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Title : VENOUS VALVING DURING STANDARD CARDIOPULMONARY RESUSCITATION (CPR)

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Introduction. Recent investigations of the mechanism of blood flow during standard CPR have led some investigators to resurrect the theory that sternal compressions move blood by diffuse elevation of intrathoracic pressure. They reason that, since right atrial pressure is approximately the same as aortic pressure, cardiac valves cannot be functional and there is no perfusion pressure to produce forward flow of blood. Since blood flow does occur, there must be a driving pressure, though it may be other than that measured between the right atrium and aortic arch. Weisfeldt[1] has suggested functional venous valving. Rosborough[2] has shown angiographically that the valve of the jugular bulb is functional during sternal compressions. To quantify the gradients that exist across the boundaries of the thorax in arteries and veins, we have performed the following study.

Methods. 6 dogs were anesthetized with halothane and fluid-filled catheters were placed in the aortic arch (AA) via the common carotid artery (CC); the superior vena cava (SVC) via the external jugular vein (EJV); the thoracic aorta (TA) via the femoral artery (FA); and the thoracic inferior vena cava (IVC) via the femoral vein (FV). After obtaining control values for each of these vascular pressures, ventricular fibrillation was induced with 100 volts AC transthoracically. Cardiac arrest was allowed to persist for 5 min. Then standard CPR was begun with external cardiac compression (ECC) at the rate of 80/min with 2 intermittent positive pressure ventilations (IPPV) with room air interposed after every fifteenth compression. ECC was performed by a Michigan Instrument "Thumper" and the 2 ventilations after every 15 ECC were performed manually. After 2 min of stabilization, and while ECC/IPPV continued, catheters were withdrawn slowly to clarify the change in pressure in the above vessels as the catheter tips left the thorax. The catheters were calibrated and the location in the vessel where the pressure change occurred was confirmed at autopsy. The anatomy of the arteries and veins leaving the thorax, both at its inlet and at the diaphragm was carefully observed at autopsy.

Results. Our results are summarized in the following table:

VASCULAR PRESSURES (torr)

	Aortic Arch	Common Carotid
Systolic	43.7±12.1	36.8±14.5
Diastolic	10.2±12.5	12.7±13.1*
Mean	26.2±12.4	24.7±12.2
	Thoracic Aorta	Abdominal Aorta
Systolic	43.0±17.0	36.2±14.4
Diastolic	13.0±12.1	12.5±12.3
Mean	26.5±13.2	23.7±12.1

VASCULAR PRESSURES (torr)

	Super. Vena Cava	Ext. Jugular Vein
Systolic	57.0±27.7	15.3±3.4**
Diastolic	8.7± 2.9	7.5±3.9
Mean	29.3±12.6	11.2±2.8***
	Thoracic IVC	Abdominal IVC
Systolic	57.7±21.5	23.7±5.2*
Diastolic	10.5± 9.0	5.0±3.4
Mean	31.5± 9.2	14.0±4.1***

Anatomical valves were found in the jugular bulb in every dog at the site of pressure drop. There were no venous valves at the IVC site of pressure drop. Asterisks (*) refer to p value computed, using the two-tailed t test, comparing the values in each row. (*) $p<0.05$; (**) $p<0.02$; (***) $p<0.01$.

The above data suggest that significant gradients develop during sternal compressions between intrathoracic and extrathoracic veins but that the same gradients are not generated on the arterial side. Functional valving gives rise to a perfusion pressure in the peripheral circulation significantly higher than that measured in the chest. We have confirmed the observation of Rosborough concerning the function of the jugular bulb valve during CPR. However, since the venous structures in the chest are highly compliant, they may function as Starling resistors. Thus, the valving seen at the diaphragm as well as a component of the valving seen at the thoracic inlet, may be due to venous compression (collapse) rather than anatomical valving. However, we also observed gradients within the chest, which are suggestive of direct compression of the great vessels. These gradients within the chest are not compatible with the theory that blood flow is generated by diffuse elevation of intrathoracic pressure.

We do not interpret out data to mean that ITP fluctuations are responsible for blood flow during CPP. The existence of this valving mechanism is a required condition for the ITP theory, however, its demonstration by no means proves ITP to be the dominant driving force for generation of circulation during standard CPR with airway open.

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References.

1. Rudikoff MT, Maughan WL, Effron M, Freund P, and Weisfeldt ML: Mechanisms of blood flow during cardiopulmonary resuscitation. Circ 61:345-352, 1980

2. Rosborough JP: personal communication