REVIEW

Direct Cardiac Compression Devices

Mehmet C. Oz, MD,[a] John H. Artrip, MD,[a] and Daniel Burkhoff, MD, PhD[b]

Ventricular assist devices employed clinically to assist the failing heart require direct contact between the device and the patient's blood. Thromboembolic events, the need for anticoagulation, hemolysis, immune reactions and infections contribute significantly to morbidity and mortality of these devices. Compressing the weakened heart from its epicardial surface, direct cardiac compression (DCC), could provide ventricular support and avoid the interactions between blood and a foreign surface. This article reviews the physiology of epicardial compression and the current progress of mechanical compression devices. J Heart Lung Transplant 2002;21:1049–1055.

Treatment of end-stage heart disease remains a major clinical challenge and the repertoire of available medical therapies is somewhat limited and frequently ineffective, necessitating mechanical ventricular assistance. Although a variety of ventricular assist devices are available, most require direct contact with the patients blood; thus, thromboembolic events and the need for anti-coagulation, hemolysis and immune reactions are ever-present problems. Accordingly, there has been renewed interest in developing techniques to support the circulation by compressing the weakened heart from its epicardial surface—that is, direct cardiac compression (DCC).

Insights into the effects of DCC on ventricular pump function have been gained from early experience with biomechanical compression therapies such as dynamic cardiomyoplasty. These surgical procedures physically wrap the patients skeletal muscle around the failing heart and electronically stimulate the muscle wrap to contract with the native heart beat. Following the surgical procedure, there is 2-week recovery that allows adhesions to form between the heart and the skeletal muscle wrap, followed by a 6-week pre-conditioning period before the skeletal muscle is entrained for ventricular support. The high peri-operative mortality rate of this procedure and the lengthy pre-conditioning period required have virtually excluded New York Heart Association (NYHA) Class IV heart failure patients, which is the group of patients that would benefit most from ventricular assist. In addition, the use of the myostimulator precludes the use of pacemakers and internal cardiac defibrillators (ICDs) in a patient population prone to conduction disturbances and ventricular arrhythmias. For these reasons, investigators have turned to mechanically driven compression devices. The basic components of these devices consist of a compression-driving system, an electrocardiographic (ECG) sensor/digital controller and an epicardial "cup" or "cuff". Adaptations to these individual components have been used to design devices specifically for cardiopulmonary resuscitation, acute ventricular support and long-term ventricular support. Although no device is currently available for clinical use, there are several systems being developed and evaluated. Despite the differences between the various support systems, the physiologic principles governing their mechanism of action are essentially the same.

PHYSIOLOGY OF DIRECT CARDIAC COMPRESSION

Our understanding of the effects of DCC on ventricular mechanics stems from investigations utiliz-

From the [a]Department of Surgery, Division of Cardiothoracic Surgery and [b]Department of Medicine, Division of Circulatory Physiology, College of Physicians and Surgeons, Columbia University, New York.

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Reprint requests: Dr. Mehmet C. Oz, Department of Surgery, Division of Cardiothoracic Surgery, College of Physicians and Surgeons, Columbia University, MHB 7-435, 177 Fort Washington Avenue, New York, New York 10032. Telephone: 212-305-4434. Fax: 212-305-2439. E-mail: mco2@columbia.edu

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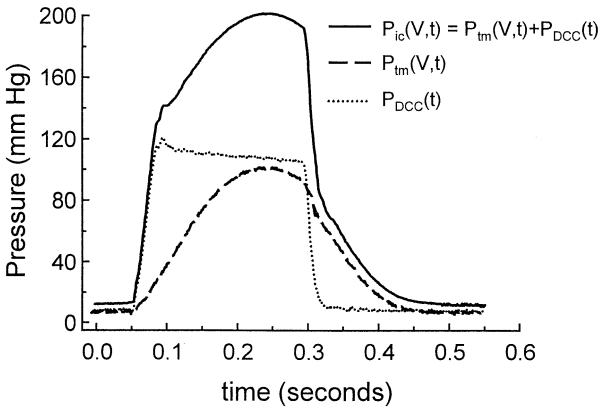

FIGURE 1 Left ventricular pressures obtained from an isolated canine heart undergoing synchronized epicardial compression under isovolumic ejection conditions. The pressure measured inside the ventricular cavity, $P_{ic}(V,t)$, is the sum of the transmural ventricular pressure, $P_{tm}(V,t)$, plus the epicardial compression pressure, $P_{DCC}(t)$.

ing isolated canine hearts placed inside compression chambers having pressure levels that could be varied in synchrony with cardiac contraction.[1–4] Results of these studies showed that net ventricular pumping capacity can be augmented by external pressure such that the forces applied to the hearts surface

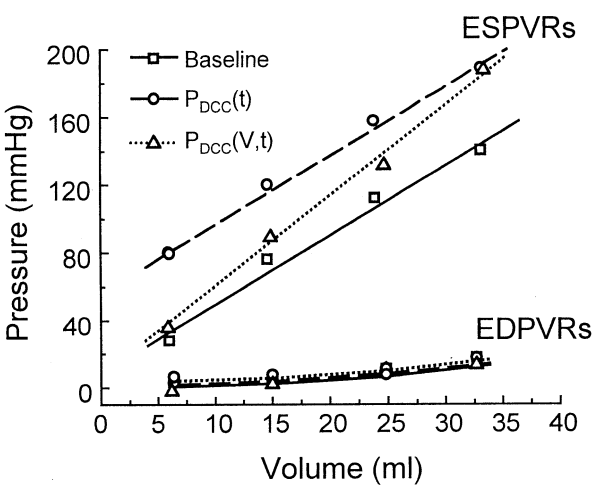

FIGURE 2 The effects of DCC on the ESPVR and EDPVR of the left ventricle of an isolated canine heart under isovolumic ejecting conditions. With compression pressures that are independent of ventricular size, $P_{DCC}(t)$, the ESPVR is simply shifted upward with no change in the slope; however, with compression pressures that vary with the ventricular size, $P_{DCC}(V,t)$, the slope of the line is significantly increased with little change in the volume-axis intercept. The EDPVR is not affect by epicardial compression.

add to the ventricular pressure generated by the contracting myocardium. This can be clearly demonstrated under experimental conditions whereby the ventricular volume remains fixed (i.e., isovolumic contractions), so that the strength of ventricular contraction is readily indexed by increases in ventricular pressure. Under these conditions, the pressure measured inside the ventricular cavity equals the sum of the pressure generated by the contracting myocardium (normally referred to as the transmural pressure) plus the pressure applied to the heart surface. This is illustrated in Figure 1 and can be expressed mathematically as:

$$P_{ic}(V,t) = P_{tm}(V,t) + P_{DCC}(t) \quad (1)$$

where $P_{ic}(V,t)$ is the intra-chamber ventricular pressure, $P_{tm}(V,t)$ the transmural ventricular pressure and $P_{DCC}(t)$ the pressure generated by direct cardiac compression.

Applying this principle over a series of fixed ventricular volumes, it becomes apparent that DCC shifts the end-systolic pressure–volume relationship (ESPVR) upward by an amount related to the compression pressure (Figure 2). With biologic wraps, the skeletal muscle used for epicardial compression is subject to myofibril length–tension variation; for this reason, the compression pressure (P_{DCC}) varies with ventricular size. As the ventricular size changes with filling volume, the length–tension relationship of the myofibrils of the muscular wrap changes, thus altering the amount of compression force delivered to the epicardium.[5] The ESPVR of a heart assisted with a skeletal muscle wrap is shown by the dotted line in Figure 2. The slope of the line (referred to as the end-systolic elastance, E_{es}) is significantly increased with little change in the volume-axis intercept (V_o).[6,7] With mechanical compression devices, $P_{DCC}(t)$ is independent of ventricular size, so that the amount of compression pressure applied to the hearts epicardial surface is the same regardless of the ventricular volume. Under these conditions, the ESPVR is simply shifted upward from the baseline by an amount equal to $P_{DCC}(t)$. The slope of the line is unchanged but the V_o value is decreased.[4] This is shown by the dashed line in Figure 2.

With the compression chamber used in the ex vivo canine heart experiment, the benefit on systolic function is achieved with no effect on ventricular diastolic function.[4] This is illustrated in Figure 2 by the similar EDPVR tracings for the assisted and unassisted hearts. Unfortunately, biologic wraps and mechanical compression devices designed for use in vivo may have

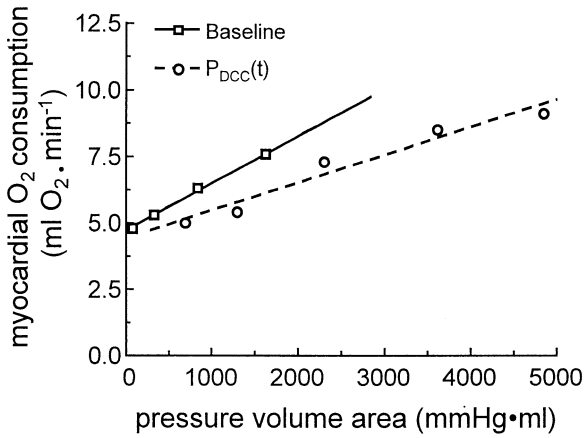

FIGURE 3 The effect of DCC on the relationship between myocardial O_2 consumption and external work. DCC decreases the slope of the mVO_2–PVA relationship with little influence on the y intercept.

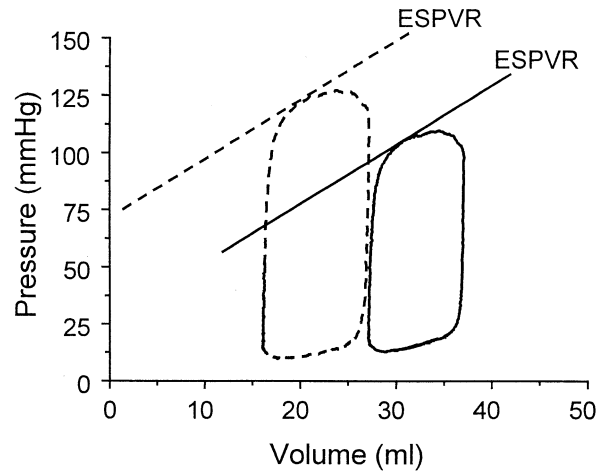

FIGURE 4 The effects of DCC on the left ventricle pressure–volume (PV) relationship of an isolated canine heart under computer-simulated physiologic ejecting conditions. With application of DCC, the ESPVR is shifted upward from baseline (dashed vs solid line). Under conditions where neither pre-load volume or after-load resistance changes, the new PV relationship increases with a significantly increased stroke volume (dotted vs solid PV loop). However, pre-load volume is reduced during DCC and the PV loop shifts left-ward (dashed PV loop), thus reducing the augmenting effects of DCC on stroke volume.

small effects on ventricular diastolic properties. Effects on diastolic properties have been inconsistently observed among various investigators, but when present will induce small left-ward shifts of the EDPVR.[8,9] These shifts of the EDPVR are indicative of increased chamber stiffness that requires a higher filling pressure to obtain the same pre-load volume. Importantly, this effect was observed both with and without active compression, suggesting that the actual fixation of these wraps/devices to the heart account for this diastolic effect. Muscular wraps may also reduce the rate of diastolic pressure decay (negative dP/dt_{max}) and increase the time-constant (τ) for ventricular relaxation, which has not been observed with mechanical compression devices.[9,10] This is likely due to the relatively slow relaxation period of skeletal muscle as compared with cardiac muscle and the fact that the heart rests within the confines of the muscular wrap. The net effect of ventricular wraps and compression devices on the ventricular diastolic properties may lead to reduced ventricular pre-loads and potentially limit the degree of output augmentation obtained.[4,7,8]

Because DCC increases the ESPVR with little influence on the EDPVR, the overall effect is to increase the pressure–volume area (PVA) confined between these two curves. This area reflects the external work of the heart and usually correlates with myocardial oxygen consumption (mVO_2). With mechanical compression from the epicardial surface, however, the PVA increases, but the mVO_2 does not change.[3,4] This is shown in Figure 3 as a significant decrease in the slope of the mVO_2–PVA relationship with little influence on the y intercept. The slope changes of the mVO_2–PVA relationship observed with DCC do not reflect effects on myocardial properties, but rather the enhanced net pressure-generating capacity in the absence of an effect on intrinsic myocardial properties.

In contrast to isovolumic contractions, with physiologically ejecting conditions similar to those encountered in vivo, the increased pumping capacity of the ventricle with epicardial compression manifests not only as increases in end-systolic pressure, but also as increases in stroke volume.[4,8] However, pre-load volumes decrease as a consequence of the increased ventricular ejection, and for this reason the degree of pressure and stroke volume augmentation anticipated by DCC is reduced to what would be expected if pre-load volumes remained constant. This has been predicted from studies using isolated hearts ejecting against a computer-simulated physiologic afterload system,[4] and has been observed in vivo in studies using muscular wraps and compression devices.[7,8] Pressure–volume tracings illustrating the pre-load shift that occurs with the increased pumping capacity of DCC are illustrated in Figure 4. These observations can be explained by the use of modern theories of ventriculoarterial coupling,[11] predicting that the amount of stroke volume augmentation will be dependent upon

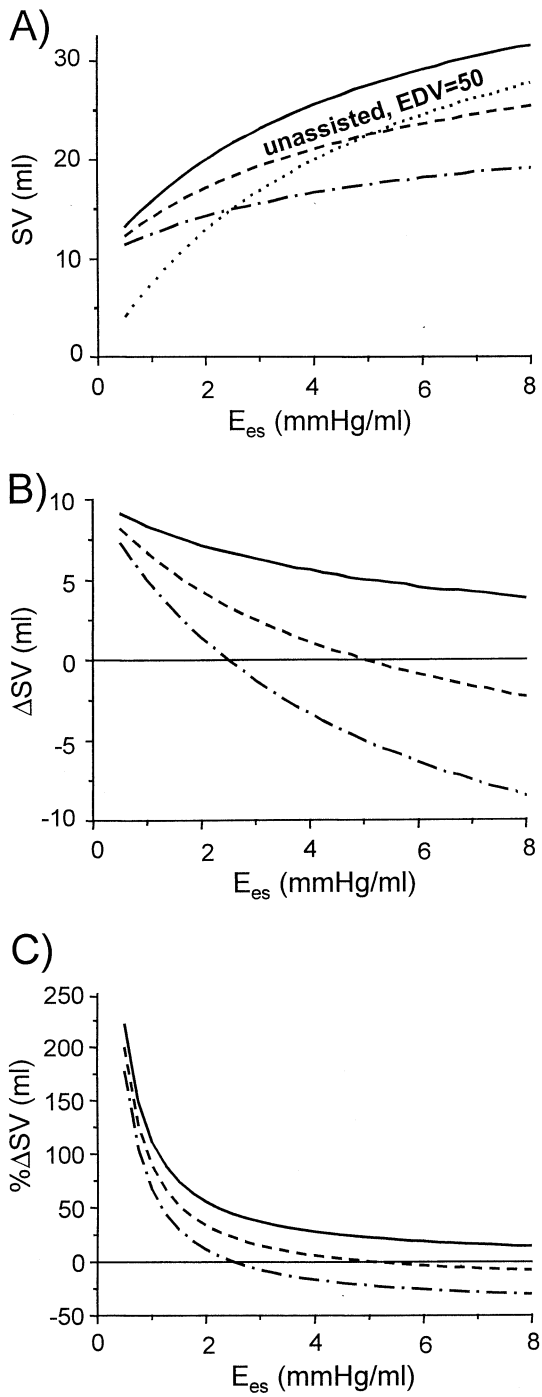

FIGURE 5 The effect of DCC on the relationship between stroke volume and ventricular contractility for three different pre-loads (EDV of 30, 40 and 50 ml) for a 24-kg adult male dog. Ventricular contractility is indexed as the E_{es} value, which is approximately 8 mm Hg/ml in a healthy adult male dog. Solid lines: EDV = 50 ml; dashed lines: EDV = 40 ml; dashed-dotted lines: EDV = 30 ml. The plots are: (A) stroke volume vs E_{es}; (B) change in stroke volume (Δstroke volume = stroke volume$_{DCC}$ − stroke volume$_{base}$) vs E_{es}; and (C) percent increase in stroke volume [%Δstroke volume = ((stroke volume$_{DCC}$ − stroke volume$_{base}$)/stroke volume$_{base}$) × 100] vs E_{es}. Stroke volume$_{DCC}$ is stroke volume with DCC and stroke volume$_{base}$ is the baseline stroke volume.

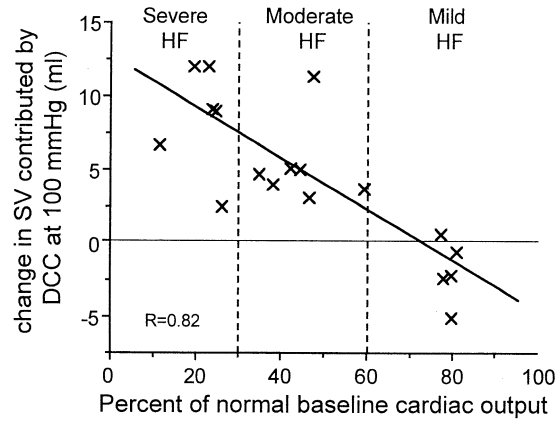

FIGURE 6 Plot showing the change in the stroke volume produced by DCC vs the degree of heart failure indexed as a percent of the normal baseline value. The correlation coefficient is 0.82.

the baseline myocardial contractile state, the baseline afterload resistance and the degree of pre-load shift caused by $P_{DCC}(t)$.[4]

The interrelationships between these parameters are further summarized in Figure 5 with the help of computerized modeling of the canine circulation. The dotted line in Figure 5A shows how stroke volume varies as a function of contractile state (indexed as E_{es}) at a fixed pre-load and a fixed after-load. The solid line at the top depicts how DCC would affect this relationship assuming that there was no change in pre-load. As pre-load decreases, however, this curve shifts downward, as shown by the dashed and dotted-dashed lines. Graphs depicting the amount of stroke volume augmentation (expressed in absolute and relative terms in Figure 1B and C, respectively) formally illustrate two fundamental aspects of the physiology of epicardial compression. First, the reduction in pre-load will blunt stroke volume augmentations and may even result in a diminution at higher baseline levels of contractility. Second, the amount of augmentation of effective ventricular contractile state afforded by DCC is a function of baseline contractile state. Substantial stroke volume augmentation can be achieved only in a weak heart (~40% normal). These principles have been verified in vivo with the use of a constant pressure epicardial compression device and a canine

model of heart failure in which graded levels of heart failure were achieved by coronary artery microembolization.[8] The plot in Figure 6 is taken from the in vivo experiments and is analogous to the plot in Figure 5B.

A full explanation of the physiology of DCC is still more complicated. DCC affects both ventricles equally and only a single ventricular analysis has been investigated. The intrinsic contractile strength and after-load resistance are lower for the right ventricle (RV) than the left ventricle (LV). Thus, the effects of DCC on the RV are proportionally greater.[4,8] Because under steady-state conditions the stroke volume must be approximately the same for both ventricles, the effect of DCC on the RV cannot translate into a larger stroke volume for the RV than the LV. The degree of cardiac output augmentation will depend upon the effects of DCC on the unequal pumping capacity and vascular resistance of the LV and RV, such that the ultimate degree of compression pressure to be used will be critically dependent on the effect of DCC on the RV.[8] That is, increasing compression pressure to values above those required to completely empty the RV will likely fail to further increase LV outputs.

CARDIOPULMONARY RESUSCITATION DEVICES

In 1965, Anstadt et al introduced an epicardial compression device that could administer efficient and sustained cardiopulmonary resuscitation (CPR).[12] This device, known as the Anstadt cup, is an elliptically shaped cup that fits over both right and left ventricular chambers. It has a semi-rigid outer shell and an inflatable inner diaphragm that delivers compression forces to the heart. Vacuum pressure (~70 mm Hg) at the apex of the heart is used to attach the cup to the heart and prevent migration of the device. Positive and negative pressure is generated within the inner diaphragm with the use of a pneumatic drive system able to deliver pulsed pressure. Because the inner diaphragm is tightly sealed to the epicardial surface, the device can provide diastolic decompression as well as epicardial compression. Cycle rates are fixed, but can be adjusted to provide the compressions at the desired rate. However, for technical reasons, the device was not designed to deliver epicardial compressions synchronized with the native heart beat. Although asynchronous ventricular assistance has been attempted, the increased frequency of rhythm disturbances and the potential for injurious effects to the myocardium have limited investigation of the utility of this device to the realm of CPR.

DEVICES DESIGNED TO ASSIST THE ACUTELY FAILING HEART

A ventricular support device designed to stabilize the acutely failing heart should be simple to apply in an unstable situation, freely adjustable to optimize ventricular outputs under a variety of clinical conditions, provide complete circulatory support in the event of cardiac arrest, and allow for easy removal following recovery of the failing heart. One system under development that meets these requirements is the CardioSupport system (Cardio Technologies, Inc, Pine Brook, NJ). The device has a "cuff-like" structure that is placed around the outside of the heart between the atrioventricular groove and the apex. Negative pressure (~200 mm Hg) applied to the apex of the heart is used to fix the device to the heart. The cuffs compression bladder circumscribes both ventricles around the base and is inflated in synchrony with the hearts natural contraction when providing cardiac support. The inside of the vacuum seal has two finely meshed electrode bands that provide a reliable ECG source needed for timing the inflation and deflation of the compression system. The electrodes can also be used for defibrillation.

Upon inflation of the cuff, the ventricular walls are compressed, expelling the blood from the ventricles. Deflation of the cuff allows the ventricles to fill with blood, which is expelled during the next cuff inflation. The compression force is provided by an air compressor and controlled with a computer console and electromechanical tether that regulates the amount, frequency and duration of the compression pressure to be delivered to the epicardium.

Individual parameters can be adjusted easily to optimize the ventricular assistance specific to the needs of the patient. In the CPR mode, the system inflates and deflates the cuff at a fixed rate that can be modified by the operator.

DEVICES DESIGNED FOR LONG-TERM VENTRICULAR SUPPORT

Epicardial compression devices designed for chronic ventricular support should be both reliable and portable. The Heart Booster (Abiomed, Inc, Danvers, MA) is under development with design specifications suited for long-term ventricular support. The interface between the heart and compression system is also a "cuff-like" apparatus consisting of several individual parallel compression tubes added serially to cover both ventricular chambers. These tubes form a band around the base of the heart and are bound firmly to the epicardium with the use of surgical adhesive. The

device uses a hydraulic drive system that fills and empties the compression tubes with fluid during the respective half-cycles. These drive systems can be relatively small, run on electricity, and can operate on a rechargeable battery, making this type of device well suited for long-term use.

SURGICAL TECHNIQUE

Application of any of the three compression devices discussed is relatively simple and can be achieved either through a left thoracotomy or median sternotomy. Sizing the of the Anstadt cup or the CardioSupport system is determined essentially by visual inspection; however, estimates based on the size of the cardiac silhouette from chest X-ray, or even measuring the circumference of the heart at the base, has proven helpful. The Anstadt cup and the CardioSupport system are sized by the inner diameter and manufactured in increments of 5 to 10 mm. The inner diameter should be approximately the same diameter as the natural heart ensuring enough space to fit both ventricles adequately. The suction line and drive lines are brought out of the chest sub-sternally or anterolaterally through an intercostal space, making sure to avoid kinking in either line. Before closing the chest, compressions are initiated and, using the CardioSupport system or Heart Booster, synchronized with the native heart beat by setting the onset of device compression pressure approximately equal to the onset of ventricular pressure increase. In the animal laboratory, this is accomplished with the use of a left ventricular pressure transducer; however, a Swan–Ganz catheter pulled into the RV chamber should provide a good approximation and is a feasible option clinically. The amount of compression pressure is slowly ramped upward until cardiac output is maximized. Chest tubes are placed and the chest closed with the device actively assisting the heart.

CLINICAL EXPERIENCE WITH EPICARDIAL COMPRESSION DEVICES

To date, there have been no clinical studies using an epicardial compression device to assist the native ventricular contraction. The CardioSupport system and the Heart Booster are still under development. Therefore, much of the clinical experience with epicardial compression comes from work done on the arrested heart using the Anstadt cup. The largest series of patients studied using this device was a report done on 12 patients.[13] The average age of the group was 48.2 ± 4.2 years with a 5:7 female:male ratio. The average time from witnessed cardiac arrest to device application was 81 ± 9 minutes, although the time from skin incision to device application was reportedly <2 minutes. Systolic and diastolic blood pressures averaged 78 ± 4 and 41 ± 4 mm Hg, respectively, with a mean cardiac output of 3.14 ± 0.18 liters/min obtained for periods ranging from 25 minutes to 18 hours (mean 228 ± 84 minutes). The Anstadt cup was compared with open-chest cardiac massage (OCCM) performed at similar compression rates and was more effective than OCCM at increasing arterial pressures (65% improvement) and cardiac output (190% improvement). Of the 12 patients reported, 4 were successfully defibrillated; however, 2 died of heart failure within the first hour after resuscitation and 2 died from cardiac failure and respiratory insufficiency within 2 days from resuscitation. In 1 patient, the device provided adequate circulatory support to be bridged to emergent cardiopulmonary bypass, but the patient later died from myocardial infarction. There were no complications associated with mechanical CPR; however, complications related to OCCM included a cardiac laceration during peri-cardiotomy and a ventricular rupture. Also of significance, but not included in the study, was a report of successful circulatory support with the Anstadt cup for 56 hours with successful bridging to transplantation and the patient remains alive and well at 1-year follow-up.[14]

POTENTIAL LIMITATIONS

The potential injurious effects of direct mechanical compression on the myocardium is a matter of concern with these or any other similar support mechanism. Myocardial contusions, increased frequency of arrhythmias and myocardial ischemia are the most apparent concerns.

Early experience with the Anstadt cup demonstrated histologic evidence of non-transmural ecchymosis present on the endocardial surface of the RV and pulmonary outflow tract when the device was used to deliver epicardial compression in dogs for periods of between 6 and 24 hours.[15,16] When the duration of compression was increased to 3.5 to 20 months, histologic examination revealed a small scar in the same region. Importantly, these studies were performed with compressions asynchronous with the native heart beat making the likelihood for myocardial injury greater. A small amount of myocardial contusion and edema from direct mechanical compression may be unavoidable, but precautions such as fine adjustment of the synchronization of device contraction with ventricular contraction, avoiding excessive compression, will likely reduce these potential injurious effects. Because mechanical force can cause myocardial depolarization, DCC may increase the frequency of ar-

rhythmias. Anti-arrhythmic therapies, such as lidocaine or amiodarone, and proper electrolyte management will be important to help control the frequency of arrhythmias. Unlike cardiomyoplasty, these devices do not represent contraindications to indwelling pacemakers or ICDs that can be used if required. The potential for myocardial ischemia has also been a concern, especially since compression of the coronary arteries may theoretically impede blood flow. Myocardial perfusion to the LV occurs primarily during diastole when the compression forces are reduced to zero, making it unlikely to cause significant impairment of the native coronary blood flow. Myocardial perfusion to the RV occurs during both diastole and systole, and may be more of a concern. However, the increased arterial pressures gained with DCC should limit any adverse effect on coronary blood flow and may actually improve it. The use of DCC devices on hearts with bypass grafts may be more problematic. Because of their epicardial location, bypass grafts may be more susceptible to the potential adverse effect of DCC on blood flow and the fine vascular anastamosis may not tolerate the additional forces of DCC. However, to date, there has been no investigation to support this.

FUTURE CONSIDERATIONS

Although there has been a considerable amount of work done with epicardial compression, the potential of this mode of therapy for patients with end-stage heart failure has yet to be realized. With the improvement in device design and our understanding of the physiology of epicardial compression, however, such devices may provide an important future mode of ventricular support. Unlike current temporary assist devices, DCC devices are very simple to apply, offer biventricular support, and avoid the need for anti-coagulation. The most immediate application of these devices will be in the setting of acute heart failure where they will act to stabilize the heart for transplantation, provide permanent LV assist device (LVAD) implantation, or even offer recovery of ventricular function. Recent studies have suggested that the heart can undergo "reverse remodeling" from prolonged ventricular unloading by conventional LVAD support.[17] and that this is accompanied by histologic and molecular changes indicative of recovery of ventricular function. Conventional methods of ventricular support provide mechanical unloading; however, they do not assist with the actual muscular shortening as does epicardial compression. Assisting myofibril shortening may offer theoretical advantages for ventricular recovery.

REFERENCES

1. Kawaguchi O, Goto Y, Futaki S, Ohgoshi Y, Yaku H, Suga H. Mechanical enhancement And myocardial oxygen saving by synchronized dynamic left ventricular compression. J Thorac Cardiovasc Surg 1992;103:573–81.
2. Kawaguchi O, Goto Y, Futaki S, Ohgoshi Y, Yaku H, Suga H. The effects of dynamic cardiac compression on ventricular mechanics and energetics. Role of ventricular size and contractility. J Thorac Cardiovasc Surg 1994;107:850–9.
3. Kawaguchi O, Goto Y, Ohgoshi Y, Yaku H, Murase M, Suga H. Dynamic cardiac compression improves contractile efficiency of the heart. J Thorac Cardiovasc Surg 1997;113:923–31.
4. Artrip JH, Wang J, Leventhal AR, Tsitlik JE, Levin HR, Burkhoff D. Hemodynamic effects of direct bi-ventricular compression studied in isovolumic and ejecting isolated canine hearts. Circulation 1999;99:2177–84.
5. Spotnitz HM, Merker C, Malm JR. Applied physiology of the canine rectus abdominus: Force–length curves correlated with functional characteristics of a rectus powered "ventricle."Potential for cardiac assistance. Trans Am Soc Artif Intern Org 1974;20B: 747–55.
6. Aklog L, Murphy MP, Chen SB, Smith WJ, Laurence RG, Appleyard RF, Cohn LH. Right latissimus dorsi cardiomyoplasty improves left ventricular function by increasing peak systolic elastance (E_{max}). Circulation 1994;90(suppl II):112–8.
7. Cho PW, Levin HR, Curtis WE, Tsitlik JE, DiNatale JM, Kass DA, Gardner TJ, Kunel RW, Acker MA. Pressure–volume analysis of changes in cardiac function chronic cardiomyoplasty. Ann Thorac Surg 1993;56:38–45.
8. Artrip JH, Yi GH, Levin HR, Burkhoff D, Wang J. The physiologic and hemodynamic evaluation of non-uniform direct cardiac compression. Circulation, 1999;100 (suppl II):236–43.
9. Corin WJ, George DT, Sink JD, Santamore WP. Dynamic cardiomyoplasty acutely impairs left ventricular diastolic function. J Thorac Cardiovasc Surg 1992;104:1662–71.
10. Salmons S, Jarvis JC. Cardiomyoplasty: The basic issues. Cardiol Chron 1990;4:1–7.
11. Sunagawa K, Maughan L, Burkhoff D, Sagawa K. Left Ventricular interaction with arterial load studied in isolated canine ventricle. Am J Physiol 1983;245:H773–80.
12. Anstadt GL, Blakemore WS, Baue AE. A new instrument for prolonged mechanical massage. Circulation 1965;31(suppl II): 43.
13. Anstadt MP, Bartlett RL, Malone JP, Brown GR, Martin S, Nolan DJ, Oberheu KH, Anstadt GL. Direct mechanical ventricular actuation for cardiac arrest in humans. A clinical feasibility trial. Chest 1991;100:86–92.
14. Lowe JE, Anstadt MP, Van Trigt P, Smith PK, Hendry PJ, Plunkett MD, Anstadt GL. First successful bridge to cardiac transplantation using direct mechanical ventricular actuation. Ann Thorac Surg 1991;52:1237–45.
15. Anstadt MP, Tedder SD, Vander Heide RS, Tedder M, Hilleren DJ, Sostman HD, Reimer KA, Lowe JE. Cardiac pathology following resuscitative circulatory support: Direct mechanical ventricular actuation versus cardiopulmonary bypass. ASAIO J 1992; 38:75–81.
16. Anstadt MP, Anstadt GL, Lowe JE. Direct mechanical ventricular actuation: A review. Resuscitation 1991;21:7–23.
17. Levin HR, Oz MC, Chen JM, Packer M, Rose EA, Burkhoff D. Reversal of chronic ventricular dilation in patients with end-stage cardiomyopathy by prolonged mechanical unloading. Circulation 1995;91:2717–20.