

# What is more important: defibrillation or compression?

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Cardiac arrest (CA) is one of the leading causes of death and is responsible for approximately 400,000 deaths per year in the United States (1). A recent investigation (American Heart Association) showed that more than 200,000 inpatients had CA in 2013 in the United States (2). China has approximately 540,000 deaths from CA each year, ranking first in the world. However, the success rate of rescue for CA is extremely low and the success rate of prehospital resuscitation for patients with CA is only 4.2% in the capital of China, Beijing (medically developed area) (3).

Ventricular fibrillation (VF) is the leading cause of CA, and defibrillation is the best treatment option for terminating VF. Early defibrillation is critical for improving the survival rate in VF, because in chest compression alone, terminating VF and restoring spontaneous circulation are almost impossible. Moreover, the defibrillation success rate of VF, the rate of return of spontaneous circulation, and the success rate of resuscitation rapidly decrease with an increasing duration of VF. Experimental data show that successful defibrillation is closely associated with the defibrillation time; for every minute that defibrillation is delayed, the success rate of rescue for CA decreases by 7–10% (4).

Although a consensus has been reached on the importance of early first defibrillation, controversy still remains on the timing and strategy for the second defibrillation. The guidelines for defibrillation have also undergone several revisions. The earliest 2000 resuscitation guidelines recommend the use of “stacked” shocks, comprising a second defibrillation within the shortest time after success of the first defibrillation attempt. The 2005 revised guidelines recommend a delayed second defibrillation attempt. Adequate chest compression between

two defibrillations can prevent a decline in compression time and quality because of multiple defibrillations, thus improving the cure rate of CA.

Researchers from the United States recently published their trial results in the *BMJ* and raised new questions about defibrillation therapy in the current CA resuscitation guidelines (5). These questions were raised because a recent trial showed that resuscitation treatment following the current guidelines failed to improve patients’ survival. This trial was conducted using a retrospective cohort study, which found that 1,121 (41%) patients received a delayed second defibrillation, among 2,733 patients with in-hospital, sustained ventricular tachycardia (VT)/VF after the first defibrillation. After risk adjustment, there was no association between the delayed second defibrillation and patients’ survival to discharge from hospital. Therefore, the authors suggest that for the treatment of sustained VT/VF, a delayed second defibrillation does not improve the survival rate, but doubles the hospitalization rate of patients (5).

The above-mentioned results are not completely contrary to the guidelines. The Bradley’s study included subjects from adult patients with in-hospital sustained VT/VF who received a second defibrillation (6). CA mostly occurs outside the hospital. Therefore, delayed defibrillation recommended by the current cardiopulmonary resuscitation (CPR) guidelines is mainly based on patients with out-of-hospital CA. The recognition time is often longer and the first defibrillation is relatively late in patients with out-of-hospital CA compared with those with in-hospital CA. Therefore, patients with out-of-hospital CA have a longer duration of VF and a lower resuscitation success rate than those with in-hospital CA. There are various differences in

the pathological processes of VF with varying time courses, and research results and resuscitation measures may also be different.

In 2002, Weisfeldt *et al.* proposed the three-stage theory of VF leading to CA (6). Stage I is an electrophysiological disorder (0–4 min) where the heart stops pumping blood. This is due to elastic recoil of large vessels and the potential of blood flow. Myocardial blood perfusion is not completely cut off and some myocardial energy supply remains in the myocardium. After defibrillation, cardiac action potentials can easily reach consistency, achieving a relatively high success rate. Stage 2 is a circulatory disorder (4–10 min) where blood perfusion is completely cut off and the myocardium relies on anaerobic metabolism for energy supply. After defibrillation, cardiac action potentials are inconsistent, thus lowering the success rate. The focus of resuscitation in this stage is to ensure adequate perfusion of the heart and brain, among various vital organs. Stage 3 is a metabolic disorder (>10 min) where accumulation of products of anaerobic metabolism and acid-base imbalance lead to severe myocardial damage and an extremely low defibrillation success rate. For long-duration VF (>7 min), the body is in the stage of a circulatory or metabolic disorder. Adequate cardiac compression can provide better perfusion of the brain and heart and further improve myocardial contractility. Cardiac compression can also supply sufficient energy to the myocardium for dealing with the shock of defibrillation and thus further improve the resuscitation success rate. The mechanism of this process may be associated with an increase in coronary perfusion, an improvement in cardiac energy reserve, and an elevation in the frequency and amplitude of VF waves (7,8).

To date, good therapeutic effects have been achieved for short-duration VF within 3–5 min. However, the effects remain unsatisfactory for long-duration VF of greater than 7 min, which seriously affects the overall effect of CA resuscitation. In patients with CA with a VF duration of longer than 7 min, performing continuous cardiac defibrillation significantly shortens early crucial chest compression time and markedly reduces the effective cardiovascular perfusion time. This situation is unfavorable for improving the resuscitation success rate. Tang *et al.* showed that one-shock defibrillation significantly improved the resuscitation success rate in pigs with long-duration VF compared with continuous three-shock defibrillation (9). An animal study also demonstrated that after the use of early defibrillation and compression, defibrillation showed no

significant difference in improving the resuscitation success rate in long-duration VF (8 min). Two clinical trials on CPR for patients with in-hospital and out-of-hospital CA showed that because of the extensive use of automatic external defibrillators, chest compression time only accounted for 51–76% of the total CPR time (10–12). Additionally, the use of three successive shocks led to a delay of 37 s from the first shock to the first chest compression, and thus significantly shortened critical early cardiovascular perfusion time in patients with CA (10–12). One-shock defibrillation can extend the CPR time compared with continuous defibrillation. Therefore, one-shock defibrillation may achieve better effects than continuous defibrillation by improving the timeliness of CPR in long-duration VF.

The experimental results of this study were based on the GWTG-R registry (5). The sample size was large, the data were informative and reliable, and the research fully complied with the guidelines. This study had an important impact on clinical practice, and such research is presently rare. However, this was a retrospective cohort study with inherent limitations. This observational study involved various confounding factors, which were unable to be completely removed, despite adjustment using statistical methods. Moreover, this study used the rate of survival to discharge from hospital as the end-point. Notably, the rate of survival to discharge from hospital was associated with various factors, especially in inpatients with VF. Other confounding factors included the control of resuscitation quality, a change in the compression and ventilation ratio, and accurate calculation of the defibrillation time, which also affected the experimental results. Nevertheless, the research of Dr. Bradley and colleagues emphasizes the importance of further knowledge and attempting to improve the guidelines.

The development of clinical guidelines is a constant improvement and developmental process based on continuous research and exploration by clinicians. With the development of medical science, the current guidelines will not remain static. In the future, when previous guidelines are referred to, they may appear to be incorrect or inappropriate, which reflects the continuous development of medical research. More clinicians need to be encouraged to continue to perform research to improve and revise the guidelines.

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### Footnote

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