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Hemodynamic Effects of External Cardiac Massage in Trauma Shock

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The effectiveness of closed chest cardiopulmonary resuscitation (CCCPR) in maintaining cardiac output has been well studied in cardiac arrest. Trauma surgeons most often encounter shock secondary to hypovolemia or cardiac tamponade, and the effectiveness of CCCPR in that setting has not been established.

To determine the hemodynamic effects of external massage in profound shock, hypotension was induced in baboons. Pressures obtained with external massage were compared to spontaneous intra-arterial pressures before compression. Although external massage increased systolic pressures in both tamponade and hypovolemia, diastolic pressures were consistently decreased.

We conclude that CCCPR does not augment arterial pressure in the clinical situations associated with decreased LVEDV and is unlikely to provide organ perfusion for trauma victims.

For more than two decades external cardiac massage has been described and accepted as a critical component in the attempted resuscitation of patients in profound shock (16). The augmentation of cardiac output, blood pressure, and vital organ perfusion that results from external chest compression has been investigated and reported in human subjects and experimental animals during spontaneous and chemically induced cardiac arrest or fibrillation. The hemodynamic benefits and clinical usefulness of external chest compression have been extended beyond acute cardiac emergencies and are now described as valuable adjuncts to resuscitation of trauma victims without palpable arterial pulses (3, 8). The recommendation that external chest compression be applied to a trauma victim in profound shock has been made without documentation of the hemodynamic effects or benefits of external cardiac massage in trauma or surgically correctable shock.

To elucidate the hemodynamic effects of external cardiac compression, we studied this intervention in two models of profound surgically correctable or trauma shock.

METHODS

Three large (21.7–35 kg) baboons (*Papio cynocephalus*) were used for the experiment. The animal handling and care complied with the *Principles of Laboratory Animal Care* and the *Guide for the Care and Use of Laboratory Animals.* The animals were initially anesthetized with ketamine hydrochloride (10 mg/kg) and xylazine (3 mg/kg) and subsequently paralyzed with pancuronium (1 mg/kg). The animals were then orally intubated and mechanically ventilated throughout the experiment. Femoral artery and vein catheters were placed for continuous pressure monitoring, arterial gas sampling, and phlebotomy. PaO_2 was kept greater than 85 torr and $PaCO_2$ was maintained between 38 and 43 torr. A left anterolateral thoracotomy was performed and a 16-gauge, 8-inch catheter was placed into the pericardium and brought out through the thoracotomy incision. The thoracotomy was closed and the intrapleural air was evacuated with suction. Intrapericardial catheters were connected to water manometers to allow continuous measuring of the intrapericardial pressure.

Ringer's lactate was then infused into the pericardium at approximately 60 cm of water pressure. The infusion was continued until all central pulses were lost to the examiner's palpation. Intrapericardial and arterial pressures were noted and external cardiac massage was initiated in accordance with ACLS guidelines. External massage was continued for 20–30 seconds. The tamponade was then relieved by gravity drainage. The tamponade model was then repeated in each animal.

Following completion of the tamponade experiment, the animals were given sodium heparin (3,000 units) followed by phlebotomy through the femoral vein catheter. The phlebotomies were continued until central pulses were lost to the examiner's palpation. External massage was again instituted and continued for 20–30

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seconds. In each animal, the spontaneous systolic blood pressure, after cessation of the external massage, was equal to or slightly greater than the point at which pulses were lost (Table I). If pulses were still not palpable, external massage was again initiated for a period of 20–30 seconds. If pulses were again palpable following cessation of external massage, the phlebotomy was continued until pulses were lost and external massage was again initiated.

After completion of this portion of the experiment the anticoagulated shed blood was reinfused, in addition to Ringer's lactate. The animals were then sacrificed with a barbiturate (Euthanol) overdose. When the arterial blood pressure had fallen to less than 10 torr, external massage was again initiated and continued for 20–30 seconds.

RESULTS

Tamponade. Before beginning the pericardial infusion the animals' baseline blood pressures averaged 105/55 (100–110/50–60) mm Hg. Femoral artery pulses were lost consistently between a pressure of 38 and 42 torr in all animals. The intrapericardial pressure required to obliterate femoral pulses ranged from 10 to 20 cm of water. After external cardiac massage was instituted the systolic pressure increased to an average of 57 torr and the diastolic pressure dropped to an average of 8 torr (Fig. 1). External massage elevated the pericardial pressure to 35–50 cm of water.

Hypovolemia. Femoral pulses were initially lost after removal of 30–40% of the estimated blood volume. Femoral pulses were consistently lost at a systolic blood pressure of 40 torr. External massage increased the systolic blood pressure to an average of 64 torr (55–80) and decreased the diastolic pressure to 4 torr (0–8) (Fig. 2). Restoration of the intravascular volume resulted in a rapid return of arterial blood pressures to baseline levels.

Cardiac Arrest. The barbiturate overdose resulted in an immediate drop of blood pressure. External chest compression increased the systolic blood pressure to an average of 108 torr (90–125) and increased the diastolic pressure to 12 torr (8–15) (Fig. 3).

DISCUSSION

In 1960 Kouwenhoven first reported the technique of closed chest cardiac massage and described its effectiveness (10). Following the initial description, external cardiac compression has become a standard component of the resuscitation of patients in profound shock in the prehospital as well as hospital setting. The mechanism of action of closed chest massage, as well as the hemodynamic consequences and time-survival relationships have been thoroughly investigated and are well summarized in multiple recent reviews (5, 9, 12–13, 16).

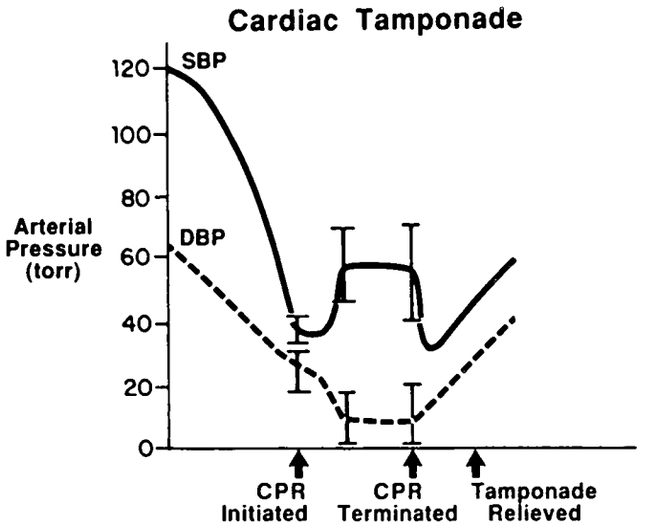
The extensive experimental and clinical data that have been accumulated from research on the effects and benefits of external cardiac compression can be briefly summarized: 1) External cardiac compression generates forward flow by a general increase in intrathoracic pressure, not by direct massage of the heart. 2) The heart functions primarily as a conduit during external compression with forward flow resulting from the presence of venous valves, the greater resistance to collapse found in the arterial system, and the larger capacitance of the venous system. 3) External cardiac massage provides approximately 25% of the baseline cardiac output and generates systolic blood pressures ranging from 30–100 mm Hg and diastolic blood pressure ranging from 10–35 mm Hg. 4) Cerebral and coronary perfusion during external cardiac compression are only about 10% of baseline. 5) This minimal maintenance of vital organ perfusion results in definite and reproducible time-outcome relations with very rare survival after 15 minutes and essentially no survival after 30 minutes (1–2, 6, 9, 12–13, 16–17). These data indicate that external cardiac massage serves only to buy time while correctable problems are addressed.

The extensive research that has detailed the effects and benefits of external cardiac compression has centered exclusively on the medical model of spontaneous or experimentally induced cardiac arrest or fibrillation. The clinical application of external cardiac massage, however, has been extended to the non-medical model and has become a standard part of the resuscitation of trauma patients with cardiovascular collapse (3, 7–8, 14). External massage initiated on trauma victims in the field is often extended into the emergency room, occasionally for protracted periods. Although there are clinical data that imply that external cardiac compression may be of benefit for trauma victims, as it clearly is for victims of cardiogenic shock, several major cardiovascular differences exist in these two patient groups.

Cardiogenic shock secondary to myocardial dysfunc-

TABLE I
Blood pressure mean (and ranges) during various experimental conditions

	Spontaneous (mean torr)		CCCPR (mean torr)		ΔSyst	ΔDiast	ΔMAP
	Systolic	Diastolic	Systolic	Diastolic			
Tamponade	38 (33–41)	25 (18–32)	57 (45–70)	8 (3–20)	+19	−17	−4
Hypovolemia	33 (32–40)	14 (10–20)	64 (55–80)	4 (0–8)	+31	−10	+4
Cardiac arrest	Pre-Arrest		Post-Arrest				
	115 (110–120)	60 (50–70)	108 (90–125)	12 (8–15)			



Hemodynamics

Spontaneous		CCCPR	
Systolic	Diastolic	Systolic	Diastolic
38(33-41)	25(18-32)	57(45-70)	8(3-20)

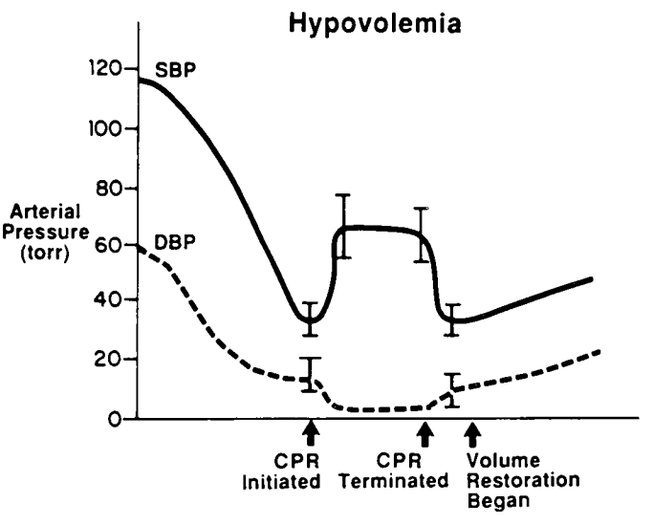
Augmentation with CPR (torr)

ΔSBP	ΔDBP	ΔMAP
+19	-17	-4

FIG. 1. Arterial pressure changes resulting from closed chest massage during cardiac tamponade.

tion or dysrhythmia is associated with normal cardiac and intravascular volumes and, particularly, normal or elevated left ventricular volume. In contrast, the inadequate cardiac output that results from trauma or surgically correctable shock is due to markedly reduced left ventricular volume secondary to inadequate intravascular volume (hypovolemic shock) or markedly restricted ventricular filling (tamponade). The dramatic reduction in blood available to exit the left ventricle during external cardiac massage would logically indicate that cardiac output and vital organ perfusion would also be significantly less than in a normovolemic patient with unobstructed ventricular filling. Although our experimental model did not directly measure cardiac output or vital organ perfusion, there was a significant difference in the intra-arterial pressures generated in the models of trauma shock as compared to that of cardiac arrest. Not only was the average augmentation in the systolic, diastolic, and mean arterial pressures greater in the cardiac model, the absolute increment in pressure was markedly less when external massage was initiated in the presence of reduced left ventricular volumes.

The dramatic rise in intrapericardial pressure that resulted from external massage in the setting of cardiac tamponade raises a particular concern about the impact of external massage in this clinical setting. Our findings are consistent with those of other investigators who have



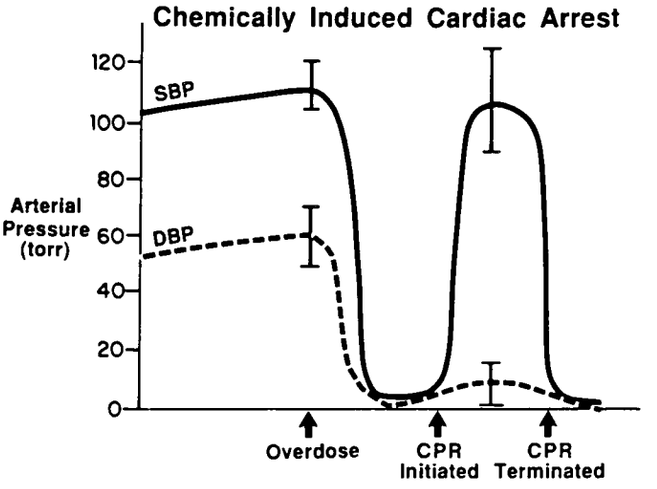
Hemodynamics

Spontaneous		CCCPR	
Systolic	Diastolic	Systolic	Diastolic
33(32-40)	14(10-20)	64(55-80)	4(0-8)

Augmentation with CPR (torr)

ΔSBP	ΔDBP	ΔMAP
+31	-10	+4

FIG. 2. Arterial pressure changes resulting from closed chest massage during hypovolemia.



Hemodynamics

Pre Arrest Spontaneous		Post-Arrest CCCPR	
Systolic	Diastolic	Systolic	Diastolic
115(110-120)	60(50-70)	108(90-125)	12(8-15)

Augmentation with CPR (torr)

ΔSBP	ΔDBP	ΔMAP
+98	+6	+40

FIG. 3. Arterial pressure changes resulting from closed chest massage during cardiac arrest.

demonstrated the relatively slight increases in intrapericardial pressure required to produce hemodynamically significant reductions in cardiac output (11, 15). It is well established that in the setting of acute cardiac tamponade, interventions that increase intrapericardial (intrathoracic) pressure (such as positive pressure ventilation) worsen the hemodynamic compromise produced by tamponade. The additive effect of the tamponade and external chest compression on intrapericardial pressure is likely to further compromise ventricular filling.

The minimal augmentation of systolic blood pressure that resulted from external compression in the models of trauma shock may not be as important as the consistent finding of a decrease in the diastolic pressure. The importance of the diastolic pressure in maintaining coronary perfusion during cardiac arrest was first described by Crile in 1906 and has been firmly established by subsequent investigators (4–5, 9, 13, 16–17). It is probable that the reduction in thoracic aortic volume that occurs in trauma shock would result in a reduction in the intraluminal pressure when the aorta recoils following compression. It is therefore possible that during the early phases of profound shock when the myocardium is still contracting with a functional rhythm, application of external compression could even reduce coronary perfusion.

The effectiveness of external cardiac massage during the resuscitation of a patient with acute cardiac dysfunction is most frequently determined by the presence of central (femoral or carotid) pulses generated by the compression. The presence of central pulses with compression is accepted as an indicator of effective compression. During medical resuscitations it is understood that the pulses felt in the groin may be fluid waves in the venous system. However, since arterial and venous pressures in the iliac system are equal during external cardiac massage, for cardiac patients the distinction of arterial or venous pulses may be unimportant. In contrast, strong femoral venous pulsation from external massage in the trauma victim can be grossly misleading. The normal or elevated central venous volume necessary to generate femoral pulses with external chest compression virtually eliminates hypovolemia as a cause for the profound shock. Femoral vein pulses from thoracic compression may well be present in acute tamponade. However, in this setting, venous pressure waves are clearly *not* a reflection of arterial pressure waves and, if misinterpreted, could result in a disastrous delay in relieving the tamponade.

The purpose of this study was to determine the hemodynamic effects of external cardiac massage in models of profound trauma shock. Our data do not prove that external cardiac massage is of no benefit for trauma victims without palpable pulses. It is apparent, however, that external cardiac massage does not produce the hemodynamic changes and potential benefits for trauma victims that are well described and clinically proven in acute cardiogenic shock. It is therefore imperative that external massage not delay correction of the underlying deficit in trauma shock, inadequate ventricular and intravascular volume.

Based on these data we do not argue that an emergency thoracotomy and internal massage are appropriate for all trauma victims without spontaneous blood pressure. It is crucial, however, to understand that internal massage is at least twice as effective in maintaining cardiac output compared to external compression and has been demonstrated to be far superior to external massage in providing cerebral and coronary profusion (1–4). Trauma victims who might potentially benefit from immediate thoracotomy, thoracic aorta occlusion, and internal massage are unlikely to benefit from protracted external compression. This is particularly true for a victim of a penetrating thoracic wound.

Routine use of closed chest massage in trauma victims should be reevaluated, and those responsible for their management should clearly understand its underlying assumptions and limitations.

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