Intrathoracic Volume Changes in Relation to the Cardiopneumogram

By W. F. Hamilton, and E. A. Lombard

The outflow of blood from the chest during the phases of the cardiac cycle is found to be nearly equaled by a phasic inflow. The net volume change exerts very small pressure changes in the intrathoracic (intrapulmonary) air. This is not due to the distensibility of the chest wall because when allowance is made for this factor and the intrathoracic blood volume change is calculated, it is found to be a very small fraction of the stroke volume.

The air pressure changes in the thorax have long been studied as an indication of cardiac events. The literature has been reviewed recently by Luisada.1 There are two hypotheses to account for the smallness of the pressure changes within the thorax that accompany the heart beat. According to one,2 the cardiac volume remains relatively constant; that is, the auricles fill as the ventricles empty (systole) and the cardiopneumatic pressure changes in the chest are the reflection of very small net changes in intrathoracic blood volume. According to the other,3 because of its flexibility the chest wall is collapsed in the amount of one-third to one-half of the stroke volume by the fall in pressure due to net ejection of blood from the thorax during systole. This fall in pressure is also said3 to be an important factor in venous return. In order to determine the merits of these two hypotheses, records were made of chest circumference changes, caval pressure changes and tracheal pressure changes.

Methods

Nine dogs were used. They were under 10 mg. of morphine and 25 mg. of sodium pentobarbital per kilogram. All records were made during the apnea resulting from over ventilation. Venous pressure was recorded by means of a sensitive strain gauge (Statham #P23B) with a citrate-filled glass cannula introduced through an external jugular into the superior vena cava. The amplification of the circuit to the direct writing galvanometer was set so that 1 mm. water equaled 0.2 mm. deflection.

Tracheal pressure was recorded by air transmission to the same apparatus whose sensitivity was set so that 1 mm. water equaled 2.7 mm. deflection. It is obvious that the intrathoracic (pleural) pressure is not the same as the pulmonary pressure being measured in these experiments, but because the trachea branches to all parts of the thorax, it was felt that it would give an integrated representation of pressure changes occurring within the thorax. This has been verified by introducing a lax balloon into the thorax and recording pressure changes from the balloon.

The trachea was connected by means of a double T tube two centimeters in diameter to (1) an artificial respirator, (2) a strain gauge and (3) a 7 liter bottle filled with air. Each connection could be clamped instantly. A balloon, connected by air transmission to a Dale-Schuster pump, was inserted into the chest to lie between the heart and the spinal column. The chest was closed around the balloon tube without significant pneumothorax. When the pump was running, the intrathoracic balloon changed in volume by 6 cc.

The movements of the chest wall were recorded by the strain gauge recorder set to a similar sensitivity and registering the pressure in a corrugated tube pneumograph surrounding the chest at various levels.

Each strain gauge record was taken with a simultaneous electrocardiogram on a two channel recorder.

Results and Conclusions

Relation of intrathoracic pressure changes to movements of blood in and out of the chest. If the chest were a rigid air-filled box, it would be possible to calculate the net changes of intrathoracic blood volume during the cardiac cycle from the intrathoracic air volume and the pressure changes recorded by an adequate
manometer connected to the trachea. Such a record would take no account of the movement of blood within the thorax (intrathoracic veins to the heart, to lungs, back to heart and to intrathoracic arteries) but would record only the excess or deficit of blood flowing into the thorax through the great veins as compared with the outflow through the arteries (net intrathoracic blood volume change). Excess in venous inflow would, of course, result in an increase in intrathoracic air pressure whereas excess of arterial outflow would result in a fall of pressure. Possible temperature changes due to compression or expansion of air in the lungs have been neglected because of the rapid transfer of heat from the lung air to the lung blood and because this factor, if it did operate, would magnify pressure changes in relation to volume changes.

The walls of the thorax, however, do not form a rigid box. Pressure changes may be expected to cause movements in the chest wall, and movements of the thoracic wall resulting from cardiac impacts may be expected to result in pressure changes other than those resulting from changes in intrathoracic blood volume. Neglecting this last, for the present, it would seem that the distensibility of the walls of the chest could be evaluated from the pressure change produced when an intrathoracic balloon was inflated and deflated by a known amount. Using the apparatus described above, the pump was run at a rate other than that of the heart. The combined pressure changes from the balloon and the heart appeared cyclic when the two were in phase and when they were out of phase (fig. 1). The average of such a cycle would be the contribution of the balloon alone because the balloon produced the larger deflections.

Table 1 gives the data for the last four consecutive experiments. It will be seen (table 1, dog 9, line 5 and fig. 1) that the pressure change produced by the pump was 4.4 mm. water and that the greatest pressure change during systole produced by the heart was 1 mm. water (table 1, line 3 and fig. 2). If the larger pressure change (line 5) was produced by a 6 cc. volume change (line 4), the smaller pressure change (line 3) would be produced by a volume change of 1.6 cc. (line 6). In other words, while the heart was putting out a stroke volume of 24.1 cc. (line 2), the venous return kept up with the arterial outflow except for 1.6 cc. and this deficit was made up late in systole and early in diastole after which outflow and inflow nearly balanced.

The 6 cc. volume change made by the pump would produce a much larger pressure change than 4.4 mm. water if we assumed a reserve air of reasonable size and rigid chest walls. The chest walls, of course, are not rigid, and, as will be seen below, it is impossible by recording the movements of the chest wall to evaluate its expansion or collapse in response to small intrathoracic pressure changes. It is possible, however, to evaluate these elastic responses indirectly by calculating a volume of confined air in which the known balloon volume change makes the same pressure change as it does in the chest. The pressure change is a small fraction of an atmosphere and the volume change, therefore, is a small fraction of the equivalent volume which, by calculation from Boyle's law, is about 13 liters (line 7).

The relation of intrathoracic blood volume change to stroke volume is similar for dogs number 7 and 9, but the other two dogs (number 6 and 8) show much smaller stroke volumes and proportionately larger intrathoracic blood volume changes. This may well be due to the fact that the venous return is less adequate to fill the auricles as their floor descends. Nevertheless, the intrathoracic blood volume change is still a small fraction of the stroke volume.

Evaluation of the volume changes in the intrathoracic blood can be accomplished by the use of Wiedemann's procedure, and the necessity of inserting a balloon into the chest can thus be avoided. One takes a record of the cardiopneumatic pressure changes with the respiratory tract connected directly to the manometer (line 3 and fig. 2) and again with a side tube open to a reservoir of known volume (line 8 and fig. 3). The greater volume of air being compressed when the reservoir is open results in a smaller cardiopneumatic pressure change. Thus, if the pressure change is reduced
Figs. 1-4. Legends on facing page.
by 20 per cent (lines 3 and 8 and figs. 2 and 3) when the known volume is added, this volume is 20 per cent of the air volume equivalent to chest elasticity, and any oscillation in intrathoracic volume (whether a natural change of blood volume or an artificial change of balloon volume) can be roughly quantitated from the pressure change resulting and the air volume equivalent to chest elasticity. A test of the validity of the Wiedemann procedure is to use it in calculating the known volume change produced by the balloon (line 11). From the pressure change produced by inflating the balloon (line 5 and fig. 1) and its diminution when the tracheal tube is opened to the reservoir (line 10 and fig. 4) values are obtained to substitute in the Wiedemann equation. For example:

Wiedemann equation:

\[ \Delta V = \frac{b(\Delta P_1 \times \Delta P_2)}{\Delta P_1 - \Delta P_2} \]

where: \( \Delta V \) = change in volume in cc.
\( b \) = known volume in reservoir.
\( \Delta P_1 \) = pressure change in parts of an atmosphere when the reservoir is not in the system.
\( \Delta P_2 \) = pressure change when the reservoir is in the system.

If \( b \) is 7000 cc, \( \Delta P_1 \) is 4.4 \((10^{-4})\) and \( \Delta P_2 \) is 2.7 \((10^{-5})\), then \( \Delta V \) comes out 5.0 cc.

By comparing the known volume change (line 4) with the calculated (line 11), it is seen that the figures come out to the right order of magnitude in three out of four of the experiments, whereas in dog number 7, the calculation is only half of that expected. A close agreement is hardly to be hoped for because cardiac impacts change the true volume of (and pressure in) the chest wall may well delay its movement in response to minute pressure changes. Using this procedure to calculate the intrathoracic blood volume change produced by the natural heart beat (line 9), we find figures of the same order as those calculated from the pump experiment (line 6) but showing random quantitative variation.

Some of the tracheal pressure curves from the dog tend to rise toward the end of systole. This indicates that more blood is in the chest at the end of systole than at mid systole. Earlier work in man indicates that in some individuals, the intrathoracic pressure comes back completely to the diastolic level well before systole is over. This experiment was repeated on one of these individuals 25 years later (figs. 5 and 6) and the intrathoracic blood volume change calculated by the Wiedemann procedure. The calculations show that the change of tracheal pressure is 2.0 mm. water without the reservoir and 1.6 mm. water with the reservoir, giving a maximum intrathoracic blood volume change of 5.6 cc. with no net change by the end of systole. These results are essentially the same as in the earlier report.

Relation between cardiopneumatic pressure changes and venous pressure changes. It may be maintained that the peripheral venous pressure remains constant during the cardiac cycle. The pressure in the great veins, however, is pulsatile. There is a pressure peak (c wave) at the beginning of systole and a decided reduction in pressure as ventricular ejection progresses. This reduction in central venous pressure during ejection is, in effect, an increase in the pressure drop between the peripheral and central veins and an increase in the force which impels blood into the auricles. As seen in figures 2, 5 and 8, the intrathoracic pressure also falls during early systole, and from uncalibrated records the hasty conclusion was drawn by Blair, Wedd and Hardwicke that the systolic drop in intrathoracic pressure is an
important factor in hastening venous return during systole. When the intrathoracic pressure changes are replotted to the same scale as the caval pressure records, it is seen that
the caval pressure change is about 10 times the actual amount of the intrathoracic pressure change. From the relations seen in figure 7, it seems obvious that intrathoracic pressure changes play no significant role in fixing the tolic increase in the pressure gradient impelling venous blood toward the heart.

Comparison of intrathoracic pressure records and records of movements of the chest wall. If an increase in intrathoracic pressure expanded

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<th>Table 1.—Analysis of Cardiopneumatic Pressure Changes</th>
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<tr>
<td>Dog Number</td>
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Fig. 7. Venous and tracheal pressure changes plotted to the same scale. ECG above. (Dog)

cyclic pattern of venous return. The systolic venous pressure drop is classically the result of the lowering of the auricular floor during ejection, and the records presented here indicate that this intracardiac event is the only important factor in the production of the systolic increase in the pressure gradient impelling venous blood toward the heart.
SUMMARY

The pressure change resulting during apnea from a known volume change within the chest has been evaluated by two independent methods. First, an intrathoracic balloon was inflated rhythmically by a known amount and the consequent pressure changes were measured as transmitted to the trachea. It was found that the volume change in the balloon produced a pressure change similar to that which would occur in a 10.0 to 20.0 liters of confined air because of the distensibility of the chest walls. Second, similar figures were derived by comparing the pressure change produced by balloon inflation before and after an air reservoir of known volume was connected to the trachea. Using these data, it was possible to compute figures of the right order of magnitude for the known balloon volume change.

Both methods gave similar figures for the change in intrathoracic blood volume during the cardiac cycle. These figures were a small fraction of the stroke volume, implying a rapid systolic inflow to the chest which nearly keeps up with the systolic outflow from the chest.

Systolic pressure drop in the vena cava is much greater than that in the thorax. The cyclic pattern of venous return is hardly influenced by thoracic pressure changes.

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