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Title : DO INTRATHORACIC PRESSURE FLUCTUATIONS OR HEART COMPRESSIONS MOVE BLOOD DURING EXTERNAL CARDIOPULMONARY RESUSCITATION (CPR)?

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Introduction. Recently, Weisfeldt concluded from his observations in large keel-chested dogs and some patients that sternal compressions in CPR move blood primarily by intrathoracic pressure (ITP) fluctuations, not by direct heart compressions.[1] Since thorax configurations may influence this mechanism, we studied in 5 broad-chested dogs, 10-22 kg) the influence of open pneumothorax on vascular pressures and blood flow during external cardiac compressions (ECC).

Methods. 5 dogs were anesthetized with ketamine and catheters inserted for monitoring blood gases and pressures in the aortic arch, right atrium, abdominal aorta & inferior vena cava. Right common carotid artery blood flow (CCABF) was monitored intermittently by bubble flow meter.[2] Two 12-14 French diameter chest tubes were placed in each pleural cavity and connected to suction. Ventricular fibrillation was induced electrically. After arrest of 2 minutes, standard external CPR was started using a Michigan Instrument's "Thumper" ECC rate was 60/min. and IPPV was with TV 10 ml/kg after every 5th compression. Ten minute periods of CPR with and without open pneumothorax were alternated by opening the chest tubes to the atmosphere and reconnecting them to suction. At the end of each experiment, wide left intercostal thoracotomy was performed, in order to visualize the heart while sternal compressions continued. Before entering the pleural cavity, lung collapse with chest tubes open was confirmed.

Results. At start of CPR, ECC produced MAP in the aortic arch of 36.9 ± 15 torr (mean 80/15 syst/diast). At the same time pressures in the right atrium were 30 ± 14 (83/3). The mean intrathoracic arterial venous pressure gradient was small, but positive at 7 torr. Open pneumothorax did not change these variables significantly.

Extrathoracic pressures were also unaffected by open pneumothorax. ECC produced mean art. pres. in the abdominal aorta of 33 ± 17 torr (67/17) and in the IVC of 13 ± 7 (24/7), resulting in an arterio- venous extrathoracic gradient of 16 torr. These values were not significantly different with open pneumothorax. Pressure gradients between thoracic and abdominal vessels demonstrated functional valving at the diaphragm, with a high thoraco-abdominal venous gradient (21 torr with chest closed and 20 torr with chest open), and a smaller gradient in the aorta of 4 and 5 torr respectively.

CCABF was 22.3 ± 17.1% of control with the chest closed and 22.8 ± 17.1 with the chest open (no difference). There was also no significant differences in arterial or venous blood gas values. The chest tubes, when open, always resulted in lung collapse. Most significant was inspection via a wide thoracotomy during ECC, which revealed direct compression of the heart between sternum and spine.

Augmentation of blood flow during external CPR with simultaneous lung inflations (1,3) does not prove that standard CPR with interposed IPPV moves blood primarily by ITP fluctuations. Coughing during VF produces some circulation[4]; this also is no proof that ITP produces flow during standard CPR. Weisfeldt[1] studied keel-chested dogs; we studied broad-chested dogs, more resembling man. When ECC does not compress the heart directly, venous valving at the thoracic inlet would be necessary for movement of blood Although this has been demonstrated recently by several investigators, it again does not rule out direct cardiac compression during standard CPR; it merely makes pneumatic circulation possible. We conclude that standard CPR with interposed IPPV produces blood flow primarily by direct compression of the heart.

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