ARTERIAL, CEREBROSPINAL AND VENOUS PRESSURES IN MAN DURING COUGH AND STRAIN


From the Departments of Physiology, Pharmacology and Medicine, University of Georgia
School of Medicine, Augusta

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It has been shown in scattered reports (1, 2, 3, 4) from this laboratory that sudden changes of intrathoracic and abdominal pressure such as occur in coughing and straining produce simultaneous changes in the arterial pressure and in cerebrospinal pressure. It was argued that whereas these pressure changes may strain the arteries of the arms, legs and skin, the more vital arteries of the thoracic and abdominal organs and of the central nervous system are protected against the extra strain made when the intrathoracic pressure goes up during a cough or strain.

The use of the differential manometer (5) has enabled us to show in a simple graphic form the differences in pressure which bear upon the peripheral and vital arteries and to differentiate the effects of direct pressure propagation from the secondary pressure changes resulting from the effects of intrathoracic pressure upon blood flow.

Technical difficulties make it hard to use the intrathoracic pressure itself to work a differential manometer. It is therefore assumed that the gross changes in the intrathoracic pressure are the same as those in the mouth when the lips and nose are closed and the glottis open. A sudden expulsive effort under these circumstances is thought to have the same effect on buccal and intrathoracic pressure and to simulate the effects of a cough upon the hemodynamics. A prolonged expiratory effort against the mouthpiece is proposed as having similar effects to a strain against a closed glottis as in a difficult bowel movement and breathing deeply through the mouthpiece against heavy resistance is thought to illustrate an exaggeration of the effects of breathing upon the blood pressure. On these assumptions the pressure relationships were recorded as follows.

The manometer is of the usual differential type as described elsewhere (5). It is a simple hypodermic manometer (1) whose moving parts are enclosed in an air tight chamber fronted with an optically plane glass plate. The manometer is thus constructed to measure the difference between pressures rather than pressure per se. The buccal pressure is led from a mouthpiece to the front chamber and the arterial pressure is led in the usual way to the manometer itself. The upper record in figures 1, 2 and 3 is from the differential manometer and measures roughly the excess of the arterial pressure over the intrathoracic pressure. That is to say it measures the pressure which strains on the walls of the intrathoracic and intra-abdominal arteries. The middle record is made by a manometer which is connected by a leaden T tube to the same arterial needle

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as serves the differential manometer. It records unmodified the brachial pressure, i.e., the stresses on the arteries of the arms, legs and skin.

**Fig. 1.** Differential pressure record of "cough". The lower tracing is from a mouthpiece into which a forcible expiration was made. The next record is of the arterial pressure while the upper record is a differential record of the middle record minus the lower record. The mouthpiece record shows pressure changes nearly identical with intrathoracic pressure changes and the differential record indicates the stresses which the coronary and similar intrathoracic arteries undergo. The short increases in "intrathoracic" pressure are similar to those in a cough.

**Fig. 2.** Same during a prolonged strain

**Fig. 3.** Same during alternate inspirations and expirations

Figure 1 shows the effect of a short expiratory effort against the mouthpiece. The abrupt increases in the peripheral arterial pressure shown in the middle
record are cancelled out of the upper record by the fact that pressures recorded in the lower record are subtracted by the action of the differential manometer. This illustrates the fact that the vital arteries are protected against such stresses.

In figure 2 pressure is maintained within the thorax and abdomen long enough to hinder venous return. The arterial pressure shows a rise for as long as the strain lasts but the differential pressure (vital arteries) shows a simple fall with a decrease of pulse pressure. This is followed by an increase in both mean pressure and pulse pressure as the blood dammed back in the veins floods into the heart.

In case of figure 3 the subject was required to breath back and forth against heavy resistance. The arterial pressure showed much greater fluctuations than the differential pressure. Each increase in intrathoracic pressure is followed by a decrease in differential arterial pressure and each decrease in intrathoracic pressure, since it aspirates blood into the heart is followed—as soon as blood can get through the lungs—by an increase in the differential arterial pressure and in the pulse pressure.

It should be stressed that conditions obtaining in this experiment are far different from those obtaining during normal respiration. A man breathing naturally shows little or no respiratory changes in the pulse pressure. Particularly if he is recumbent the venous pressure is such that blood can get to the heart unhindered by the respiratory fluctuations in thoracic pressure. If the venous pressure is high enough so that the normal fluctuations in intrathoracic pressure do not affect cardiac inflow, then the respiratory changes in arterial pressure are simple propagations of intrathoracic pressure. Normally this is so but if breathing is against resistance such as accumulated mucus or laryngospasm, or if the venous pressure is abnormally low, the respiration can evoke changes in blood flow which give rise to the phenomena shown in the figure.

When a cough or strain occurs and the pressures in the thoracic and abdominal cavities rise the pressure bears upon the intervertebral foramina. This results in the movement of small amounts of material back into the cerebrospinal canal through the intervertebral foramina. The material which is forced into the canal includes the cerebrospinal fluid under the dural evaginations of the spinal roots, venous blood, loose fatty and areolar tissue as well as the spinal nerves themselves (6). The craniospinal canal is so rigid that only a minute amount of such substance need go into the canal to equalize the pressure to that in the thorax and abdomen during the cough or strain. The result is that the cerebrospinal pressure goes up immediately to a figure that is equal to that in the thorax and abdomen.

It is generally recognized that a rise in jugular pressure is accompanied by a rise in cerebrospinal pressure and that the arterial pressure is also closely related to the cerebrospinal pressure (7). The following observations show, however, that the intrathoracic pressure rise is not propagated to the cerebrospinal canal by either the arterial or venous channels. Figure 4 shows simultaneous records of internal jugular, spinal and intrathoracic pressure. The rise in jugular pressure lags behind the rise in both thoracic and spinal pressure and unless the
effort is prolonged the jugular pressure does not reach as high a figure as the spinal and thoracic pressure.

The rise in arterial pressure which accompanies the cough is also of no consequence in the transmission of the cough pressure to the cerebrospinal cavity. A rise of blood pressure of 40 mm. Hg accompanying an ordinary arterial pulsation has no counterpart in the cerebrospinal pressure while an arterial pressure rise of similar height but accompanying a cough is exactly reproduced in the spinal pressure (see fig. 5).

![Coughing](image1)

**Fig. 4.** Jugular, spinal and intrathoracic pressures during a cough and strain

![Straining](image2)

**Fig. 5.** Arterial pressure and cerebrospinal fluid pressure during two coughs. The lower record is a graphical reconstruction of the difference between the pressures recorded in the upper curves.

The fact that the cerebrospinal pressure has such small pulsations is rather puzzling in itself. Normally the contours show evidences of a systolic rise which is aborted by a fall simultaneous with the descent of the C wave of the venous pulse. The diastolic rise is not so evident in the cerebrospinal pulse as it is in the venous pulse. In short the contours of the cerebrospinal pulse indicate that it is normally a summation of arterial and venous pulsations and since these are essentially reciprocal the actual pressure pulse in the normal cerebrospinal canal is very small.
When the arterial pulsations are exaggerated as they are after the termination of the Valsalva experiment or in dogs after a dose of epinephrine the pressure pulses in the cerebrospinal canal become greater and assume the arterial contour, i.e., rise in systole and go down in diastole (1).

The opposite is true when the venous pressure is increased and the venous pulsations can be transmitted more strongly to the cerebrospinal canal. In cases of congestive failure the pressure pulses of the cerebrospinal canal are exaggerated but they have the venous contour, i.e., they rise in diastole and decline in systole (see fig. 6).

Figure 6 also illustrates another physiological phenomenon that is worth consideration. When the strain begins the intraspinal (intrathoracic) pressure rises and there is an equal rise of the blood pressure. In contrast to the normal there is no falling off of the blood pressure and the pulse pressure because venous pressure is so high that blood fills the heart in spite of the high intrathoracic pressure. When the strain is over the arterial pulsations go back to normal with no compensatory increase in pressure such as is seen in normal individuals when the blood dammed out of the chest surges back in.

It has been shown (8) that reflex adjustment of heart rate to blood pressure changes is absent in dogs with aortic regurgitation. Figure 6, from a case of aortic regurgitation, indicates that the same may be true in man.

When, during the cough, the effective arterial pressure increases in the periphery there is an increase in the arteriolar run off in all beds outside the thorax, abdomen and cerebrospinal canal. This has a striking effect in certain cases with hypodynamic circulation, upon the net aortic blood pressure as is seen in figure 7, A. The aorta and its branches are emptied of blood through the increased peripheral drainage and the net pressure within them goes down very rapidly. During the rise of intrathoracic pressure (measured directly from a pneumothorax) it descends to 10 mm. Hg. Later when this same individual had a paroxysm of coughing, successive coughs reduced the net aortic pressure successively more quickly and more extensively until it reached the value of zero. On occasions the pressure remains below 20 mm. Hg for most of diastole and since the heart gains much of its blood during diastole it probably has its blood supply restricted during the whole of a cardiac cycle. When the effective aortic
EFFECT OF COUGH AND STRAIN ON CIRCULATORY PRESSURES

pressure is zero and the peripheral arterial pressure is maintained by the intrathoracic pressure only, there is no head of pressure to irrigate the coronary or other beds which are entirely within the thoracic, abdominal or cerebrospinal cavity.

This situation does not obtain in individuals whose circulation is normal. Careful measurements have been made in eight normal subjects. The net aortic pressure went down during the cough in nearly all cases but it did not go down more than a few millimeters because the peripheral resistance was not abnormally low.

Fortunately the lowering of the effective aortic pressure is only temporary. The high pressure phase of the cough is followed by the expulsive phase. During this phase the effective aortic pressure will rise even during diastole. The rise can be shown only in cases with hypodynamic circulation and in cases of congestive heart failure. This phenomenon is illustrated in figure 7, B. During

The expulsive stage of the cough, which may begin considerably before the peak of the intrathoracic pressure curve (1) the lungs are being forcibly emptied of air. The same effort that empties them of air also empties them of blood. It has been shown (9, 10, 11, 12) that the capacity for blood is less when the lungs are deflated than when they are distended. If then a forcible expiration can expel blood from the lungs with sufficient pressure and if the pulmonary valve is competent the pulmonary blood will necessarily push open the mitral and aortic valves and enter the aorta. It is difficult to explain the diastolic rise in net aortic pressure in any other way. The rise as we have reconstructed it comes from the fact that the brachial pressure may continue to rise during the expiratory phase of the cough. When not actually rising the arterial pressure often falls less rapidly than the intrathoracic pressure. Pressure energies which are dissipated in moving the lung tissues or the lung air are a part of the thoracic pressure curve and hence are subtracted from the blood pressure in making the construction of net aortic pressure. A disproportionate rise in this factor would
tend to lower rather than raise the net aortic pressure. The rise in net arterial pressure cannot be explained by the fact that transmission of pulsations over arteries is slow because the contours of the surges appear almost the same whether a time interval for this factor is or is not allowed in the construction.

An increased abdominal pressure acting upon the abdominal arteries will probably increase the general blood pressure but not to as great an extent as the combined action of a simultaneous increase of both thoracic and abdominal pressure. If the abdominal pressure exceeds the thoracic pressure during the cough, some of the excess arterial pressure recorded could be due to excess abdominal pressure. Since hydrostatic differences were cancelled by the fact that the subject was recumbent, the important factors which might raise the abdominal pressure above the thoracic pressure are 1, inertia of the viscera; 2, tension on the relaxed diaphragm; 3, contraction of the diaphragm. Rough calculations would indicate that the first factor would involve pressures less than 2 mm. Hg. The second factor might operate to some extent as the lungs are finally emptied of air and since the expulsive stage of the cough is free of inspiratory efforts it is probable that the diaphragm does not contract. Although we have no records of simultaneous abdominal and thoracic pressure, we feel that the abdominal pressure does not rise above the thoracic pressure during the cough enough to account for the excess arterial pressure.

If the expiratory effort drives blood out of the lungs and into the aorta against the systemic pressure a very high total pressure must exist in the pulmonary veins. These veins are without valves and the pressure must be backed up as far as the semilunar valve, distending the capillaries. Both sets of cardiac valves on the left side must be open and the heart be filling the pericardium.

The pressure which can push blood out into the aorta is the pressure distending the pulmonary vein minus the net aortic pressure. Since the first stage of the cough depleted the aortic blood in individuals with a hypodynamic circulation (fig. 7 A) the net aortic pressure is very low and blood is free to move from the lung vessels through the left heart and into the aorta. This it does until the pressure in the aorta equals the sum of the pressures in the thorax and pulmonary vessels, raising the aortic pressure in some cases to a figure that is 30 to 60 mm. Hg above the thoracic pressure. This difference must be the pressure in the pulmonary vein during the expulsive effort. This figure seems high but it is no higher than that seen after epinephrine (9). Its effect on the net aortic pressure is made evident by the fact that the pressure is rising during diastole. When the pressure is falling the aortic valve may be closed and the pulmonary venous pressure may be less than the net aortic pressure. This may also be true during systole when the mitral valve is closed.

The low net aortic pressure which occurs in individuals with hypodynamic circulation favors the movement of blood into the aorta. A high pulmonary venous pressure would be expected to do the same thing. When the net aortic pressure was plotted during the expulsive stages of coughing in a case of congestive heart failure a diastolic surge of pressure similar to that shown in figure 5 B was evident on several occasions. The excess of aortic pressure over intra-
thoracic pressure was 60 to 80 mm. Hg. This is probably the pulmonary venous pressure obtaining at the time.

The pressure which acts upon the walls of the alveolar capillaries is probably much greater than the figure above because the air pressure in the alveoli can hardly be as high as the intrathoracic pressure. During the expiratory phase of a cough the energy which is present in the high intrapleural pressure is available to move the lungs, to accelerate air out of the bronchial tree and to accelerate blood out of the pulmonary vascular tree.

There is no way of measuring what part of the expiratory intrathoracic pressure energy is dissipated in moving blood, what part in moving lung tissue and what part in moving lung air. On the guess that the energy used in moving lung air and tissue is a relatively small fraction of the total it follows that the expiration produces a small rise in air pressure in the alveoli. This pressure change cannot be measured but circumstances point to the idea that during the expiratory phase of the cough the pressure in the pulmonary capillaries which is sufficient to drive blood into the systemic arteries is probably far above the pressure of air in the alveoli. High pressure across the capillary walls must result in filtration of fluid into the alveoli. Paroxysmal coughing therefore may clear froth from the bronchioles and alveoli but it also establishes a vicious cycle which results in the transfer of more fluid into these spaces and the further stimulation of the cough reflex. The fact that coughing raises the pressure in the pulmonary vascular tree in addition to the mechanical movements involved increases the risk of pulmonary hemorrhage during this activity.

If the circulation is normal and the lungs not at all congested, the coughing does not, as has already been said, produce an appreciable surge of blood into the arteries. It is probable, however, that violent coughing does increase the capillary pressure in the lungs and does exaggerate the tendency of fluid to accumulate in the alveoli. The physiological picture thus outlined may, when it is recognized, help to decide in an individual case whether coughing should be encouraged for the purpose of clearing the lungs or whether it should be repressed with sedatives to keep down the transudation of fluid into the alveoli as well as for other well recognized purposes.

SUMMARY

1. Differential pressure records are shown which separate the changes in arterial pressure which are due to simple propagation of intrathoracic pressure from those which are due to changes in blood flow. It is shown that increases due to the first of the above causes strain only the peripheral arteries whereas increases due to changes in blood flow or to changes in peripheral resistance strain also the vital arteries to the brain, spinal cord and viscera.

2. The nature of the cerebrospinal pressure pulsations is discussed.

3. During the preliminary pressure rise of the cough people whose circulation is hypodynamic show arterial pressures which are no higher than simultaneous intrathoracic pressures. During brief intervals there is therefore no effective head of pressure to irrigate the coronary or other vital vascular beds.
4. During the expulsive phase of the cough the arterial pressure may continue to rise while the intrathoracic pressure is going down or the arterial pressure may descend more slowly than the intrathoracic pressure. This signifies that the pressure distending the aorta is rising and, since it often occurs during diastole, it implies that during the expulsive phase of a cough blood is forced from the lungs through the relaxed left heart and into the aorta.

5. The cough may force blood into the aorta in cases with hypodynamic circulation and in cases with congestive heart failure. This may occur in normal individuals but no evidence has been obtained to support the idea that it does.

REFERENCES

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