AS IT GROWS in the cardiac fossa, the heart is surrounded by the soft lungs, although there is the rather rigid diaphragm below and the bony sternum in front. The contrast between upright and lateral decubitus chest x-rays at functional residual capacity (fig. 1) shows how soft the supports of the heart normally are. The pericardium has long been considered the heart's usual and indeed sole restraint. That it only sets the limit to cardiac enlargement, however, is suggested by its "paper bag" type of pressure/volume relationship (fig. 2). My thesis is that the lungs, although always acting as the "good hands" supporting the heart, may be physiologically important as the normal external constraint on its distention.

It is conceivable that when the lung volume changes in breathing, the cardiac fossa keeps its shape exactly in relation to the shape of the heart. Yet the heart must locally displace the relatively soft lung as it beats. In fact, gas flow at the mouth was observed to oscillate with the heartbeat many years ago. Body plethysmographic studies showed this was partly due to unequal O₂ and CO₂ exchange and to the more rapid exit of blood from the thorax than the venous return into the thorax, and also to the direct local mechanical distortion of the lungs. These oscillations agitate the alveolar gas and, to some extent, promote gas exchange like those from high-frequency ventilators, although they are imposed from the other end.

Several things may affect the way the beating heart moves the lungs. Larger stroke volumes, as in aortic insufficiency, increase the cardiogenic oscillations in the gas flow at the mouth. Fluid or scar tissue involving the pericardial sac between the lungs and the heart can cause tamponade. Deep inspiration pulls down the fossa so that it elongates and its radius of curvature increases. Both the pleura and the lung parenchyma become more tense and difficult to deform, tightening the supports around the heart. This is beautifully shown when, after a full inspiration, the inflated lungs support the heart in the lateral decubitus position, in marked contrast to the displacement of the heart that they allow at functional residual capacity (fig. 3).

The appropriateness of the fossa size for the heart should be readily assessed by measuring the intrapleural pressure between the lungs and the heart. If it is too small, the pressure would be positive; if too big, negative. The problem is that intrapleural pressure, far from being constant throughout the chest as was once thought, varies considerably. In the cardiac fossa at any moment, it depends on the size and shape of the undistorted heart relative to the undistorted fossa, and the change of size and shape of the heart and lungs that has taken place from the undistorted state. It is not easy to measure the average global surface pressure exerted on the variably curved heart by the structures surrounding it, including the flat anterior chest wall, retrocurved diaphragm and inwardly curved lung notch, because they are changing size and shape asynchronously. There is no simple way to determine if the rise in cardiac output on inhalation and the fall in cardiac output with mechanical lung inflation can be explained solely by the effect of intrathoracic pressure changes on venous return, or if other factors, including a strange ventricular dysfunction, are involved.

Brookhart and Boyd understood these problems in 1947 when they suggested that the constraints of the cardiac fossa might embarrass the heart when the lungs...
were distended. Culver et al.\textsuperscript{4} recently used a pneumothorax preparation in anesthetized dogs so that they could alter intrathoracic pressure at constant lung volumes (fig. 4). When lung volume was fixed, the effect of positive intrathoracic pressure on cardiac output and filling pressure was predictable from the classic concepts of venous return. However, when lung volume was altered, even with the chest widely opened to atmospheric pressure, the relationship of cardiac output to apparent transmural pressure was altered (fig. 5), suggesting that the ventricular function curves had been shifted toward higher pressures or had been depressed. When the actual juxtacardiac pressure was more closely approximated using a minimally distorting, wafer-shaped sensor inserted between the heart and the lungs, the pressure around the heart became progressively more positive as the lungs were distended in the open chest.

That the apparent depression of ventricular function was artifactual and due to the positive pressure exerted on the heart by the constraints of the fossa rather than the pericardium was confirmed because pericardectomy had little effect compared with selective lobar inflation.\textsuperscript{7} Distention of the lower lobes abutting on the heart altered right and left ventricular function, whereas inflation of the upper lobes alone did not. These lobar distention studies also suggested that the increased pulmonary vascular resistance of lung inflation was not the problem. This was confirmed by snaring the pulmonary artery, since pulmonary arterial pressure had to be increased at least three times higher than that caused by lung distention to cause a comparable decrease in cardiac output. When the cardiac output was varied in the closed-chest dog by a venous return pump and the cardiac fossa pressures were measured by the wafer sensor, the ventricular function curves were no longer changed by lung distention, even to a positive end-expiratory pressure (PEEP) of 15 cm H\textsubscript{2}O\textsuperscript{8} (fig. 6). Further strong evidence for the interaction of the heart with its fossa comes from the observation in these experiments that, with the lungs held distended by PEEP, the juxtacardiac pressure rose as the diastolic heart volume was increased. Thus, the decrease in output of the right and left ventricles with PEEP appeared to be due to both the generalized increase in intrathoracic pressure and the additional increased constraint of the cardiac fossa caused by the lung inflation.

It is particularly apt for this editorial that in 1948

\begin{figure}
\centering
\includegraphics[width=0.5\textwidth]{figure1.png}
\caption{Chest x-ray at functional residual capacity. (top) Upright position. (bottom) Left lateral decubitus position. Note the marked displacement of the heart when lying on the side.}
\end{figure}

\begin{figure}
\centering
\includegraphics[width=0.5\textwidth]{figure2.png}
\caption{Pressure-volume relations of dog's pericardium showing abrupt limitation of distention. Fluid was first injected into the pericardial sac with the heart in situ and then the volume of the heart was measured and added to give the total volume within the sac.\textsuperscript{1}}
\end{figure}
FIGURE 3. Chest x-ray at total lung capacity after full inspiration (usual maneuver for chest x-ray). (top) Upright position. (bottom) Left lateral decubitus position. Compare with figure 1. Note how the distended lung supports the heart.

FIGURE 4. Schematic of dog's chest and method of changing intrathoracic pressure independently of lung volume. The pressure within the pneumothorax is altered via the multiple intrapleural catheters. Lung volume is independently fixed by the airway pressure set by the underwater blowoff valve.4

FIGURE 5. Heart function relationship. The effect of an increase of intrathoracic pressure at constant lung volume was consistent with movement down a normal function curve (solid line). The effect of an increase of lung volume at atmospheric pressure in the open chest (arrow) was to depress or displace the curve to the right (dashed lines).4 RA and LA = right and left atrial.

Dickinson Richards and his colleagues had reached the same basic conclusions.7 Lloyd8 confirmed these speculations about the effect of the fossa in an ingenious way. He substituted a lax, gas-filled bag for the heart in the fossa. This showed that the constraints of the fossa increased at high lung volumes as judged by the pressure caused by stepwise volume additions into the mock heart, and that local distortions (as by distention of only one ventricle) caused more reaction than overall enlargement of the heart. In 1966 Wong, Escobar, Martinez, Rapaport and I9 could not explain why external chest distention by negative pressure decreased heart volume and stroke output in the dog, instead of increasing it. Sixteen years later, we may have the answer—the distended lungs resulted in an apparently decreased left ventricular compliance because of their constraints on the heart.

FIGURE 6. Left and right ventricular function relationships with and without lung distention by 15 cm H2O positive end-expiratory pressure in 10 closed-chest dogs. The transmural pressure was calculated as the difference between intracavitary pressure and pressure sensed by a flat sensor between the lungs and the heart. Stroke volume was altered by a venous return pump. The solid line represents the relationship at low lung volume with end-expiratory airway pressure of 0 cm H2O and the dashed line at high volume with an end-expiratory airway pressure of 15 cm H2O.
What is implied by this interaction of the lungs with the heart? First, the external impedance imposed on cardiac movement by the lungs probably includes some component of dynamic resistance in addition to the static elastance of the lung tissues. This suggests that there is an optimal heart volume and heart rate at which any impairment of cardiac function at high lung volumes is least evident. Second, ventricular interdependence is enhanced by the mechanical limitations imposed by the cardiac fossa. The role of the pericardium in this is probably slight until the limits of its distention are approached. Two recent reviews have stressed the importance of ventricular interdependence in buffering the effects of acute physiologic changes in the output of one ventricle on the other. Left ventricular output does not go up inappropriately when systemic venous return increases with a deep breath (fig. 7). This is probably because its afterload increases and the right ventricular enlargement from the inspiratory increase in systemic venous return causes the diastolic left ventricle to be transiently less accommodating of the inflow from the reservoir of the pulmonary veins. Nor does left ventricular output fall dramatically with coughing or speaking, even if intrathoracic pressures increase markedly. This is presumably because its afterload decreases and a decrease in systemic venous return makes the right ventricle smaller so the left can accommodate more blood from the lung.

There are no evident lung volume changes with positive pressure (Valsalva) and negative pressure (Müller) maneuvers. However, both maneuvers are associated with some shape changes of the chest, gas compression and, most important, with blood volume shifts out of and into the chest. Schrijen and colleagues showed how a positive intrathoracic pressure reduced and a negative pressure increased left ventricular afterload. They thought a transient Valsalva might assist the failing heart. Criley and colleagues suggested that the transient Valsalva maneuver of a cough may actually substitute for the heart pump during ventricular fibrillation. When their patients maintained rhythmic coughing, transient pulses in blood pressure occurred and, since loss of consciousness was thereby delayed, they postulated that these pressure transients were associated with forward flow pulses. This was shown to be true in man by Cary and colleagues, who measured flow noninvasively by photoplethysmograph or external Doppler flowmeter during cough in normal athletes with extreme bradycardia. They coughed when the aortic valve was closed between beats, and the harder the cough, the greater the flow transient in the arm or ear (fig. 8). Thus, the blood pressure spike of a transient Valsalva maneuver is associated with forward flow.

The reason that blood does not flow out of the thorax in the veins as well as the arteries with a cough is that there are valves in all the veins that enter the chest and abdomen (fig. 9). These allow the abdominal cavity to act like a foot pump when compressed and may account for the success of cough resuscitation. Furthermore, although no technique has been shown superior to manual heart squeezing with the chest opened, the idea that the forward flow during conventional cardiopulmonary resuscitation (CPR) is always due to squeezing ("massaging") the heart between the sternum and vertebrae is probably erroneous. For this mechanism to work, the mitral valve should be closed during compression. However, both angiography and echocardiography show that the mitral valve remains open when the sternum is thumped during conventional CPR. Blood would thus go both ways from a directly compressed left ventricle.

The implications of cough resuscitation led Chandra...
and colleagues\textsuperscript{16} to introduce simultaneous chest compression and airway pressurization as the best way to mimic cough and squeeze blood out of the chest. When the airway and chest pressures are released, the compressed heart and vessels rebound and are refilled and primed for the next compression. Flow out of the thorax rises concomitantly with the rapidity and extent of intrathoracic pressure development. Simply pushing on the relaxed chest wall, even when the airway is pressurized, wastes effort by displacing the diaphragm and abdominal contents (fig. 10). During a cough, the powerful abdominal muscles are contracted hard. The pressure transient is fast and maximal. Thus, a major improvement in the CPR technique is to pressurize the abdomen along with the chest, for the functionless chest and abdomen of the unconscious patient is now recognized to act as a single cavity with much the same pressures transmitted throughout. Pressurizing the abdomen with a weight (seated person) or inflatable belt not only renders chest pressure more effective, but compresses the abdominal vessels so flow out of the thorax is directed headward. This CPR technique depends on an apparatus for simultaneously pressurizing the airway with a pressure up to 80 mm Hg via an endotracheal tube and compressing the chest with a vest or thumper at a rate of about 30 compressions/min.

Unfortunately, unlike conventional CPR, a bystander could not do this. It does maintain the cerebral flow necessary for long-term survival of brain function, as shown by Luce and colleagues in studies of dogs with ventricular fibrillation.\textsuperscript{17} However, coronary flow, which is most important for sustaining myocardial function, is frequently woefully inadequate, as are the flows to all other organs inside the thoracic-abdominal cavity.

Coronary flow during CPR when the ventricles are fibrillating results from regurgitation down the aorta to the closed aortic valve during the relaxation phase after the positive pressure transient. Because it depends on the pressure difference between upstream (aortic) and downstream coronary pressure (which is related to right atrial pressure), coronary flow should be improved by increasing aortic pressure and decreasing right atrial pressure. Increasing aortic pressure also depends on higher stroke volumes into the arterial system (higher squeeze pressures acting on larger intrathoracic blood volumes) and an increased recoil of the aorta and arteries. This increased recoil of the arterial system may come from abdominal binding and the use of drugs such as epinephrine. Lowering atrial pressure by imposing negative airway pressures during the relaxation phase has not been very successful, possibly because it decreases lung volume. However, using these principles, one can defibrillate dogs routinely after an hour or more of ventricular fibrillation and CPR.

The role of this modified CPR as a cardiac assist device is exciting, but beyond the limits of this editorial. It must be emphasized that these experimental methods require validation in man. They should not be adopted before they have been fully assessed.
Acknowledgment

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