Interposed Abdominal Compression–CPR
Low Technology for the Clinical Armamentarium

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W hen Kouwenhoven et al.1 introduced the technique of closed-chest cardiac massage in 1960, they wrote, perhaps somewhat exuberantly, that “anyone, anywhere can now initiate cardiac resuscitative procedures. All that is needed are two hands.” It is somehow fitting that in this era of high technology medicine more than 30 years later, a promising advance in resuscitation technique, reported by Sack et al2 in this issue of Circulation, also involves use of the hands. In this sense, the technique of manual interposed abdominal compression–CPR (IAC-CPR)3 represents a novel “low technology” approach to health care at the opposite end of the complexity and cost continuum from high technology imaging.

The most recent report by Sack and coworkers2 describes the efficacy and safety of this relatively simple and practical modification of cardiac life support—the addition of manual IAC to otherwise-standard CPR—in a population of exceedingly difficult-to-resuscitate patients discovered with ECG findings of asystole or electromechanical dissociation (EMD). Sack et al’s data, demonstrating 33% 24-hour survival in such patients treated with IAC-CPR compared with 13% in a well-matched control group receiving standard CPR, represent a 2.5-fold improvement. Such results certainly merit careful attention.

Like high-technology life support systems, however, low-technology IAC-CPR has the potential to produce life prolongation without return of consciousness or quality of life. Although Sack and coworkers suggest in their conclusion, as has Steuven,4 that the ECG recognition of asystole or EMD might be a specific indication for IAC-CPR, none of their patients left the hospital alive and neurologically intact. Therefore, from the point of view of the patient and the patient’s family, there is little reason to support such an indication at the present time. In both the preclinical laboratory3,5,6 and the clinic,7,8 the addition of manual abdominal counterpulsation to otherwise conventional CPR has been shown to approximately double indicators of artificial circulation generated during resuscitation. Such hemodynamic improvement very likely occurred in Sack et al’s patients with asystole or EMD sufficient to enhance the probability of return of spontaneous circulation from 28% to 49%. The dismal long-term results, however, continue to remind us that the major rationale for continued resuscitative efforts in such patients is to make a reasonable attempt to exclude an incorrect diagnosis of asystole or of EMD—either because fine ventricular fibrillation is masquerading as asystole in the particular ECG lead used for monitoring9 or because of the inability to palpate a weak pulse that is actually present.10 Once such a false-positive diagnosis is excluded, even IAC-CPR, which has been shown to more than double long-term survival to hospital discharge in a more general population of hospitalized cardiac arrest victims,11 produces in patients with asystole or EMD only temporary return of the circulation, prolongation of hospitalization with attendant costs, and, occasionally, survival with severe and permanent neurological deficits. Heroic efforts are not justified in such patients until we learn to better resuscitate the brain as well as the heart.

The real virtue of Sack et al’s study is not in having discovered a way to meaningfully revive patients with asystole and EMD but rather in having tested a practical modification of cardiac life support—IAC-CPR—in perhaps the most difficult clinical model available: a population of patients for whom the probability of a satisfactory long-term outcome essentially is zero.12 The fact that they were able to achieve any improvement at all, albeit short-term, supports the robustness and efficacy of manual abdominal counterpulsation as a simple, low technology adjunct to conventional CPR.

In terms of safety, their findings confirm previous work8,11,13 suggesting that, unlike simultaneous chest and abdominal binding,14 manual abdominal counterpulsation does not lead to intra-abdominal trauma, probably because the liver in particular is free to move in response to transphrenic pressure gradients. Sack and coworkers speculated that a bare-handed technique causes better coupling of pressure from the skin surface to the great vessels than the original method,3,5 in which the air bladder of a blood pressure cuff was placed between the compressing hands and the abdomen to monitor and limit applied pressure. If the risk of causing internal injury is minimal, then the coupling bladder is not needed, and an even more simple and effective technique of abdominal counterpulsation may be possible.

Another virtue of Sack et al’s recent study is the apparent demonstration of a practical means of greatly improving coronary perfusion during resuscitation noninvasively through the combination of simultaneous epinephrine administration and IAC-CPR. Previous work in electronic models of the circulation has shown true synergy between peripheral vasoconstriction and abdom-

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inal counterpulsation\(^6\) in the sense that the increase in coronary perfusion pressure produced by both interventions together was greater than the sum of the increases produced by either alone. In vivo, there is the further effect of coronary vasodilation mediated by stimulation of \(\beta\)-adrenergic receptors. Although the authors did not directly measure myocardial perfusion, the improved short-term survival, despite a 41% incidence of coronary artery disease in the study population, provides convincing evidence that improved coronary perfusion pressures and myocardial blood flow did occur, extending prior preclinical\(3,5,6,15\) and clinical\(8\) work. This capability to establish improved perfusion of the myocardium—and probably the brain as well—during resuscitation could lead to better delivery of novel resuscitative drugs. Consider, for example, the following.

Physiologically, EMD and asystole probably most often represent posts ischemic myocardial dysfunction, also known as "myocardial stunning," following the global ischemia of cardiac arrest. In the laboratory model of myocardial stunning, a heart is rendered ischemic for a period of time such as 15 minutes. Thereafter, perfusion is restored, and myocardial contractility is profoundly but temporarily depressed. Experimental asystole and EMD are produced during the early, extreme phases of stunning at the beginning of reperfusion and can be partially reversed in the isolated heart by continued coronary perfusion with oxygen-replete medium. This appears to have been what was achieved in Sack et al's patients who were resuscitated by IAC-CPR.

There is reasonable experimental evidence\(16,17\) that free radical mechanisms involving the reactive oxygen species superoxide and hydrogen peroxide, together with the redox cycling of iron,\(18,19\) play important roles in the pathophysiology of stunning and related forms of reperfusion injury. In many models, stunning and reperfusion injury can be prevented by administration of antioxidants and iron chelators after the onset of reperfusion.\(20-22\) Assuming that IAC functions as a method to noninvasively perfuse the myocardium in patients with asystole or EMD—and so, too, to deliver drugs—it follows that possible combinations of anti stunning agents with IAC-CPR can be investigated clinically in patients with asystole or EMD.

Intriguingly, iron chelators have been shown to increase long-term survival in an animal model of asystole or EMD and resuscitation by IAC-CPR.\(24,25\) In this animal model, EMD or asystole is induced in rats by intracardiac injection of KCl and chest restriction to prevent autoresuscitation through gasping. Intravenous administration of the iron chelator deferoxamine at the onset of external chest and abdominal compression increases neurologically intact survival at 7 days from approximately 30% to approximately 60%. Were a similar effect to be demonstrated in human beings and were the interventions effective in resuscitating the brain as well as the heart, a real indication for novel therapy in cardiac arrest with asystole or EMD might well evolve.

**References**


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