



The mechanism of blood flow in cardiopulmonary resuscitation—introducing the lung pump

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Abstract

The mechanism of blood flow in cardiopulmonary resuscitation remains controversial. Inconsistencies in the cardiac pump model necessitated the development of the thoracic pump. Neither hypothesis alone can account for blood flow during external massage. Using the postulates that a cyclical pump has three components, an inlet valve, a compression chamber and an outlet valve, we propose the lung pump hypothesis where these components are represented by the pulmonary valve; the lung vasculature/left atrium/left ventricle; and the aortic valve. We explore the mechanism and effect of this hypothesis and match it to previous observations. The implications to cardiopulmonary resuscitation are addressed. © 1997 Elsevier Science Ireland Ltd.

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1. Introduction

The current era of cardiopulmonary resuscitation (CPR) began in 1960 with the report of Kouwenhoven et al. [1]. They stated that “pressure on the sternum compresses the heart between it and the spine, forcing out blood”. With the widespread application of CPR it became evident there were inconsistencies with this cardiac compression model. The role of the heart as the pump was questioned by observations that intrathoracic venous pressure was similar to arterial pressure during external cardiac massage [2,3]. The cardiac compression hypothesis was further challenged by the inability to perform effective CPR in patients with flail chest until the chest was bound [4]; suggesting that an increase in intrathoracic pressure was required to obtain an effective output.

Criley et al. described ‘cough CPR’ after observing patients in ventricular fibrillation generating a cardiac output by continuous coughing [5], a situation where there is no external cardiac compression.

Accumulating evidence necessitated the concept of a ‘thoracic pump model’ of blood flow in CPR. Intrinsic to this theory is a generalised increase in thoracic pressure and the presence of thoracic inlet venous valves [6,7]. More recent observations assessing the mechanism of blood flow during CPR in humans have suggested that the thoracic pump hypothesis alone cannot fully explain the mechanism of generating flow during CPR [8].

We will introduce the concept of the ‘lung pump’ as an explanation of the mechanism to explain blood flow during CPR which fits with known observations. In this model the heart is an essential part of the pump. This model has important implications with regard to improvements in CPR.

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2. The lung pump model

The three essential components of any cyclical pump are;

- an inlet valve,
- a compression chamber,
- an outlet valve

In describing any pump these components must be precisely described. In the normal beating heart these three components are represented by atrioventricular valves, ventricles and ventriculo-arterial valves. These components of the pump are similar during internal heart massage.

In proposing the lung pump model, the inlet valve is represented by the pulmonary valve; the compression chamber by the lung vasculature, left atrium and ventricle; and the outlet valve by the aortic valve. Thus the heart is neither the entire pump nor a passive conduit as described by the cardiac or thoracic pump hypotheses but is an essential part of the lung pump.

2.1. Mechanism of blood flow

During chest compression there is a rise in intrathoracic pressure which is transmitted equally to all intrathoracic structures. There is minimal regurgitation of blood from the pulmonary artery into the right ventricle and vena cavae until the pulmonary valve closes. Thus, blood pressurised within the pulmonary vasculature must exit via the left side of the heart. Upon release of the compressive force the intrathoracic pressure falls below that of extrathoracic vasculature and blood flows into the thorax via the cavae and aorta. Pulmonary vascular volume is restored by blood flow from the right side of the heart through open tricuspid and pulmonary valves. Retrograde aortic flow is checked by closure of the aortic valve. The cycle is repeated upon chest compression.

3. Matching the lung pump model with previous observations during CPR

3.1. Chest compression

Combined observations show that pressure is transmitted approximately equally to all intrathoracic structure with chest compression [6,7,9]. There is a surge of blood into the cavae [7] and the pulmonary valve closes [10,11]. Blood is squeezed from the pulmonary vasculature and left heart to the aorta [7,12]. The aorta does not increase in size during compression [7] contrary to what would be expected with the heart pump model.

3.2. Relaxation

With chest relaxation intrathoracic pressure falls below extrathoracic pressure, resulting in the inflow of blood to the thorax via the aorta and cavae. The pulmonary vascular volume is restored via flow through the right side of the heart [7,12,13]. The aortic valve closes [10,11] due to retrograde aortic flow preventing further inflow into the thoracic cavity by this route.

3.3. Role of the atrio-ventricular valves

Observation of the phasing and competency of mitral and tricuspid valves has previously been used to denote the mechanism of blood flow as due to a cardiac or a thoracic pump. Closure of the mitral valve during compression implies no forward flow from the pulmonary vasculature, therefore flow must arise from left ventricular compression, consistent with the cardiac pump hypothesis. An open mitral valve during the compression phase has been used to support the thoracic pump model.

Initial animal echocardiographic studies confirmed mitral valve closure with external massage supporting the cardiac compression model [11,14]. In contrast to these findings early echocardiographic reports in human subjects reported an open mitral valve during the compression phase [10,13]. Mitral valve closure was not found to be necessary for forward flow during CPR [10]. More recent observations with transesophageal echocardiography in human subjects have shown both closed and open mitral valves during the compression phase of CPR [8,15,16] suggesting that neither the cardiac nor thoracic pump models alone can explain the mechanism of forward blood flow.

An open mitral valve during compression phase is associated with a greater elapsed time from commencing CPR [16] and longer duration of CPR [14]. This may explain the apparently discordant results reported in earlier studies.

Recently Ma et al. [16] observing retrograde pulmonary venous flow and forward mitral flow during the compression phase of CPR proposed a left atrial pump. However, the left atrial pump does not have an inlet valve and as such fails to meet the postulates of a cyclical pump.

Similarly Criley et al. [5] in describing cough induced cardiac compression presented data showing equalisation of left atrial and aortic pressures during the cough 'compression', i.e. no pressure differential across the mitral valve. This finding is consistent with the postulated lung pump but not a cardiac compression model.

With the lung pump hypothesis atrioventricular valves are not essential for pump action as they simply divide different compartments of the compression chamber. The lungs, left atrium and left ventricle are

the compression chamber. The contribution of each part of the compression chamber may vary depending on the blood volume of each component and local distortions related to the technique of compression.

3.4. Role of the ventriculo-arterial valves

Because the pulmonary and aortic valves are essential components of the lung pump severe pulmonary or aortic valve disease will compromise the lung pump function. Patients with Fontan physiology, (systemic venous to pulmonary artery connection with absent pulmonary valve) provide a unique opportunity to examine the merits of the lung pump hypothesis compared to the thoracic and cardiac pump hypotheses.

The lung pump hypothesis would predict that Fontan patients would not generate a satisfactory output during external massage because of the lack of an inlet valve to prevent retrograde blood flow out of the lungs during compression. This is supported by clinical reports of unsuccessful resuscitation of patients with cavopulmonary connections [17].

In contrast, cardiac output from both the thoracic pump (reliant on thoracic inlet venous valves) and the cardiac pump (with an intact systemic ventricle) should be unaffected by the Fontan procedure. Neither theory would predict the described difficulties of CPR with Fontan physiology.

A recent report [18] described a case of successful resuscitation of a patient with Fontan physiology, where chest compression was initially unsuccessful as would be predicted by the lung pump hypothesis. After adding abdominal compression, the systolic blood pressure improved and the patient recovered spontaneous circulation. The abdominal compression would be expected to increase systemic venous return and brace the diaphragm during chest compression improving cardiac output in accordance with the lung pump hypothesis.

4. Implications of the lung pump model applied to techniques of CPR

This model has important implications with regard to efficiency and methods of CPR. Lung pump performance will be compromised by loss of energy across the diaphragm, a flail chest or an open airway. Animal [6] and human studies [19] have shown increased carotid blood flow with abdominal binding, which prevents paradoxical movement of the diaphragm thus maximising the compression on the lungs and heart, improving pump output.

The lung pump model would predict that simultaneous compression of abdominal and thoracic cavities would minimise diaphragmatic movement, thus, improving pump function. The technique of simultaneous

abdominal and thoracic compression awaits evaluation. Predictably the variant of abdominal counterpulsation, interposing abdominal with sternal compression [20] did not improve outcome [21].

Simultaneous ventilation and compression would also be expected to increase the intrathoracic pressure. These techniques have been confirmed to improve carotid blood flow in animals and humans [7,22]. Surprisingly a trial of simultaneous ventilation compression failed to show an improvement in survival [23].

With the 'lung pump' hypothesis sternal movement is not essential; an output could be generated by a pulsatile compressive binder, preferably encompassing thorax and abdomen. Ideally this should be synchronised to the phasing of the ventilator, such that compression and ventilation coincide, maximising pressure on the pulmonary vasculature. Studies in animals and humans have confirmed increased blood flows utilising a pneumatic thoracic vest, abdominal binding and simultaneous ventilation [24,25]. Halperin et al. demonstrated improved survival with vest CPR in dogs [26] and improved short term survival in humans [27].

Active compression decompression CPR as described by Cohen et al. [28,29] showed improved cardiopulmonary circulation when tested in animals and humans. They postulated that active decompression improved venous return to the heart. Initial reports utilising active compression decompression suggested a quicker initial return of spontaneous circulation and 24 h survival [30,31]. This is consistent with the lung pump model as priming of the compression chamber would be augmented because of increased venous return during the decompression phase of the cycle. However, two large trials recently reported no improvement in survival or neurologic outcomes using active compression decompression [32,33], suggesting that further research is required to improve the methodology and outcomes of CPR.

5. Conclusions

The three postulates required for a cyclical pump are; an inlet valve, an outlet valve and a compression chamber. In the performance of CPR, we suggest that these structures are represented by the pulmonary valve as the inlet valve; the pulmonary vasculature and left heart chambers as the compression chamber; and the aortic valve as the outlet valve. We have termed this the 'lung pump', hypothesised how it would behave and have matched this to scientific observations. The implications of this model on methods and performance of CPR have been explored. Describing a pump by its three components provides a logical basis for evaluation of the mechanism of blood flow in cardiopulmonary resuscitation.

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