Resuscitation in the past, the present and the future

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“O, that I could but call these dead to life!” King Henry VI, William Shakespeare.

“There is no malice in this burning coal; The breath of heaven has blown his spirit out . . . But with my breath I can revive it, . . .” William Shakespeare.

BACKGROUND

Respiratory System

One of the earliest recordings in Egyptian mythology of resuscitation was when Isis restored her husband Osiris by breathing into his mouth. The Bible has several accounts of resuscitation using the mouth or nostrils. In Genesis it was recorded that when forming Adam, God is said to have “… breathed into his nostrils the breath of life”. In the first book of Kings, Elijah resuscitated the dying son of his landlady and we are told how he first prayed and then stretched himself along the sick child “… and the soul of the child came into him again, and he revived”. In the second book of Kings it is recorded how Elisha, when summoned to a house where a child had died “up and lay upon the child and put his mouth upon his mouth, and his eyes upon his eyes, and his hands upon his hands; he stretched himself upon him; the flesh of the child waxed warm and the child sneezed seven times, and the child opened his eyes”.

When obstructed breathing is the cause of the collapse of the patient, varying attempts throughout the ages have been made to obtain direct access through the trachea. Dating back to 2000 BC, Egyptians are recorded as using reeds through a hole in the skin entering the windpipe and Homer in 356 BC described opening the trachea to relieve those choking. Even Alexander the Great when seeing a man choking from a bone lodged in his throat is said to have punctured the trachea with the tip of his sword.

Sudden Death

For many centuries, sudden death unrelated to trauma has been recognised as a clinical entity. But sudden death due to cardiac causes was first described about the first century AD. Pliny the Elder at this time in his work “Natural History” . . . studied many citizens of Rome – physicians, senators and businessmen – who had dropped dead. Since there were no post mortem examinations, these deaths were usually attributed to “an act of the Gods”. Frequent records of sudden death were made throughout the middle ages and in the 17th and 18th centuries. Lusitanus in 1560 described “A reverend abbot from the Isle of Croma, one or two miles distant from Ragusa, when he was in good health and talking to several persons, said that he suddenly felt pain in his heart and with his hand moved rapidly toward the region of the heart, he fell, though slowly, to the earth and rapidly lost all his animal faculties. When called in I said he was dead. Not only was the pulse at the metacarpium, and the temples missing, but even no motion upon the heart could be perceived. In order to satisfy the assistants I brought to the nostrils a burning candle whose flame did not move at all. Also a bright mirror was advanced near the mouth and nothing of respiratory contraction was seen on it. We then applied a glass vessel filled with water upon the thorax but the water was unmoved”.

Lancisi (1707) performed post mortems on citizens of Rome who died suddenly during 1705-1706. He found a natural cause for death in every case and he referred particularly to diseases of the blood vessels with “obstruction therefrom of the free flow of blood”.

Sudden deaths due to coronary artery disease still remain one of the greatest challenges in contemporary society. In 1971 Gordon and Kannel stated that “we are faced with a disease which is
extremely common and highly lethal, which frequently attacks without warning and in which the first symptoms are all too often the very last. Also, it is a disease which can be silent even in its most dangerous form.  

At present there are approximately 120,000 deaths from coronary heart disease in the United Kingdom per year. Each year in the United States approximately 800,000 individuals suffer an acute myocardial infarction, of whom approximately 550,000 die. More than half of the 550,000 deaths occur outside the hospital. Two thirds of the deaths from coronary artery disease of those aged less than 65 years are unexpected and occur outside hospital. More than one half are sudden and occur within one hour of the onset of the first symptom. Two thirds of sudden coronary deaths occur among patients who do not have previous clinical or electrocardiographic evidence of coronary artery disease. Death is more likely to be sudden in younger individuals and sudden coronary death is more likely in men than in women.

Sudden death and ventricular fibrillation

Sudden death and ventricular fibrillation are considered synonymous since in more than 90% of sudden deaths outside hospital, ventricular fibrillation has been documented. Sudden death need not be a manifestation of an acute myocardial infarction but may represent a brief ischaemic episode with a high tendency to recurrence.

The electrical storm of ventricular fibrillation was probably known to Vesalius but Hoffa and Ludwig in 1850 provided the first clear description. Their investigations showed that electrical stimulation of the mammalian heart led to ventricular fibrillation and death. In 1887 the Aberdeen physiologist McWilliam suggested that sudden death was due to ventricular fibrillation and in 1889 indicated there was a high probability that ventricular fibrillation was the cause of sudden death in patients with angina pectoris. In 1911 Hoffman was the first to obtain an ECG in a patient showing ventricular fibrillation and in 1933 Hamilton and Robertson recorded ventricular fibrillation on the ECG during a fatal attack of angina pectoris in a patient. In 1939 Smith and Miller first documented ventricular fibrillation as the cause of sudden death in man following acute myocardial infarction.

Electric current applied to the heart and defibrillation

The application of electric currents to the heart using direct current derived from a Leyden jar commenced in the 18th century. Abildgaard in 1775, the Danish Veterinarian – Physician, recorded that chickens could be stunned and revived by electrical shocks administered to the head and to the heart. Benjamin Franklin in the United States at approximately the same time shocked goats and killed fowl with charges of static electricity.

Prevost and Battelli in 1899 successfully defibrillated the ventricles of a dog’s heart by applying either DC or AC countershock directly to the myocardium. In 1933 Hooker et al showed that internal cardiac massage and the direct application of AC shock to the ventricles corrected experimental ventricular fibrillation. But it was not until 1947 that Beck and co-workers successfully corrected ventricular fibrillation by internal cardiac massage and direct application of AC countershock to the heart of a 14 year old boy undergoing thoracic surgery.

Time had been shown to be a critical factor in determining recovery from cardiac arrest. In 1940 Weinberger et al looking at clinical recovery from cardiac arrest noted that if more than four minutes elapsed before resumption of an adequate cardiac output the chances for salvage without irreversible neurological damage were poor. Thus the widespread application of resuscitative measures was limited.

Mouth to mouth respiration and external cardiac massage

In 1743 Tossach demonstrated the effectiveness of mouth to mouth respiration as a means of artificial ventilation by resuscitating a coal miner overcome by gaseous fumes. The American physiologist James Elam was the first to prove that expired air was sufficient to maintain adequate oxygenation. During a poliomyelitis epidemic in Minnesota in 1946, Elam first carried out mouth to nose breathing on patients with acute bulbar poliomyelitis demonstrating their survival until transfer to mechanical ventilation.

The problem however that was still outstanding was extending the survival time of the collapsed patient in ventricular fibrillation until external defibrillation was available. In 1960
Kouwenhoven et al at the Johns Hopkins Hospital in Baltimore first showed that external cardiac massage with compression of the lower portion of the sternum during cardiac arrest maintained an adequate circulation thus lengthening the delay in effective resuscitation by more than four minutes and removing the feeling of uselessness when confronting a crisis in which a delay of four minutes was the limit for effective resuscitation. They produced peak blood pressures of 80 mm Hg by closed chest cardiac massage. Thus by utilizing external cardiac massage and mouth to mouth ventilation, survival time to successful defibrillation was prolonged and ultimately the survival of the patient.

Defibrillation (internal and external)

In 1956 Reagan and co-workers and Beck and co-workers first reported successful resuscitation in hospital from ventricular fibrillation complicating acute myocardial infarction by internal cardiac massage and direct defibrillation of the heart. Yet in 1961 there were fewer than 20 reports of patients successfully resuscitated not surprisingly since the chest wall required to be opened and access for defibrillation to occur. During this time sudden death was thought to be associated with very severe coronary artery disease and therefore resuscitation would be unsuccessful despite the pathological studies showing that the myocardial damage was not infrequently small. Despite the statement by Burns in 1809 “Where, however, the cessation of vital action is very complete, and continues long, we ought to inflate the lungs, and pass electric shocks through the chest: the practitioner ought never, if the death has been sudden, and the person not very far advanced in life, to despair of success, till he has unequivocal signs of real death”, and the demonstration by several workers that experimental ventricular fibrillation could be removed by transthoracic countershock, essential application was delayed until 1956. In that year Zoll and co-workers corrected ventricular fibrillation by an externally applied electric countershock to the thorax ie AC shock and in 1962 Lown and co-workers introduced direct current shock (capacitor discharge) in the successful correction of ventricular fibrillation externally. Thus, resuscitative measures were available that could be rapidly applied by trained medical and auxiliary staff in the correction of ventricular fibrillation.

RESUSCITATION – THE PRESENT

Cardiac Arrest 1966 - 1969 (Belfast)

Looking at our first 193 cases of cardiac arrest outside hospital in Belfast from January 1, 1966 – December 31, 1969 where median onset of symptoms to arrest was 10 minutes, it was noted that when resuscitation was started within four minutes of the collapse and was efficient, of the 50 patients, 46 were in ventricular fibrillation of whom 40 (87%) were initially resuscitated. However if no resuscitation took place within four minutes of collapse then the majority of the 106 patients were in asystole ie 85 and only 21 in ventricular fibrillation. Of these 21 in ventricular fibrillation, only one patient became a temporary survivor. Of the 193 cases of cardiac arrest outside hospital, in 139 the cardiac arrest took place in the patient’s home. This is the most frequent area for cardiac arrest to occur and clearly the most difficult area to access with urgent medical care. In 106 of these 193 cases, the initial resuscitation was commenced by varying people including first aid workers and the family doctors. Fifty five of the 193 cases were initially successfully resuscitated outside hospital of whom 38 became longterm survivors.

Long term survivors

It was also important to show that survivors to leave hospital had a good outlook. In 1970 during a follow up period of 36 months we found that those who had ventricular fibrillation within 4 hours of the onset of symptoms were younger, usually had had a mild coronary attack and had the most favourable longterm prognosis in comparison with those in whom ventricular fibrillation occurred later.

Initiation of ventricular fibrillation

The initiation of ventricular fibrillation which could have relevance to its defibrillation characteristics we first described in 1982. This was a series of patients managed outside hospital and who developed ventricular fibrillation shortly after monitoring commenced. The most frequent initiating beat was an R on T ectopic ie on the peak of the T wave of the QRS of the patient there was a wide complex QRS (ventricular ectopic beat). Other methods of initiation but less frequent were a late cycle ventricular ectopic, ventricular tachycardia, ventricular flutter or idioventricular rhythm.
Ventricular defibrillation

Whilst ventricular fibrillation in a human rarely self-terminates, if the initiation of ventricular fibrillation is witnessed, a chest thump has a reasonable chance of success ie blow to the lower part of the sternum depressing it approximately a half an inch. However the majority of patients require external defibrillation in order to correct ventricular fibrillation.

The most important factor influencing survival is the delay to defibrillation. In the early days of ventricular defibrillation, the majority of studies advocated the maximum stored energy of the defibrillator ie 400 W seconds. From this stored energy most commercially available defibrillators delivered 270 to 330 W seconds through a resistance of 50 Ω. It was originally thought that depolarisation of every cell in the ventricles was necessary to terminate ventricular fibrillation. However it has been shown that successful defibrillation occurs when a critical mass of myocardium is depolarised.

Our earliest research in external defibrillation indicated that the position of the paddles/pads and their size ie circumference were essential in ensuring first DC shock success. For the anterior approach the upper paddle/pad requires to be under the right clavicle and to the right of the sternum in approximately the second intercostal space and the apical paddle/pad requires to be placed over the region of the apex of the left ventricle otherwise defibrillation is unsuccessful. In 1987 we showed that the greater the overall pad diameter the higher the percentage successful defibrillation.24 This was related to the transthoracic impedance which was lowest for the largest pads. Of the pads tested those producing the greatest percentage success with the lowest trans thoracic impedance measured 12 cms diameter.24 The number of shocks to correct ventricular fibrillation at the time of the initial arrest averages 2.25

In 1975 the Americans felt that "For 7 out of 10 patients, the present defibrillators may be powerful enough. But more important, for 3 out of the 10 it may not be". We therefore addressed this in 1975 and found no difference in the percentage success rate to 200 W S (stored) DC shocks for ventricular fibrillation for those in the heavy weight ranges ie > 90 kgs in comparison to those < 90 kgs.26 A single 200 W S shock was associated with a 85% initial first shock success.27

The energy used was half that previously proposed and is now standard for DC defibrillation (damped sine wave).

Since the majority of cardiac arrests occur in the patients’ home, the automatic detection of cardiac arrest rhythms was crucial, with either semi-automatic or automatic defibrillation availability. In 198628 using a Microprocessor-based ventricular fibrillation detection system, ventricular fibrillation was detected and a shock advised. This advice only took a matter of seconds. Thus public access defibrillation was initiated. We looked at other techniques for defibrillation using even lower energies for defibrillation thus reducing the size of the defibrillator capacitor. In 1989 we explored the oesophageal access route for defibrillation and showed ventricular fibrillation initiated in the Electrophysiological Laboratory was corrected by 50 joules delivered transoesophageally.29

Trans-telephonic defibrillation

In 1987 to improve further accessibility to defibrillation we tested the world’s first trans telephonic defibrillator where the detection circuitry for ventricular fibrillation was carried in a briefcase and after two pads had been placed on the chest wall the ECG could be transmitted by digital telephone to a central station where the ECG rhythm could be viewed by trained personnel and defibrillation or not advised.30

Impedance cardiography

There are many rhythms associated with cardiac arrest – some shokcable and some not. To ensure that the rhythm is shokcable it is imperative that the rhythm disturbance is associated with a low or no cardiac output. Whilst public access defibrillation is commendable there have been reports of the inappropriate delivery of direct current shocks to patients without ventricular tachyarrhythmias. The use of automatic external defibrillators by minimally trained personnel who lack the skills to differentiate cardiac arrest from other causes of collapse will increase the potential for inappropriate defibrillation. Clearly, the inclusion of a non-ECG haemodynamic sensor in an automatic external defibrillator device could increase its specificity.

Impedance cardiography is a non-invasive method for measuring cardiac output, and parameters derived from the impedance cardiogram have been used as indices of myocardial contractility.

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and aortic blood flow. The impedance of the thorax (Z) can be recorded by passing a high frequency low amplitude current between two electrodes and recording the resulting voltage. Ejection of blood into the aorta causes small fluctuations in this impedance (ΔZ). The impedance cardiogram is a recording of the first time derivative of ΔZ (ie dz/dt) against time.

Traditionally the impedance cardiogram has been recorded using four circumferential band electrodes. The upper voltage electrode is placed around the base of the neck and the lower voltage electrode around the thorax at the level of the xiphisternum. The two outer current electrodes are placed at least 3 cm away from the voltage electrodes. Clearly it was not practical to employ such an electrode configuration in the cardiac arrest setting. Thus, a system was developed in which the impedance cardiogram could be recorded through two ECG/defibrillator pads, one placed at the second right intercostal space to the right of the sternum just beneath the clavicle and the other placed over the fifth left intercostal space in the mid-clavicular line.

The purposes of this research were firstly, to compare the impedance cardiogram recordings using a traditional four band electrode technique with the novel two ECG/defibrillator pad technique and secondly, to determine if one or more of the impedance cardiogram parameters, recorded through two ECG/defibrillator pads, could act as a haemodynamic sensor for an automated external defibrillator. The impedance cardiogram was recorded in 20 male subjects in sinus rhythm using sequentially the traditional four-band electrodes and the new two ECG/defibrillator pad technique.

Over a 14-month period, simultaneous recordings of the ECG and impedance cardiogram were made at 116 cardiac arrest calls in 110 patients. The baseline characteristics age, sex, site of arrest, delay to cardiopulmonary resuscitation and delay to intensive care were documented for each of the patients. There were 39 females and 71 males. The age of one out-of-hospital arrest patient was not known. Of the remaining 109 patients the mean age was 66 years (range 38-87 years). Sixty cardiac (5 1.7 %) arrest calls were attended by the mobile coronary care unit outside hospital and 56 (48.3 %) were inside the hospital.

In the control subjects the ECG, impedance cardiogram and baseline impedance (Z) were recorded using a prototype device. This device could use either the four-electrode technique or the new two ECG/defibrillator pad technique. It passed a high frequency (64kHz) low amplitude constant AC current (1 mA RMS) between two electrodes (ie the outer two band electrodes in the four electrode technique and the two ECG/defibrillator pads in the two electrode technique) and recorded the impedance cardiogram through two electrodes (ie the inner two band electrodes in the four electrode technique or the two ECG/defibrillator pads in the two electrode technique). The signals were digitized and stored on a portable computer for subsequent analysis using a commercial software package.

The circuitry for the two ECG/defibrillator pad technique was then incorporated into a portable ECG/impedance cardiogram recording unit and hardware was added to protect the device from high voltage direct current shocks. This unit could be connected to each portable defibrillator using a locking plug. Again this unit passed a high frequency (64 kHz) low amplitude constant AC current (1 mA RMS) between the two ECG/defibrillator pads. The ECG and impedance cardiogram were detected and the signals simultaneously digitized and stored on memory cards for analysis off-line.

Immediately on arrival at a cardiac arrest patient, cardiopulmonary resuscitation continued or was initiated by the junior doctor manning the out-of-hospital mobile coronary care unit or attending the in-hospital cardiac arrest and ECG/defibrillator pads positioned as for cardiac arrest management.

The impedance cardiogram waveform has A, C and O waves and contains B, X and Y points (figure 1). The A and C waves occur during atrial and ventricular systole, respectively, and the O wave corresponds with ventricular filling. The B point has been related to aortic valve opening and the X and Y points to aortic and pulmonary valve closure, respectively.

Impedance cardiogram recordings are sensitive to motion artefact and electrical interference and thus subjected to ensemble averaging. Analysis software was written and the peak of the R wave of the ECG was identified and used as a reference point. The ECG and impedance cardiogram signals were digitally sampled over five cardiac cycles and synchronized with the R wave. The synchronized cycles were digitally summed and

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Fig 1. Impedance cardiogram with simultaneous ECG recording at 25 mm/s during sinus rhythm. A=A wave representing atrial systole, B=B point corresponding with aortic valve opening, C=C wave representing ventricular systole, X=X point corresponding with aortic valve closure, Y=Y point corresponding with pulmonary valve closure and O=O wave representing ventricular filling. ICG= impedance cardiogram.

averaged to provide the ensemble averaged complex. In the cases of ventricular fibrillation no R waves were present and ensemble averaging was performed using the peaks of the fibrillatory waveform that were greater than a threshold value (0.2 mV) as reference points. Similar amplitude criteria are employed in the detection algorithms of current automatic external defibrillators. In agonal rhythm, the peak of the ECG complex was used as a reference point. In asystole no reference point was available and averaging was performed at second intervals over a 5 s period.

Using the same analysis software, features of the ensemble averaged complex were manually extracted. These features included Peak dz/dt (the peak of the impedance cardiogram measured from the line dz/dt=0 Ωs⁻¹), Peak-trough (the peak-to-trough measurement of the impedance cardiogram Ωs⁻¹), Area 1 (the area under the C wave of the impedance cardiogram above the line dz/dt=0 mΩ) and Area 2 (the area under the impedance cardiogram 50 ms on either side of the Peak and above the line dz/dt=0 mΩ) (figure 2).

The analysis software also measured the R-R interval enabling a heart rate to be calculated. No R-R interval exists in either asystole or ventricular fibrillation. In agonal rhythm the interval was measured between the peaks of two consecutive complexes.

In the control group, Peak dz/dt measured using the two-electrode technique was correlated with the four-electrode technique. Baseline impedance (Z) and Peak dz/dt in the two groups were compared using the paired t-test.

The arrest rhythms encountered were divided into two groups: Group 1 contained rhythms associated with haemodynamic collapse ie no pulse – asystole, ventricular fibrillation, agonal rhythm and electromechanical dissociation or

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shockable ventricular tachycardia as defined by ventricular tachycardia associated with loss of consciousness, pulselessness or a systolic blood pressure of less than 80 mm Hg. Group 2 contained rhythms of patients where a cardiac arrest call was initiated but on arrival with the patient sinus rhythm was present in five (respiratory arrest) and in 20 with non-shockable ventricular tachycardia the patient was conscious with a pulse.

There was a significant correlation between Peak dz/dt measured using the four-band electrode technique and the two ECG/defibrillator pads method (r=0.61, 95% confidence interval 0.43 to 0.83, p<0.01). However, the two ECG/defibrillator technique resulted in significantly greater values of Peak dz/dt and Z (1.540 ± SD 0.649 vs 0.908 ± SD 0.192 Qs⁻¹, P < 0.001 and 65.7 ± SD 13.9 vs 22.8 ± SD 2.8, P < 0.001 respectively).

Of the 116 recordings, nine were rejected because of severe motion artefact and/or electrical interference. No recordings were rejected as a result of other adverse physical circumstances. Of the remaining 107 cardiac arrest calls, the rhythm initially recorded was: asystole in 19 (17.8%), ventricular fibrillation in 14 (13.1%), agonal rhythm in 20 (18.7%), electromechanical dissociation in 22 (20.6%), shockable ventricular tachycardia in 7 (6.5%), non-shockable ventricular tachycardia in 20 (18.7%) and sinus rhythm in five (4.7%). For each impedance cardiogram parameter there was a progressive fall in the mean value from sinus rhythm to non-shockable ventricular tachycardia to shockable ventricular tachycardia to each of the pulseless rhythms (ie electromechanical dissociation, agonal rhythm, ventricular fibrillation and asystole).

There was no significant difference between Groups 1 and 2 with regard to age or sex. There were significantly more out-of-hospital arrest calls in Group 1 and a significantly greater delay to cardiopulmonary resuscitation and intensive care. The mean initial heart rate, where assessable, was significantly greater in Group 2 and each of the impedance cardiogram-parameters was greater in Group 2 (ie Peak dz/dt, Peak-trough, Area 1 and Area 2).

Using multiple logistic regression the variables age, sex, heart rate, Peak dz/dt, Peak-trough, Area 1 and Area 2 were removed in a stepwise fashion until statistical significance was reached. The two parameters which best predicted a low or absent cardiac output (Group 1) were Area 1 and the Peak-trough measurement. Using these two parameters 78 of 82 (95.1%) patients in Group 1 and 20 of 25 (80%) patients in Group 2 were correctly classified. In particular the higher the value of the peak-trough of the impedance cardiogram the less likely is the rhythm to be shockable. This should help confirm the decision making process where the lay public alone are using the defibrillator.

**Follow-up**

In 1991 we looked at a 20 year follow up of our out-of-hospital cardiac arrest patients with ventricular fibrillation. The major factors contributing to in-hospital mortality were cardiogenic shock after defibrillation, coma on hospital admission, age ≥ 60 years and ≥ 4 shocks to correct ventricular fibrillation. After five years follow up, 41% were alive.

**Aetiology of cardiac arrest in the community**

Whilst ischaemic heart disease is the commonest cause of cardiac arrest, nevertheless there are other causes. One that is increasing in frequency is that seen following substance abuse and drug overdose. In this city two 16 year old males have survived resuscitation from cardiac arrest associated with either butane gas inhalation or glue sniffing. It is well known that the mortality from volatile substance abuse in the United Kingdom has its highest incidence in those aged 15-19 years. The most frequent solvents commonly encountered in abuse related deaths are fuel gases – cigarette lighter refills (butane), propane and gasoline, and typewriter correction or dry cleaning fluids or that contained in fire extinguishers. Others less frequently inhaled are adhesives (toluene and aerosol propellants ie Halons and/or Butane). The mechanism of cardiac arrest from substance abuse can be either a cardiac arrhythmia (major risk), or anoxia with respiratory depression and vagal stimulation, or aspiration of vomit or trauma or a combination of all three.

It is estimated that 220 out-of-hospital cardiac arrests occur among 500,000 people each year and with present resuscitation facilities 30-40 individuals should leave hospital alive. Ventricular fibrillation despite being a chaotic rhythm has a peak frequency and this peak is higher the shorter the time from onset of ventricular fibrillation to successful defibrillation.
RESUSCITATION – THE FUTURE

Defibrillators for out-of-hospital use are getting lighter with the minimum of controls for the lay public. One of these is now produced in Belfast (Samaritan Defibrillator) and is shown in Figure 3 where after the two electrode pads are placed in the appropriate positions on the chest wall and the monitor turned to on, and following the advice to defibrillate for ventricular fibrillation the shock can be delivered by pressing the third button. There is a facility to download the information on arrival in hospital and thus a hardcopy write out can be obtained.

In the words of Hilaire Belloc (1870-1953) “For a Sun Dial” – Loss and possession, death and life are one, there falls no shadow where there shines no sun. Both death and life are one in the process of resuscitation and the sun still continues to shine casting a shadow pointing to newer developments with the potential to improve the longterm survival for victims of cardiac arrest.

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