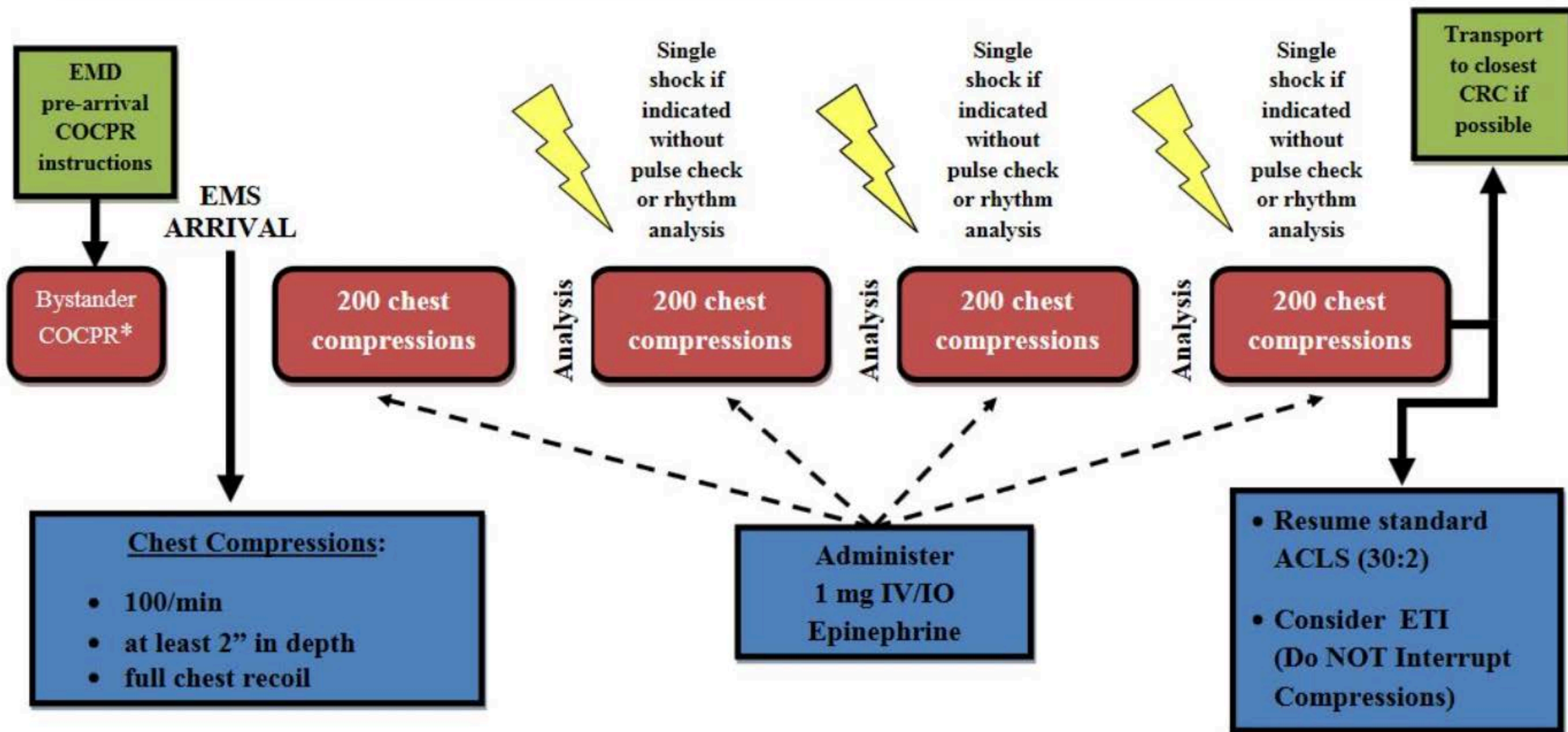


October 21, 2017 - Capitola, CA



CARDIOCEREBRAL RESUSCITATION (CCR)

(aka Minimally Interrupted Cardiac Resuscitation)



*If adequate uninterrupted bystander chest compressions are provided, EMS providers perform immediate rhythm analysis

COCPR=compression-only CPR

CRC=cardiac receiving center

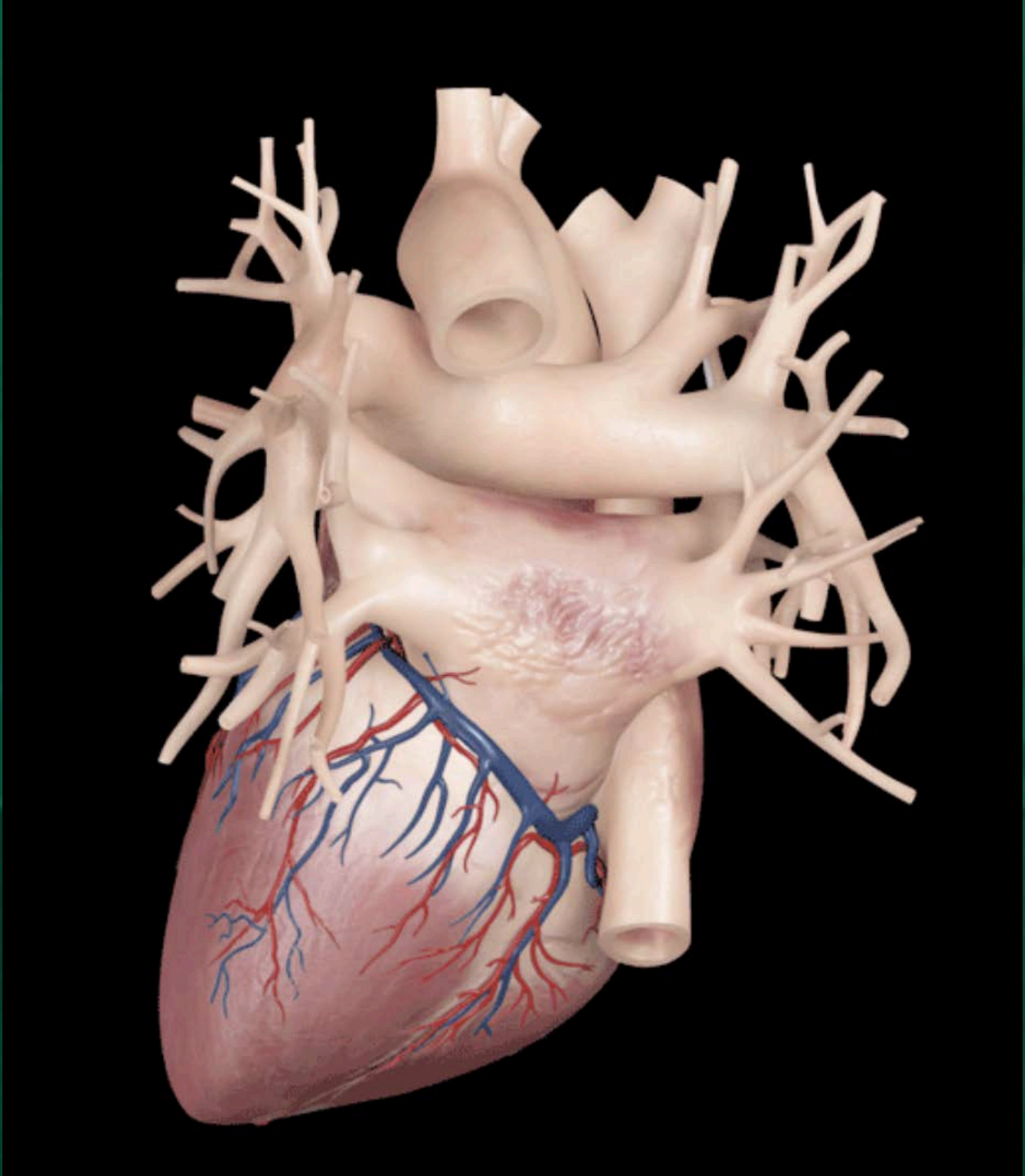
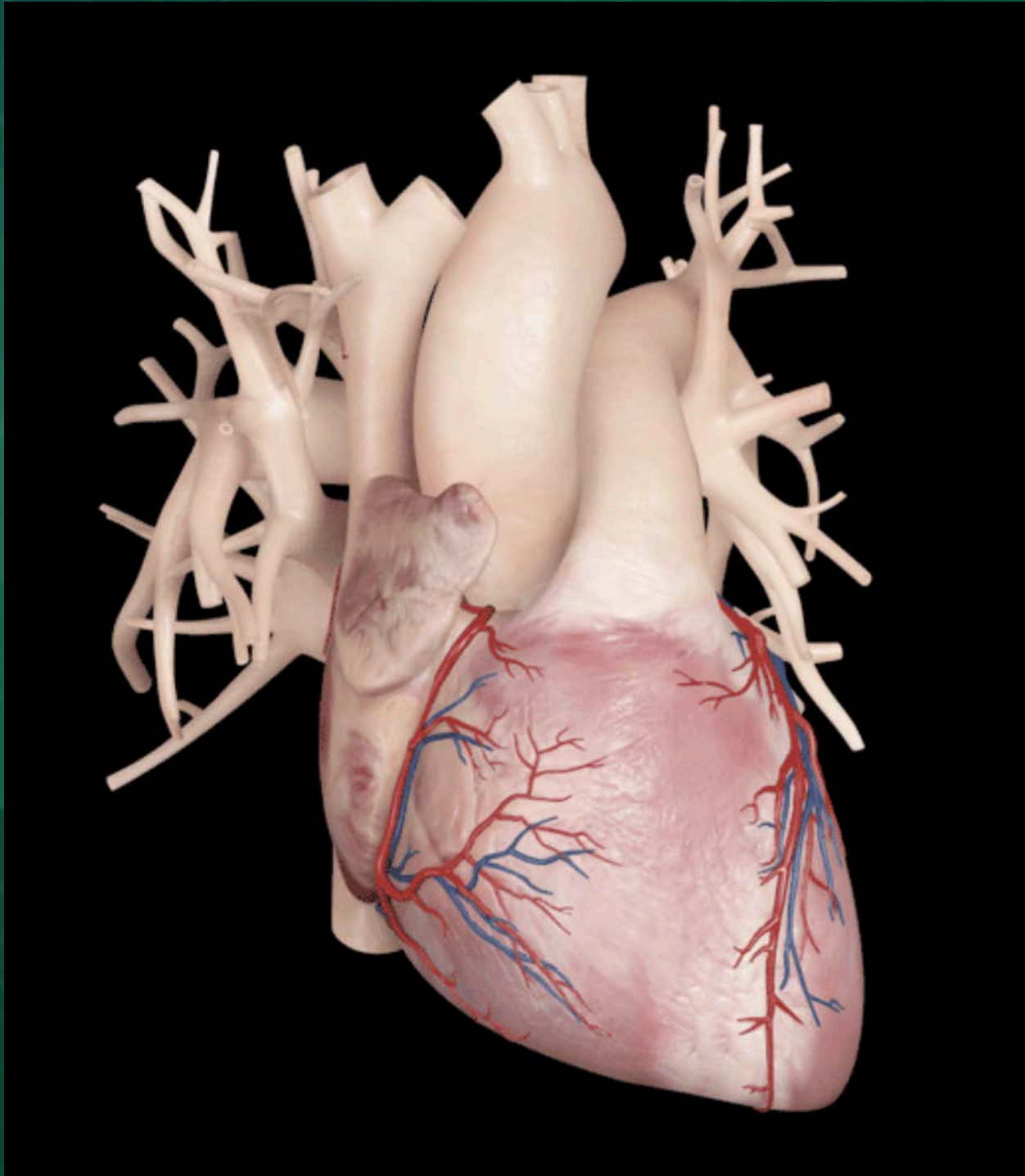
EMD=emergency medical dispatch

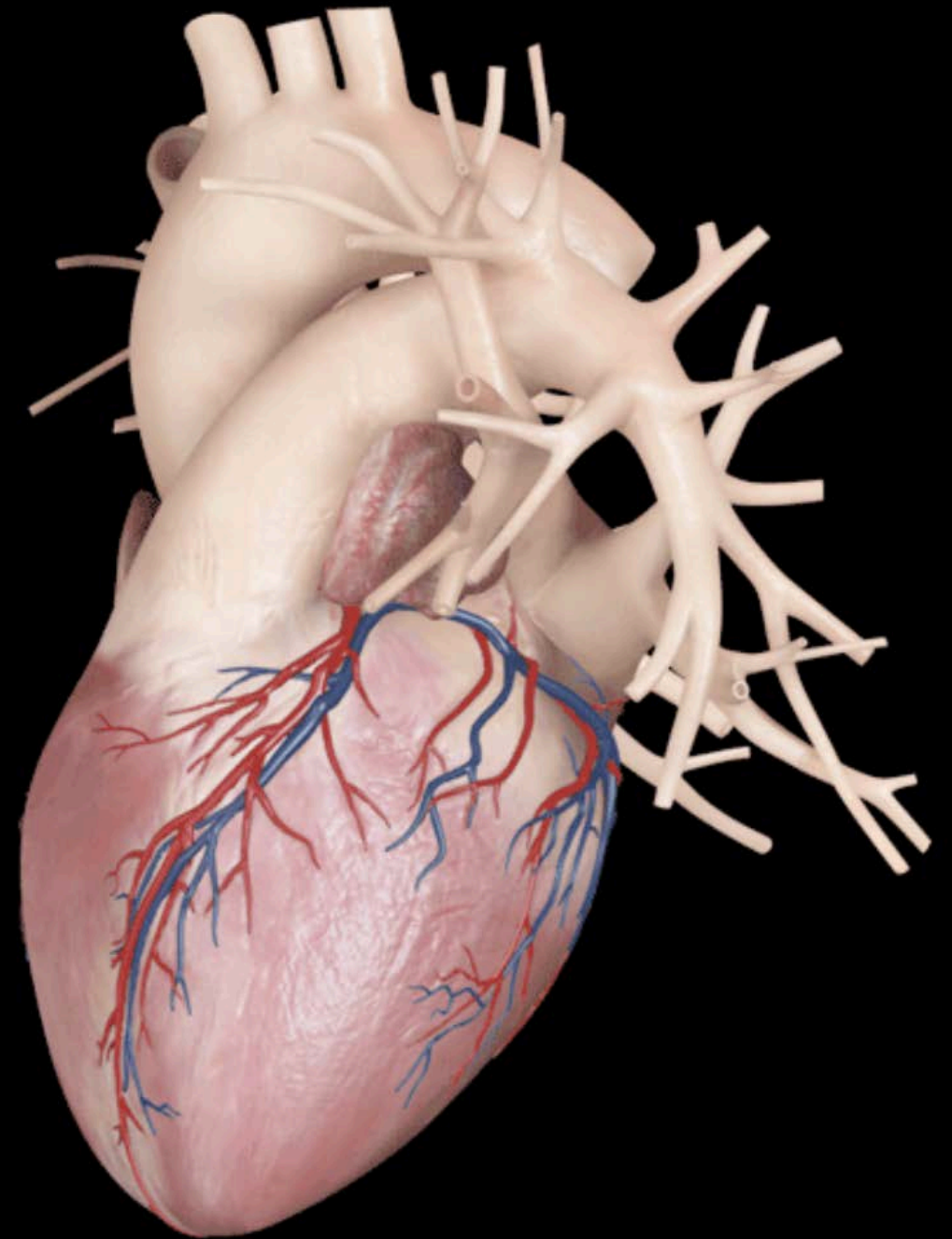
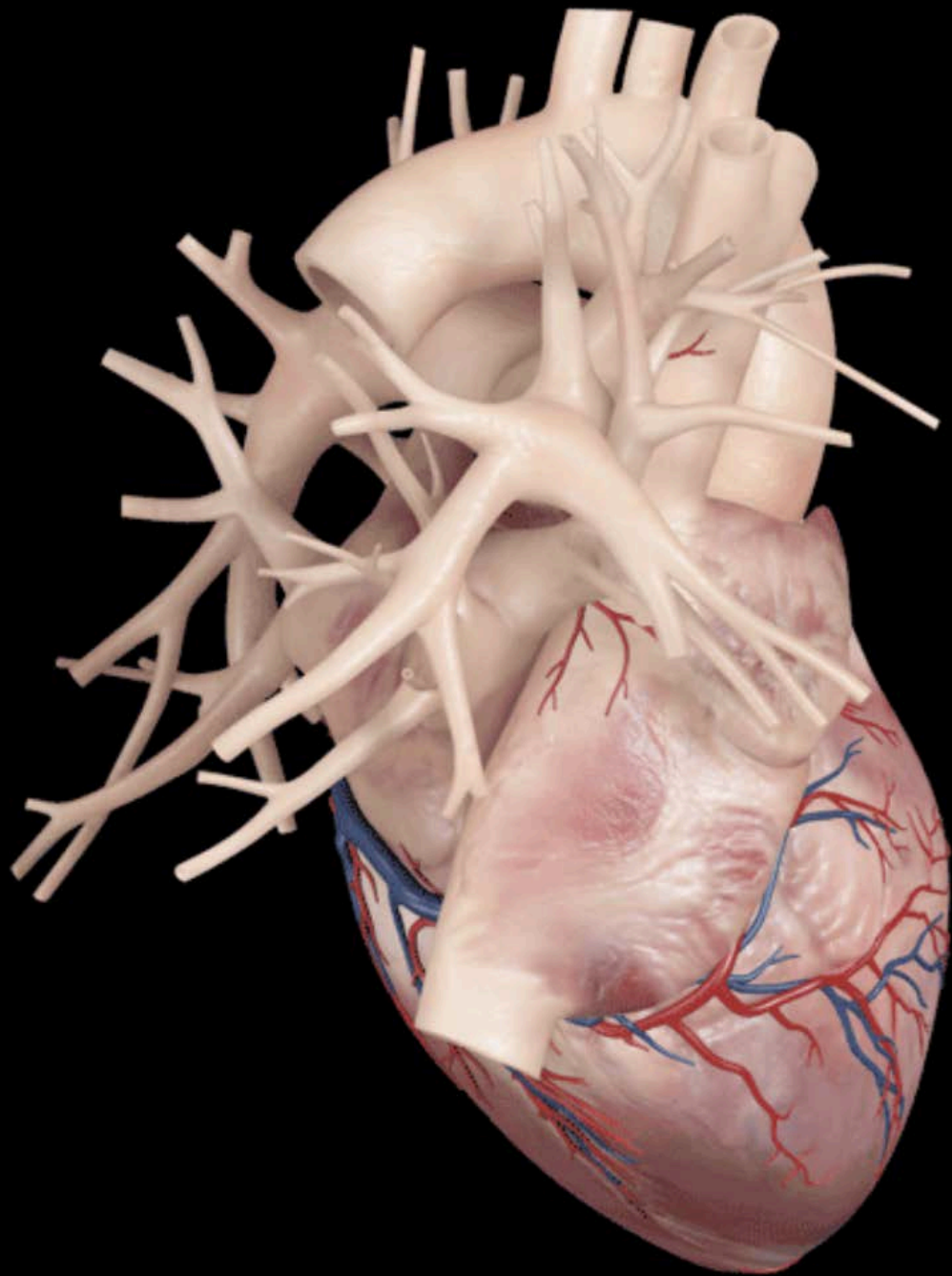
AHA: Not All CPR Is Equal

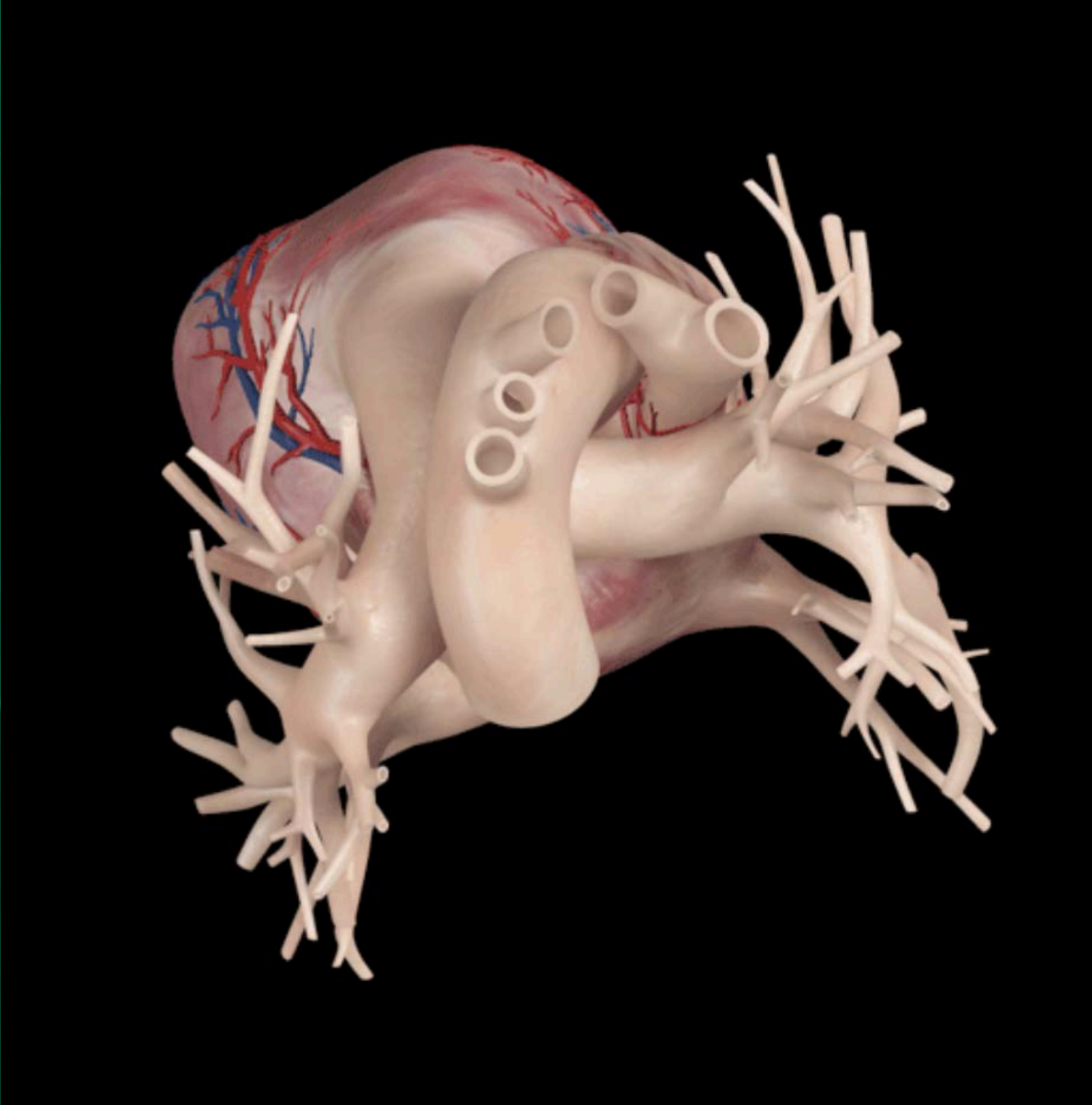
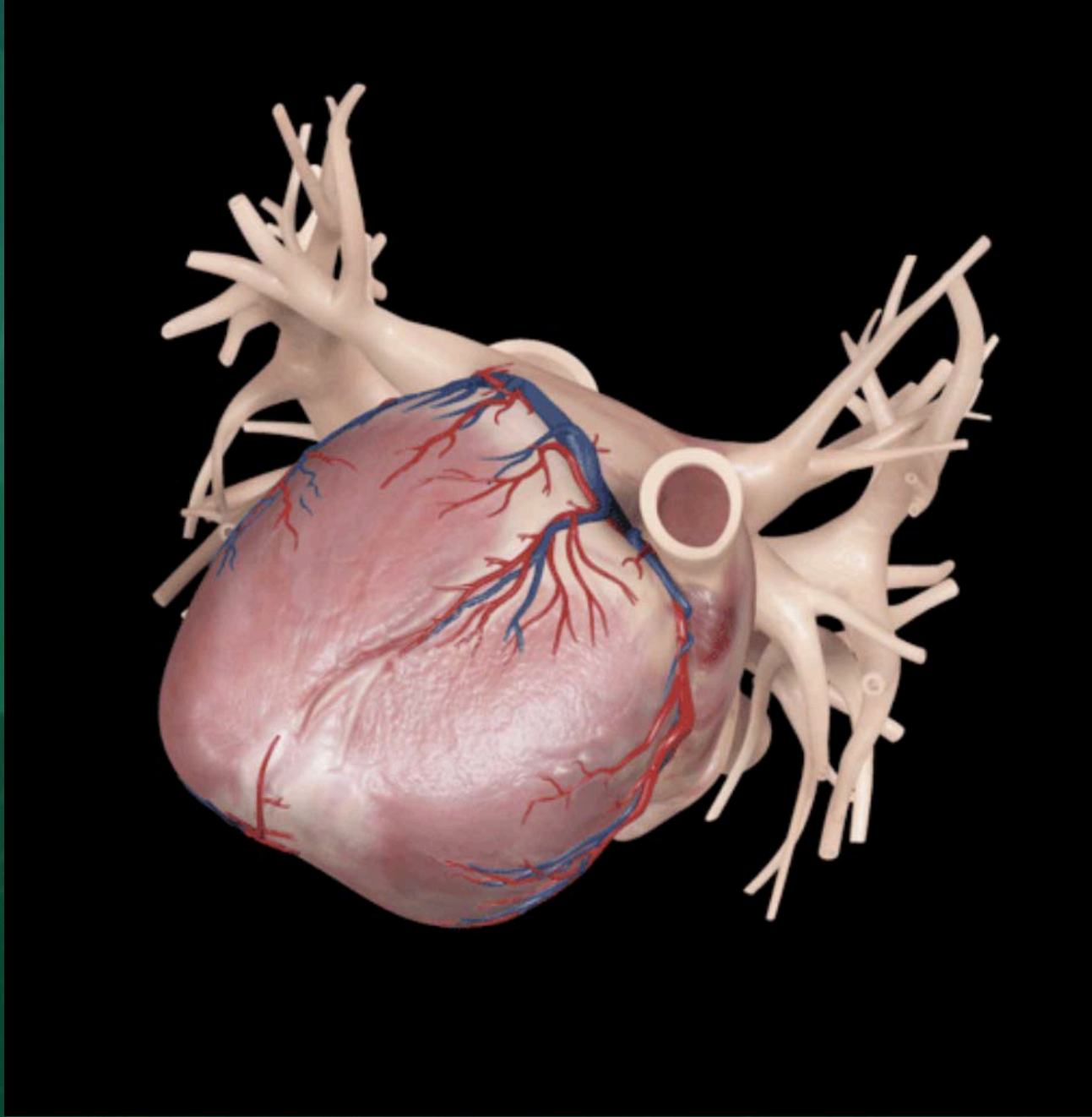
-Only 40% of patients got effective treatment.

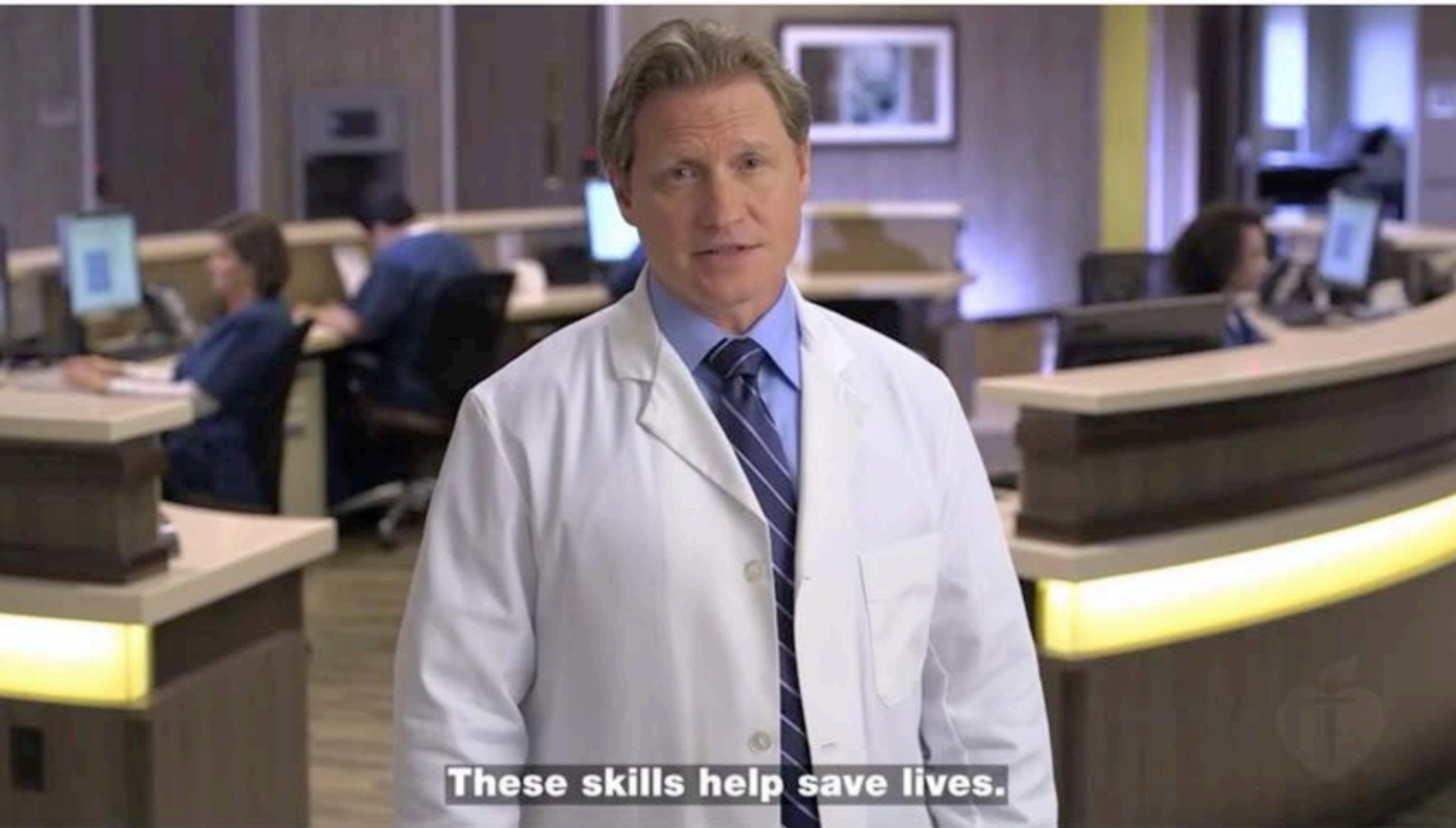


Only about 40% of patients in the trial who performed CPR were guideline compliant in delivering proper compression rate, compression depth and compression fraction, said [Demetris Yannopoulos, MD](#), associate professor of medicine at the University of Minnesota, Minneapolis.









These skills help save lives.

**** Each step in the process should be defined and must be performed repeatedly in the same manner. Any variations in the process will most likely increase cycle time and cause quality issues. ****

"All this has been said before—but since nobody listened, it must be said again."

AHA Consensus Statement

Circulation

July 23, 2013

Cardiopulmonary Resuscitation Quality: Improving Cardiac Resuscitation Outcomes Both Inside and Outside the Hospital

A Consensus Statement From the American Heart Association

Endorsed by the American College of Emergency Physicians and the Society of Critical Care Medicine

- In a hospital setting, survival is >20% if the arrest occurs between the hours of 7 AM and 11 PM but only 15% if the arrest occurs between 11 PM and 7 AM.⁹ There is significant variability with regard to location, with 9% survival at night in unmonitored settings compared with nearly 37% survival in operating room/postanesthesia care unit locations during the day.⁹

- Patient survival is linked to quality of cardiopulmonary resuscitation (CPR). When rescuers compress at a depth of <38 mm, survival-to-discharge rates after out-of-hospital arrest are reduced by 30%.¹⁰ Similarly, when rescuers compress too slowly, return of spontaneous circulation (ROSC) after in-hospital cardiac arrest falls from 72% to 42%.¹¹

The variations in performance and survival described in these studies provide the resuscitation community with an incentive to improve outcomes. To maximize survival from cardiac arrest, the time has come to focus efforts on optimizing the quality of CPR specifically, as well as the performance of resuscitation processes in general.

Chest Compression Rate of 100 to 120/min

The 2010 AHA Guidelines for CPR and ECC recommend a chest compression rate of $\geq 100/\text{min}$.²⁸ As chest compression rates fall, a significant drop-off in ROSC occurs, and higher rates may reduce coronary blood flow^{11,32} and decrease the percentage of compressions that achieve target depth.^{10,33} Data

Chest Compression Depth of ≥ 50 mm in Adults and at Least One Third the Anterior-Posterior Dimension of the Chest in Infants and Children

Compressions generate critical blood flow and oxygen and energy delivery to the heart and brain. The 2010 AHA Guidelines for CPR and ECC recommend a single minimum depth for compressions of ≥ 2 inches (50 mm) in adults. Less

Full Chest Recoil: No Residual Leaning

Incomplete chest wall release occurs when the chest compressor does not allow the chest to fully recoil on completion of the compression.^{44,45} This can occur when a rescuer leans over the patient's chest, impeding full chest expansion. Leaning is known to decrease the blood flow throughout the heart and can decrease venous return and cardiac output.⁴⁶

Four areas related to CPR quality will be addressed:

- Metrics of CPR performance by the provider team
- Monitoring and feedback: options and techniques for monitoring patient response to resuscitation, as well as team performance
- Team-level logistics: how to ensure high-quality CPR in complex settings
- CQI for CPR

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The AHA recommends the use of audio and visual feedback devices to ensure that CPR quality is maintained and optimized during a resuscitation attempt.

It has been noted that although " ... staff at hospitals across the country are required to undergo CPR recertification every two years, studies have shown that *their skills begin to degrade the moment the training session ends, and most people have lost effective skills within 90 days.*"

Where there is no vision, the people perish.

But happy is the one who heeds the instructions of wisdom.



LEAD

SIZE

ALARM
SUSPEND

RECORDER

ON
OFF

PACER

Please note the R Series Plus is in Manual Mode



X Series CPR Feedback



Watch later

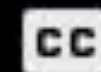


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More videos



0:00 / 2:53



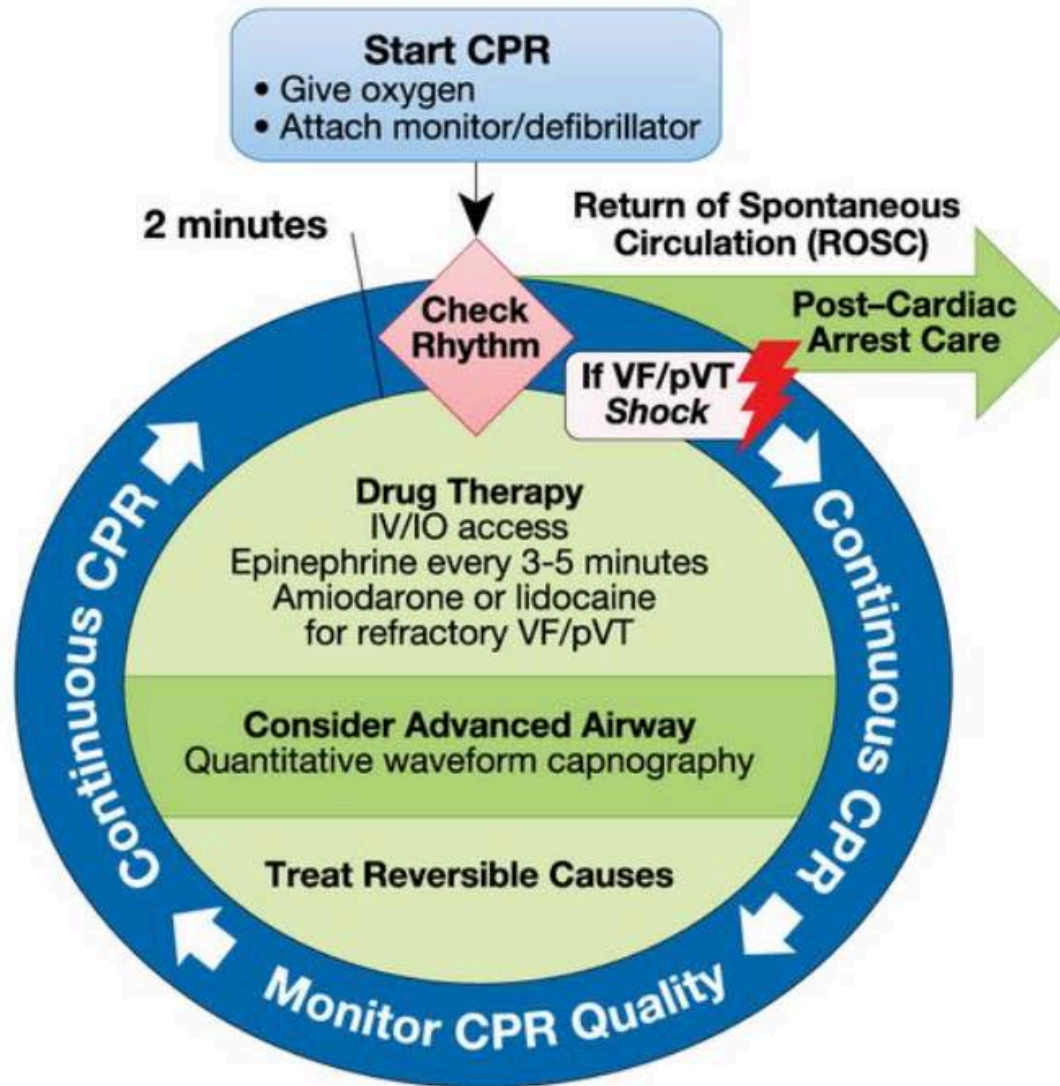
YouTube



In the land of the blind, the one-eyed man is king.



Adult Cardiac Arrest Circular Algorithm – 2018 Update



CPR Quality

- Push hard (at least 2 inches [5 cm]) and fast (100-120/min) and allow complete chest recoil.
- Minimize interruptions in compressions.
- Avoid excessive ventilation.
- Change compressor every 2 minutes, or sooner if fatigued.
- If no advanced airway, 30:2 compression-ventilation ratio.
- Quantitative waveform capnography
 - If PETCO₂ <10 mm Hg, attempt to improve CPR quality.
- Intra-arterial pressure
 - If relaxation phase (diastolic) pressure <20 mm Hg, attempt to improve CPR quality.

Shock Energy for Defibrillation

- **Biphasic:** Manufacturer recommendation (eg, initial dose of 120-200 J); if unknown, use maximum available. Second and subsequent doses should be equivalent, and higher doses may be considered.
- **Monophasic:** 360 J

Drug Therapy

- **Epinephrine IV/IO dose:** 1 mg every 3-5 minutes
 - **Amiodarone IV/IO dose:** First dose: 300 mg bolus. Second dose: 150 mg.
- OR-**
- **Lidocaine IV/IO dose:** First dose: 1-1.5 mg/kg. Second dose: 0.5-0.75 mg/kg.

Advanced Airway

- Endotracheal intubation or supraglottic advanced airway
- Waveform capnography or capnometry to confirm and monitor ET tube placement
- Once advanced airway in place, give 1 breath every 6 seconds (10 breaths/min) with continuous chest compressions

Return of Spontaneous Circulation (ROSC)

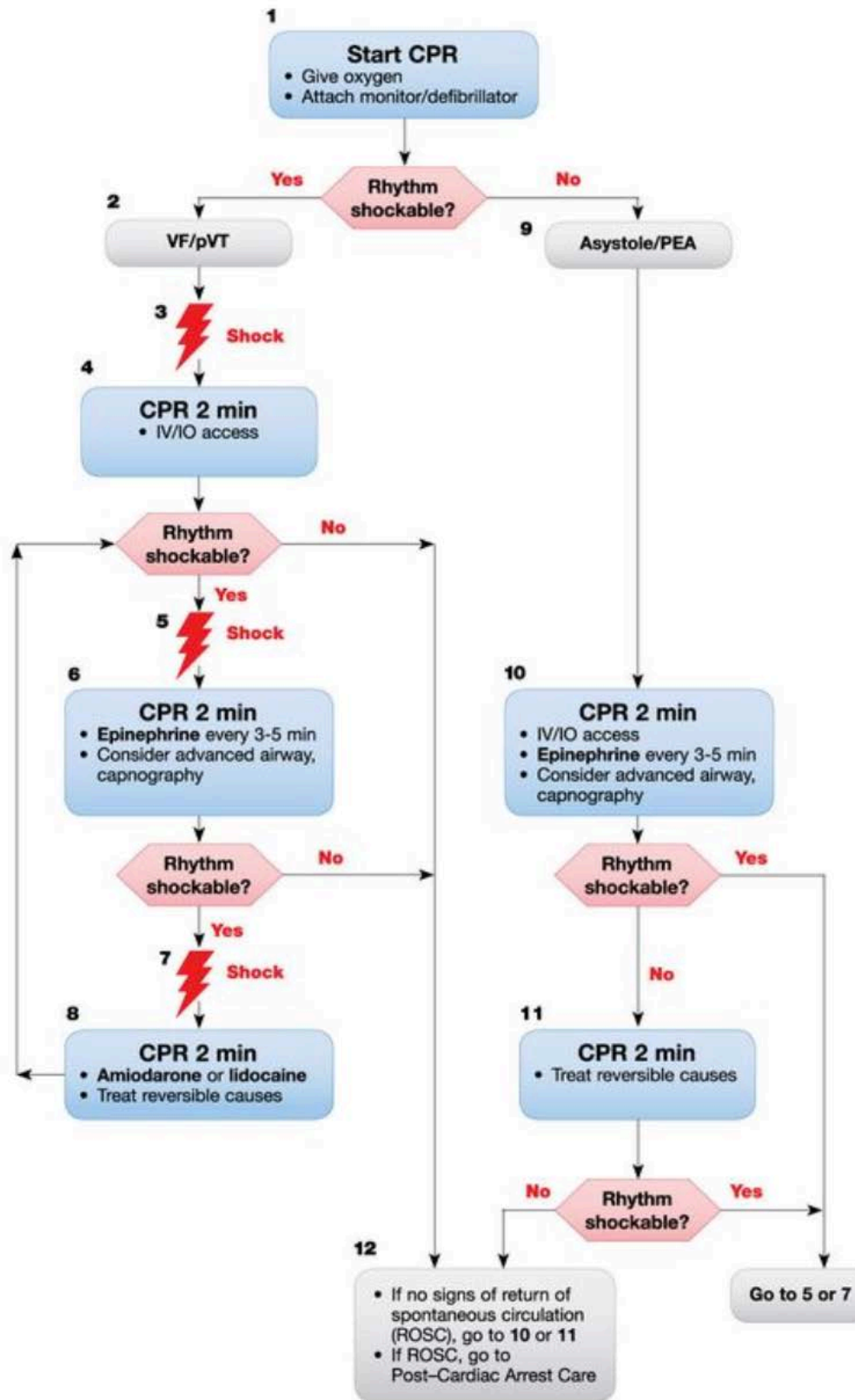
- Pulse and blood pressure
- Abrupt sustained increase in PETCO₂ (typically ≥40 mm Hg)
- Spontaneous arterial pressure waves with intra-arterial monitoring

Reversible Causes

- | | |
|---------------------------|-------------------------|
| • Hypovolemia | • Tension pneumothorax |
| • Hypoxia | • Tamponade, cardiac |
| • Hydrogen ion (acidosis) | • Toxins |
| • Hypo-/hyperkalemia | • Thrombosis, pulmonary |
| • Hypothermia | • Thrombosis, coronary |



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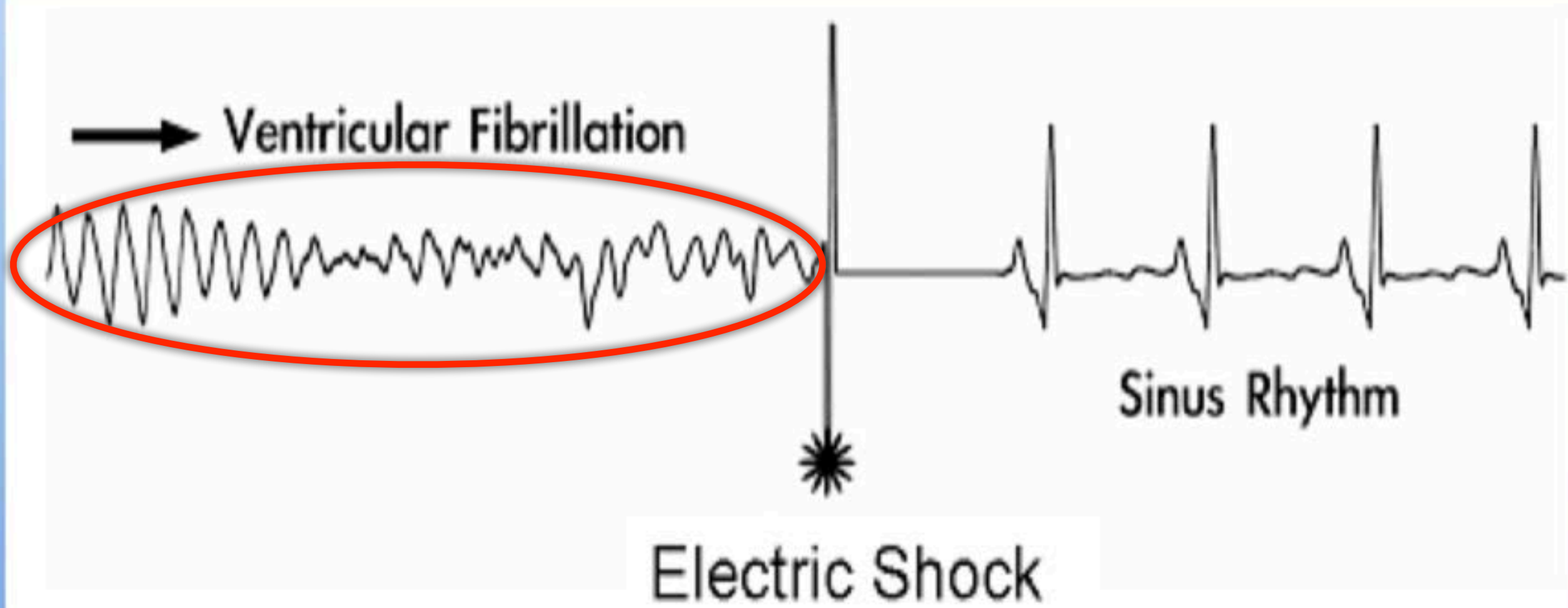


8th Annual Dr. Ed Waits Respiratory Care Conference
June 19, 2019

CQI for CPR

Providing
Real-Time Performance Feedback
During CPR

James R. Boogaerts, MD, PhD, FACC
UAB Division of Cardiovascular Disease



***Only* High Quality CPR
Can Provide**

**Adequate
Coronary and Cerebral Perfusion**

***After* Sudden Cardiac Arrest**

***Before* ROSC**



