Transesophageal Echocardiography during External Chest Compression in Humans

To the Editor—The classical concept that blood flow during cardiopulmonary resuscitation (CPR) results from direct compression of the heart is being replaced by a new theory of a thoracic pumping mechanism.1,2 However, more direct evidence regarding blood flow during CPR is required to establish the validity of this theory.

We performed transesophageal M-mode echocardiography3 to investigate valvular motions in three severely injured patients during conventional external chest compression. With compression the mitral valve, which was nearly closed, immediately opened widely (fig. 1); this was sustained until compression was released. At the beginning of the release phase, both leaflets nearly closed and then opened moderately. These leaflets then moved irregularly, maintaining a near midposition throughout the release phase.

The aortic valve, as expected, was fully open during the compression phase and completely closed during the release phase. The time lag from the start of compression to the opening of the aortic valve was virtually identical to that of the mitral valve.

The left ventricular dimension of the short axis, as measured on the echogram obtained at a position where the tip of the mitral leaflet was visible, showed no distinctive shortening throughout the artificial cardiac cycle. By contrast, the left atrial dimension, at the position where the aortic valve was recorded, decreased significantly during the compression phase.

These findings indicate that the pressure gradient during chest compression is in the order of left atrium > left ventricle > aorta and that the force moving the blood into the aorta is related to the pressure developed in the left atrium, the pulmonary veins, and the lung. Furthermore, the remarkable changes in left atrial dimension suggest that this chamber is the main contributor to the flow generated in the early phase of compression.

The valvular motions recorded by the esophageal echocardiograms provide evidence that supports the thoracic pumping mechanism.

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REFERENCES

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