Engineering Better CPR	Slide	Talk
• TItle	Slide 1 Title	
▼ Story of Cardiac Arrest in 1959	Slide 2 Blank	I would like to tell you a story of a 23-year-old woman.
• Arrest		It had been a bad summer for her asthma. 8 times already she had suffered attacks. But on that August morning, it was worse, much worse. Sitting upright, eyes closed, she tried biting the good air and swallow it into her lungs. But even the sounds of her wheezes were failing. Within 30 minutes, she shuffled through the door of the emergency room held up by the taxi driver, and then collapsed. Pulseless.
• CPR		She was immediately attended to. In the next several instants she was intubated and chest compressions were started. It took five minutes before her heart began beating on its own. But, she was left comatose.
• Hypothermia		Bags of ice were packed around her to cool her. During the first several hours, there were episodes of posturing. By 18 hours after her arrest though, she was responding to touches. Pupillary reflexes returned shortly after. The hypothermia was stopped at 24 hours after arrest and she allowed to warm slowly. 48 hours after the arrest she was awake and responsive. She was discharged neurologically intact, 11 days later.
• People	Slide 3 People	Similar stories happen in every emergency department every day. Whats amazing about this one, is that it was published as a case report by these two people, G Rainey Williams and Frank Spencer, in the Annals of Surgery…
• 1959	Slide 4 1959	…in 1959. Despite 50 years between us and this case report, every element of CPR is recognizable and essentially unchanged.
▼ Dismal Outcomes	Slide 5: Build 0 Graph	Including our dismal ability to..
• ROSC Graphg	Slide 5: Build 1 ROSC	…get the heart pumping again, …
• Survival Graph	Slide 5: Build 2 Survival	and even worse have the patient survive.
▼ Improvements		
• By stander CPR	Slide 6: Build 1 By stander	Countless effort has been expended to improve survival, including encouraging bystander CPR, …
• AED	Slide 6: Build 2 AED	widespread deployment of AEDs, …
• Depth/Rate of chest compressions	Slide 6: Build 3 Quality	emphasizing quality chest compressions, …
• Ventilation rate	Slide 6: Build 4 Ventilation	…controlling the ventilation rate, …

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• Hypothermia	Slide 6: Build 5 Hypothermia	and of therapeutic hypothermia. Despite all of that survival rate is unacceptable.
• We need to improve	Slide 6: Build 6 No text	What it would it take to double the survival rate? Is it possible to triple it? Can we apply our better understanding of arrest to engineer better devices, systems, and medicines to create a better CPR. A more successful CPR.
▼ Hemodynamics of Arrest	Slide 7: Build 0 Blank	When the heart arrests…
• Forward flow continues for several minutes after arrest until Ao pressure falls to Ra pressure.	Slide 7: Build 1 Graph/Ao Press	…forward flow in the aorta falls rapidly, but takes about 4 minutes before the pressure of the arterial and venous system equalizes and flow stops completely.
• Right ventricular swells	Slide 8: 0-4 Swollen Vents	The blood emptying from the arteries pools in the venous system markedly distending the right ventricle.
•	Slide 9: Build 0 Graph/Ao Press	
• Carotid Blood Flow	Slide 9: Build 1 Carotid Flow	Carotid flow decreases similarly but more rapidly.
• Coronary Blood Flow	Slide 9: Build 2 Coronary Flow	Coronary blood flow falls even more precipitously, and then actually flows backwards for several minutes before stopping all together.
▼ Hemodynamics of CPR	Slide 10: Build 0 Chest compresion	During chest compressions, the heart is compressed between the sternum and vertebra,
•	Slide 11: Build 0 Zoom in on heart	
• Cardiac Pump	Slide 11: Build 1 Cardiac Pump with Arrows	and blood is squeezed out of the ventricles and forward into the aorta against closed AV valves.
• Thoracic Pump	Slide 11: Build 2 Thoracic Pump with Arrows	However, if the chest compressions are not effective in squeezing the heart (poor quality CPR or difficult anatomy), blood is pushed forward only by the increased thoracic pressure which causes it to flow towards lower pressures areas outside of the chest. In this case, the heart is passive conduit and the mitral valve remain open.
• Coronary blood flow is critical for ROSC	Slide 12	There are only two goals for chest compressions during cardiac arrest. First, you must generate enough coronary blood flow to deliver oxygen to the profoundly ischemic heart. The higher the coronary perfusion pressure, the better chance we have of achieving ROSC. This is made tougher, because forward blood flow in the coronary arteries only occurs during the decompression phase of chest compressions (equivalent to diastole). It also takes several seconds to restore coronary blood flow after any interruptions, because the pressure gradient needs to be reestablished.

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• Carotid Flow		Brain cells become ischemic when tissue oxygen content is less than <8mmHg. The second goal of chest compressions is to deliver oxygen to the brain to prevent neurologic injury. Fortunately, cerebral perfusion occurs during systole and diastole.
• Cardiac Output		Standard chest compressions typically deliver about 25% of normal cardiac output. To improve our ROSC and survival rates we need to generate more cardiac output, and direct it as much as we can towards the heart and brain.
• Mechanical CPR	Slide 13 LUCAS	Mechanical chest compression devices are able to provide perfect tireless quality chest compressions. They compress exactly to 50mm, at a perfect rate of 100. They don't lean against the patient, and don't ever stop until we tell them to. The LUCAS device shown here uses an electrically driven piston. There are other devices that use a band or vest. They are not the complete answer. Despite the high quality guideline-driven chest compressions, the LINC trial among others have not shown to improved ROSC or survival rates.
▼ Active Compression-Decompression	Slide 14: Build 0 Compression	During chest compressions, blood flow is dependent on refilling the heart during the decompression phase. Increasing venous return will deliver more blood to the heart, which can be driven forward with each compression. Passive recoil of the chest, draws air into the lung and blood into the chest, but is not very effective and gets worse as rib fractures occur.
• Active Decompression	Slide 14: Build 1 Decompression	But we can actively expand the chest during decompression we can generate larger negative intrathoracic pressure. This negative pressure will reduce the right atrial pressure and draw more blood into the thoracic cavity and fill the heart. On each subsequent compression cycle, there will be more blood available for cardiac output.
• Reduces ICP		The larger negative intrathoracic pressure has the additional benefit of lower intracranial pressure, which enhances cerebral blood flow by lowering venous back flow.
▼ Intrathoracic Impedance Device/Intrathoracic Pressure Regulator	Slide 15: Build 0	It is difficult to keep maintain a large negative intrathoracic pressures with a typical airway circuit.
• ITD Schematic	Slide 15: Build 1 ITD Neutral	A check-valve type device known as an ITD (impedance threshold device), can help maintain the negative pressure though.
• Compression	Slide 15: Build 1 ITD Compression	During chest compression, air is forced out of the thoracic cavity through the device.
• Decompression	Slide 15: Build 2 ITD Decompression	During decompression, the check valve closes and air is prevented from entering the thoracic cavity, so that the negative pressure can be maintain and drive venous return.

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• ITD relief valve	Slide 15: Build 3 ITD Relief Valve	Excessive negative pressures can injure the lung so an escape valve will relive the negative pressure if it exceeds -16 cmH2O.
• ITD BVM Ventilations	Slide 15: Build 4 ITD BVM Breath	There is no impedance to providing BVM as required.
• ResQ Trial	Slide 16 ResQPod/ ResQPump	Trials testing the ITD and active compression-decompression independently did not show benefit, but when used together in the ResQ Trial showed a 50% improvement in neurologically intact survival at 1 year for cardiac arrest of cardiac origin. A confirmatory study showed a 34% improvement for all cardiac arrests regardless of etiology.
• LUCAS not ACD		The LUCAS device should not be considered a ACD device. It only applies 3 lb force, as compared to 20-25 lbs tested in the trials.
• Circulatory Volme	Slide 17	If we reduce the circulatory volume, we can redirect more of our cardiac output to the heart and brain. Various ways to do this. Limb tourniquets, lower abdominal binding, or even endovascular occlusion of the aorta. By themselves, these techniques raise right atrial pressure and create higher ICPs, but these effects are eliminated and when using ACD-ITD.
• Heads-up	Slide 18 Heads Up	A mechanical device delivering chest compressions allows for possibilities that couldn't be considered before. Small elevators in Korea have been a problem for EMS personnel transporting patients. They had been transporting patients with legs up attempting to increase venous return, but likely inadvertently raising ICP and leading to worse neurologic outcomes. However, using a LUCAS device and ITD, they were able to place the patient in a heads up position. Something difficult to do with manual chest compressions. In this position, the right atrial pressure is lower, which increases both coronary, lowers ICP which leads to more cerebral blood flow. Without the active compression-decompression and ITD, to ensure adequate venous return, the cardiac output would be less.
▶ SNPeCPR	Slide 18	To optimize our blood flow we want high arterial resistance everywhere except the heart and brain, where you want low resistance. An approach is to use a potent vasodilator such as sodium nitroprusside. However, to maintain adequate coronary and cerebral perfusion pressures ACD-ITD and LB is used. It has shown, Improved 24-hours survival, neurologic function, LV function, and vital organ blood flow.
▼ Feedback System	Slide 20: Build 0 Open Loop	To achieve breakthrough improvements, we should not be by trying to make a device that delivers perfect unfailing one-size fits all guideline chest compressions.

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•	Slide 20: Build 1 Closed Loop	We can realize its full potential when it is connected to a controller and incorporated into a feedback system. What becomes possible when a controller and feedback loop are incorporated. The respond to the patient during CPR.
▼ EtCO2	Slide 21	End-tidal CO2 is an excellent measure of cardiac output in low-flow states, such as when a person is in arrest.
• EtCO2 physiologic measure of cardiac output in low-flow states		It measures the alveolar carbon dioxide content at the end of expiration. As long as ventilations are provided, then the EtCO2 is reflection blood flow through the pulmonary circuit which is the same as cardiac output.
• Effective chest compressions		It gives a visual and effective representation of efficient chest compressions. Higher ventilation rates lower EtCO2. High compression depth increases EtCO2. You want to target >20.
• Visual indication of ROSC		You can see a dramatic rise in EtCO2 when the patient has ROSC.
▼ Reduce hands off time		Checking pulses to differentiate PEA from ROSC is notoriously inaccurate and time consuming
• Checking pulse to differentiate PEA and ROSC is difficult and time consuming		
• Pulse checks notoriously inaccurate		
• Connected to a LUCAS		If connected, can the LUCAS start and stop based on the ROSC.
• Automated Rhythm Analysis	Slide 22	The controller with some advanced mathematics could analyze the rhythm during ongoing chest compressions and detect shockable rhythm, without ROSC based on the EtCO2. Signaling the defibrillator, a shock can be provided with ongoing chest compressions. The practice of CPR cycle loops become obsolete.
▶ NIRS	Slide 23	Cerebral oximetry measures the tissue oxygenation in the frontal lobe. Helpful it indicating a non perfusing rhythm (PEA), and studies show ROSC is impossible with rSO2 <30%
• Stutter CPR	Slide 19 Graph	Stutter CPR, a method of post ischemic conditioning requires a complicated series of pauses and starts to chest compressions. Something humans would find unworkable. A smart device could handle we ease.
▶ Hemodynamic Directed CPR	Slide: 24	Can we move beyond the guidelines towards customized CPR. Continuous variation of the compression depth, duty cycle, rate, based on arterial pressures, EtCO2, NIRS. Move away from the same dose for every patient. Individualize the CPR based on the patient’s body type, underlying condition, cause of arrest, and treatments.
▼ On the horizon		
• Post-conditioning		

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▼ Severofluourane for post-conditioning		
• May affect mitochondrial apoptosis. Trying to prevent ischemic reperfursion injury of mitochondria.		
• One cardiac arrest researcher calls emergentologitis "mitochondriologists" because that is the organ we are most concerned with.		
▶ Conclusion	Slide 25: Build 0 Blank	
• End	Slide 26: Build 0 Blank	Thank you very much.