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CIRCULATORY DYNAMICS OF VENOUS RETURN DURING POSITIVE-NEGATIVE PRESSURE RESPIRATION * † ‡

CHARLES A. HUBAY, M.D., ROBERT C. WALTZ, M.D., GERHARD A. BRECHER, M.D., PH.D., JULIUS PRAGLIN, PH.D., AND ROBERT A. HINGSON, M.D.

INTRODUCTION

As more radical and extensive surgical operations have been developed for the alleviation of crippling diseases of thoracic and abdominal viscera, newer, more effective, and more easily controllable types of anesthesia have become an essential prerequisite.

The development of mechanical respirators by Crafoord (1) in Sweden, Mautz (2) and others in America, and recently by Blease (3) in England, has opened a new horizon promising benefit to patients, surgeons and anesthetists. Controlled mechanical respiration by intermittent positive pressure lung inflation has been used in many centers where its advocates claim considerable merit for its use. Despite this, there has not been widespread acceptance. Watrous, Davis and Anderson (4) summarized the published data in their excellent review article and concluded that controlled (intermittent positive pressure) respiration is a logical and desirable technique.

A controversy has existed for many years concerning the value of a negative phase in the respiratory cycle. The amount of experimental and clinical evidence for or against the use of a negative pressure phase in controlled respiration is small, and to date, no evidence exists that it benefits pulmonary ventilation.

Another question is still being debated whether or not in the closed chest the interposition of negative pressure between periods of posi-

* From the Departments of Surgery, Physiology and Anesthesia, Western Reserve University School of Medicine and University Hospitals of Cleveland.

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tive pressure lung inflation can improve the circulation. Maloney (5) and associates and Motley and Cournand (6) have reported observations in normal patients which indicate little change in blood pressure and cardiac output when intermittent positive pressure and intermittent positive-negative pressure respiration are compared. Maloney (5) reported a lower cardiac output with intermittent positive pressure than with intermittent positive-negative pressure respiration; however, his series of observations was small. In contrast to normal individuals, however, in patients studied by Maloney (5) who had respiratory or circulatory inadequacy, or both, positive pressure respiration resulted in a fall of blood pressure and cardiac output, sometimes of sufficient magnitude to threaten the life of the patient. The danger of increased positive pressure in the airway of patients in shock or in poor condition has been emphasized by Beecher (7). Experimental support for this observation is furnished by Carr and Essex (8) who demonstrated that positive pressure respiration is especially detrimental when hypovolemia is present.

Approaching this problem from another angle, Fenn and his associates (9) in their plethysmographic study of lower limbs noted a significant shift of blood to the extremities when normal individuals were subjected to positive pressure respiration.

Thompson (10), and Birnbaum and Thompson (11) as early as 1942 demonstrated a beneficial effect on the circulation by means of positive-negative pressure respiration in asphyxiated animals, and more recently Sarnoff (12, 13) and his associates emphasized that the circulatory effects of positive pressure breathing were important in the treatment of respiratory failure.

The effect of respiration on one phase of the circulation, namely, venous return, has been the basis for much previous experimental work. Two theories for venous return have been advanced. The classical aspiration theory of Albrecht Von Haller (14) is based on the assumption that veins behave physically like uniformly patent tubes, and that flow in them will vary directly with the existing pressure gradient. Simply stated, if the pressure gradient between the jugular vein and the right atrium is increased by inspiration, flow to the heart would be augmented. The other view, the collapse theory (Holt), (15, 16) maintains that the extrathoracic veins collapse at their entry into the chest, and, that as Duomarco (17) stated, negative pressure in the thorax and its variations are incapable of influencing venous return. Brecher (18) has studied the mechanism of venous flow under different degrees of aspiration, and by means of recording the instantaneous pressure and flow changes, has demonstrated that the two theories do not conflict but actually complement each other.

The failure to recognize that collapse does not occur instantaneously, but is a biphasic process, a function of time *and* pressure gradient, has accounted in the past for the disagreement. Brecher (18) has in-

troduced the concept of the "depleting stage" and the "collapsed stage" in venous return. The "depleting stage" is characterized by a continuous reduction of the peripheral venous reservoir. During this period venous flow to the thoracic veins is increased in direct proportion to the pressure gradient, while resistance remains practically constant. In the second or "collapsed stage," the veins have emptied part of their content and have assumed a reduced filling state during which inflow to the thoracic veins remains constant but the resistance is increased with greater pressure gradients. With expiration, venous return is slightly reduced since the pressure gradient is less and the extrathoracic venous reservoir requires a certain period of time for replenishment.

At the University Hospitals of Cleveland, we have been studying mechanical respirators and more recently have been interested in the addition of a negative phase to the respiratory cycle. The morphologic and ventilatory effects are to be reported elsewhere. This report is concerned with the circulatory effects of an experimental positive-negative pressure respirator.

The maintenance of blood pressure and cardiac output is the result of several factors among which, changes in pulmonary bed resistance and venous return are considered to be of great importance. Measurement of cardiac output alone cannot reveal which of these two factors is responsible for changes in the output.

The purpose of this study is to obtain clear-cut knowledge of the effect of intermittent pressure respiration on venous return. Only direct measurement of blood flow can give reliable information and is preferable to indirect methods of the type employed in cardiac output determinations. Direct flow measurements (19) have already been made with spontaneous respiration and positive-negative respiration, the latter, however, without the use of a clinical mechanical respirator.

Recently, two of our group (20) have developed a method of quantitatively measuring venous return directly by the use of an electrically recording stream-bristle flowmeter. The advantages of this flowmeter over previous types are: (1) compactness; (2) high fidelity response (125 cycles per second); (3) negligible resistance to blood flow, and (4) registration of backflow. Superior vena cava flow was selected for measurement because of previous experience and simplicity of study.

APPARATUS

The cannula illustrated in figure 1 was used to record flow in the superior vena cava. Part Z is inserted and ligated into the vessel about 1 cm. distal to the right atrium. Part Y protects the recording element and contains a side arm for removal of air and for lateral pressure recording. The RCA 5734 mechano-electronic transducer tube is cemented into Part X. The three elements can be dissembled for

cleaning. To avoid corrosion, it is necessary to coat exposed portions of the transducer tube with grease which has a high melting point. Fibrin deposition was avoided by coating the cannula with silicon.

The deviation of the stream-bristle, B, by the blood stream is proportional to flow. The force developed is transmitted to pin P (fig. 1) to which the bristle is soldered. This pin P protrudes through a flexible diaphragm at one end of the vacuum tube and is connected internally to the plate of the vacuum tube (fig. 2). When the bristle position changes, the distance between the plate and cathode of the tube varies. A 6 volt battery heats the cathode indirectly by means of the tube filament to a temperature that causes electron emission from the cathode. A 180 volt battery is used for current to induce

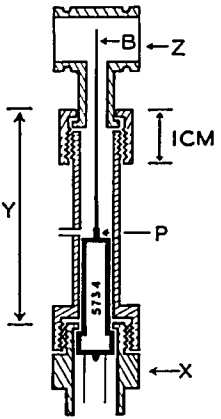

FIG. 1. Diagram of the electrically recording stream-bristle flowmeter, with RCA 5734 mechano-electronic transducer tube (see text). (Reproduced by permission of Proc. Soc. Exper. Biol. and Med.)

electrons to flow through the 500,000 ohm resistor and the tube. This current is constant as long as the 5734 plate does not move with respect to the cathode. The electrons have to traverse either a longer or a shorter path through the tube's vacuum, depending upon the distance between the plate and cathode. Since the number of electrons flowing depends on the distance between the plate and cathode, the amount of current flowing around the circuit is then proportional to the position of the bristle. Also, by Ohm's law, the voltage developed across the 500,000 ohm resistor is proportional to the current, and a voltage will be developed at the cathode in proportion to the bristle movement.

This voltage is not of great enough amplitude to drive a recording

device and therefore must be amplified by a direct-coupled amplifier. It is necessary that the amplifier be direct coupled in order that very slow changes in flow will be accurately reproduced. Of the several commercial amplifiers available, a Keithly Universal Chopper amplifier was selected because of its very great stability as regards the base line drift. Connection to the amplifier cannot be made directly from the cathode of the transducer tube owing to the fact that the 180 volts are divided between the 5734 tube and the 500,000 ohm resistor. This places the cathode of the 5734 tube about — 35 volts with respect to ground. The amplifier, however, requires a signal at ground potential, and therefore it is necessary to place a battery (C) in series with the signal to raise it to ground potential. Balancing of the instrument so that zero signal produces zero output is accomplished by

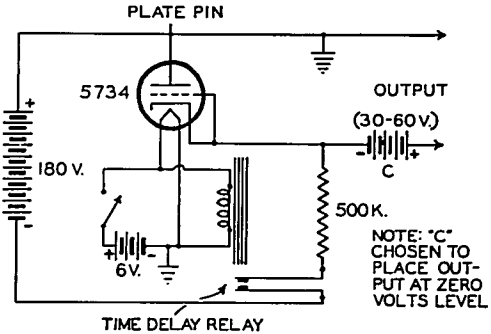

FIG. 2. Circuit diagram of the RCA 5734 mechano-electronic transducer tube for its connection to a direct current amplifier.

clamping the vessel briefly and setting the balance control of the amplifier.

The frequency response of the instrument is uniform over the range of zero to at least 125 cycles per second. Thus, even the quickest phases of flow are accurately reproduced. The zero drift or base line instability of the instrument is negligible over the time of an experimental observation even though the 5734 tube is rather sensitive to changes of temperature. This temperature sensitivity is minimized when the flowmeter is enclosd by living tissues. Furthermore, the column of static blood in section Y of the cannula insulates the tube from the cooling effects of flowing blood.

Mean flow was recorded by electrically decreasing the speed of response of the recording system until changes occurring at a rate faster than about 1 cycle per second were not individually recorded

but were averaged by the system. This was accomplished by shunting a coupling network in the Keithly amplifier with a large capacitor.

It should be noted that the plate of the 5734 tube is at ground potential. This is necessary because the plate is not insulated from the cannula. The negative terminal of the battery and the cathode of the tube are insulated and can be placed at a high voltage with respect to the animal. The animal as well as the positive terminal of the battery should be grounded to a common point to avoid 60 cycle pick-up and electrical shock both to the animal and to the experimenters.

PROCEDURES

Eight experiments were carried out on heparinized dogs whose weight ranged from 15 to 22 kg. The animals were anesthetized with morphine sulfate, 1.5 mg. per kilogram, subcutaneously and phenobarbital, 15 mg. per kilogram, intravenously, and were fixed in the supine position.

A closed airway was created by passing a number 40 Magill endotracheal catheter and distending the distal balloon. The endotracheal tube was then attached by tubing to a standard anesthesia machine with a carbon dioxide absorber in the expiratory limb of a closed circuit. An experimental positive-negative pressure respirator designed and constructed for our use § was then attached to the anesthesia machine. The anesthesia bag was enclosed in a plastic case similar to that used in the Mautz (2) respirator. Pressure about the bag could be changed rapidly from positive to negative or maintained at atmospheric.

The right side of the chest was entered between the third and fourth ribs and the stream-bristle flowmeter inserted into the superior vena cava. By Gregg optical manometers, pressure recordings were obtained simultaneously from the aorta and the superior vena cava or the right atrium. Endotracheal pressures were registered by air transmission through a Frank capsule. Pressures and flow were optically recorded. The beams of the manometers were calibrated at the end of each record at zero pressure, set at the center of the right atrium. Zero flow was established at the beginning and end of each record by clamping the superior cava distal to the stream-bristle flowmeter. At the end of each experiment, the manometers were calibrated over a wide range of pressures and the flowmeter was calibrated at different rates of steady flow. After several records were taken with the chest open, the thorax was tightly closed and normal intrathoracic pressures were re-established.

In three of the experiments a specially designed differential flowmeter was used instead of the stream-bristle flowmeter in order to measure flow under the best physiological conditions. This previously

§ Constructed by the Ohio Chemical Surgical Equipment Company, Madison, Wisconsin.

described flowmeter (21) is introduced like a catheter into the superior vena cava through the external jugular vein without the necessity of opening the thorax. In one of the experiments the bristle flowmeter and a differential flowmeter developed by Mixter (22) were checked against each other by inserting both meters in tandem into the superior cava.

RESULTS

The changes of venous return measured with the two types of differential flowmeters as well as with the stream-bristle flowmeter were consistently the same in all experiments. There was no difference in the results whether the chest was left intact as with the use of the catheter flowmeter, or when the thorax had been secondarily closed, with the use of stream-bristle and the Mixter differential flowmeters. For this reason, the results are presented in a series of comparable original records of one experiment, which typifies the flow changes observed in the others. In this particular experiment superior vena caval flow was recorded with the stream-bristle flowmeter.

Effect of Negative Endotracheal Pressure in the Closed Chest

The record shown in figure 3 demonstrates the increase of venous return in the closed chest when the atmospheric endotracheal pressure is suddenly lowered from 0 to −130 mm. of water. It is noted that superior vena caval flow shows marked accelerations and decelerations within each cardiac cycle, the details of which, however, are not the subject of this study. The flow pattern during the first three cardiac cycles is approximately the same. This is the period during which endotracheal pressure is zero. This period corresponds in its pressure relations to the expiratory pause between two normal spontaneous inspirations. Although airway pressure is atmospheric, intrathoracic pressure is negative, which is borne out by the fact that the pressure in the superior vena cava is below zero. Flow in the vena cava is quantitated by measuring the area under the flow tracing. Inflow into the right atrium from the superior vena cava amounted to 7 cc. during each of the three cardiac cycles. Immediately following the reduction of the endotracheal pressure, as indicated by the arrow, right atrial inflow increased rapidly.

The phasic recording of instantaneous flow changes permits us to state that this flow augmentation occurred within the first cardiac cycle after the endotracheal pressure was lowered. This indicates that augmentation of the superior vena caval flow cannot be the result of a sudden reduction of the resistance to flow in the pulmonary artery bed when the lungs are rapidly deflated below their normal state of expansion. Decrease of resistance of the pulmonary artery could have had its earliest effect in the second heart cycle after the lowering of endotracheal pressure. Therefore, only the decrease of intrathoracic

pressure, or in other words, greater thoracic aspiration, can be responsible for the sudden augmentation of vena caval flow. The increase of thoracic aspiration is indicated by the greater negativity of intrathoracic pressure as seen by the fall of superior vena caval pressure with the lowering of the airway pressure. Right atrial inflow from the superior vena cava increased from 7 cc. in the third cardiac cycle to 10 cc. in the fourth and 12 cc. in the fifth cycle.

As previously shown (23), thoracic aspiration, as the responsible mechanism for the increase of venous return with respiration, accom-

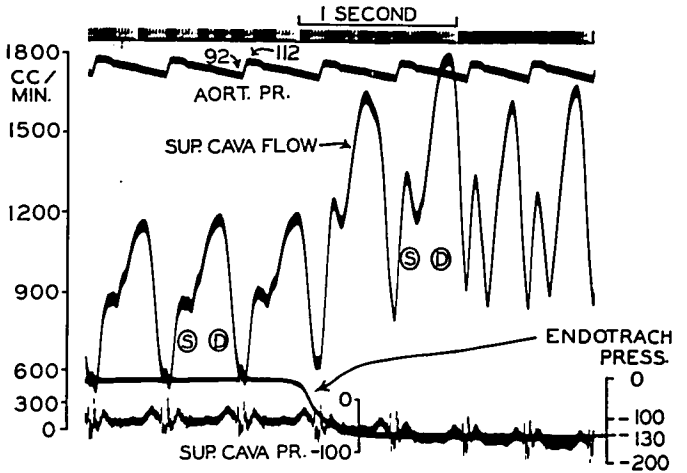

FIG. 3. Segment of an original optical record showing the effect of negative entotracheal pressure on phasic blood flow in the superior vena cava in the closed chest. Tracings from top to bottom: time, aortic blood pressure in millimeters of mercury; superior vena cava flow in cubic centimeters per minute; endotracheal pressure in millimeters of water (atmospheric pressure is at 0); superior vena caval pressure in millimeters of water. "S" is flow during ventricular systole; "D" is flow during ventricular diastole.

plishes the flow augmentation by two successive stages. The first, the "depleting stage," during which the extrathoracic veins are depleted of their blood into the thoracic veins is characterized by a greatly increased flow into the central veins. The depleting stage lasts in the record of figure 3 for about two cardiac cycles(fourth and fifth). The second or "collapsed stage" is characterized by the partial collapse of the extrathoracic veins which increases resistance to blood flow in spite of a more favorable pressure-gradient from the peripheral veins to the right atrium. Flow during this period decreased slightly, but

was still substantially greater than flow without the additional thoracic aspiration by lowering endotracheal pressure. Flow during the collapsed stage is illustrated by the flow tracings during the sixth and seventh cardiac cycles in the record of figure 3.

Four primary conclusions may be drawn from this experiment. In the first place, flow increases significantly when endotracheal pressure is lowered below atmospheric pressure. Second, thoracic aspiration and not reduction of pulmonary bed resistance is the immediate cause of augmentation of the flow. Third, the existence of a depleting stage followed by a collapsed stage is identical with that previously described (18) to occur with normal and forced spontaneous inspiration. Finally, the fact that increased thoracic aspiration is the responsible factor for the augmentation of flow makes the application of negative endotracheal pressure with a clinical respirator physiologically comparable to the effect of normal spontaneous inspiration as far as venous return is concerned. Anatomically, however, the thoracic cage is increased in diameter with spontaneous inspiration but decreased with artificial application of negative pressure to the trachea with the respirator.

Effect of Positive Pressure Lung Inflation in the Closed Chest

It is necessary to record instantaneous flow changes within the course of a single cardiac cycle for the accurate analysis of the time events at the moment of endotracheal pressure change, but it is laborious to compute mean flow from these phasic flow curves. In order to determine mean flow values for the duration of entire respiratory cycles the flow tracing of the bristle-flowmeter was electrically averaged. The high mechanical frequency response of the flowmeter was not altered by this procedure. The flow tracings in the following figures, therefore, are "mean flow" in which the flow fluctuations during the individual cardiac cycle are less noticeable.

The record presented in figure 4 demonstrates not only the beneficial effect of negative endotracheal pressure on venous return but also the detrimental effect of positive pressure lung inflation in the closed chest. In an animal with normal blood volume, the record shows that mean flow was 1,064 cc. per minute during atmospheric endotracheal pressure. Flow increased to 1,160 cc. per minute with negative endotracheal pressure of -120 mm. of water, and was reduced to 984 cc. per minute for the duration of the positive pressure phase. Again, the pressure in the right atrium reflects the changes in intrathoracic pressure that were brought about by the pressure in the airway. Atrial pressure decreased with negative endotracheal pressure and increased with the application of positive pressure. As has previously been shown (21), the rise of right atrial pressure during the positive pressure inflation of the lung reduces the pressure-gradient

from peripheral veins to the heart and diminishes inflow into the right side of the heart. As a result, blood accumulates in the peripheral veins, thereby causing a compensatory rise in the pressure-gradient. Thus, vena caval flow decreased quickly at the beginning of lung inflation and remained constant even though the atrial pressure continued to rise, as seen at the extreme right of the record.

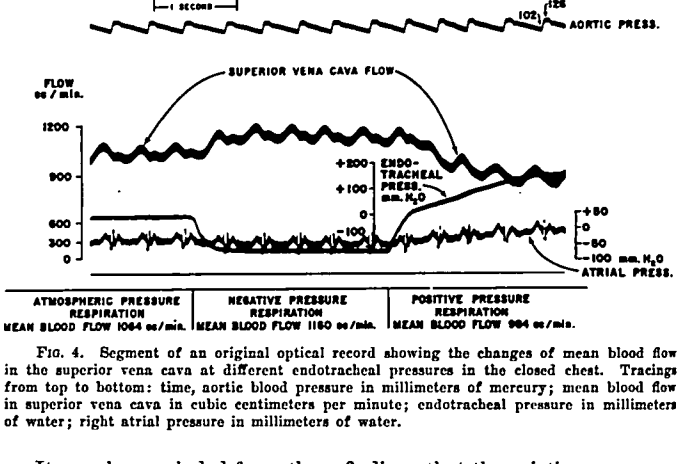

FIG. 4. Segment of an original optical record showing the changes of mean blood flow in the superior vena cava at different endotracheal pressures in the closed chest. Tracings from top to bottom: time, aortic blood pressure in millimeters of mercury; mean blood flow in superior vena cava in cubic centimeters per minute; endotracheal pressure in millimeters of water; right atrial pressure in millimeters of water.

It may be concluded from these findings that the existing pressure-gradients from the peripheral veins to the right atrium are an important factor in determining venous return at different pressures in the airway.

Effect of Positive-Negative Pressure Respiration in the Closed Chest

Even though the records shown in figures 3 and 4 demonstrate an immediate flow increase with the use of negative endotracheal pressure, it cannot be concluded that alternation of positive-negative pressure respiration necessarily must augment venous return over that existing during positive-atmospheric pressure respiration when it is measured over a longer period of time. It might be assumed that more blood is dammed up by a positive pressure lung inflation when alternated with atmospheric than by a lung inflation when a negative phase has preceded (during which the peripheral venous reservoir has been depleted more thoroughly). If this is the case, any flow increase during a period of negative pressure might be canceled by a correspond-

ingly greater reduction of flow during the following positive phase. It was necessary, therefore, to determine whether or not, over longer periods of time, positive-negative pressure respiration actually results in a *net* increase of flow over that obtained with positive-atmospheric pressure respiration.

Figure 5 shows a segment of a record demonstrating the *net* increase of venous return by the use of intermittent positive-negative pressure respiration over that measured during positive-atmospheric pressure respiration. Mean superior vena caval flow was measured over two complete positive-atmospheric respiratory cycles and compared with two positive-negative cycles. For reasons of reproduction the segment of the record shown in figure 5 is limited to the last portion

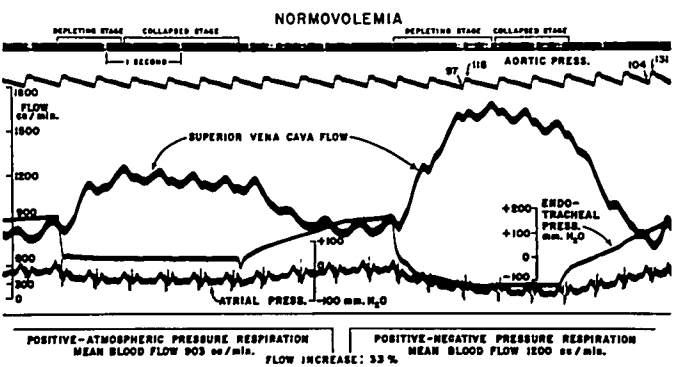

FIG. 5. Segment of an original optical record demonstrating the augmentation of venous return with positive-negative pressure respiration in the closed chest with normal blood volume. (Tracings as in fig. 4.)

of the positive-atmospheric phase and the first portion of the positive-negative cycle.

Mean superior caval flow during the two positive-atmospheric cycles was 903 cc. per minute. The corresponding measurement during the two positive-negative cycles was 1200 cc. per minute. This is a *net* flow increase of 33 per cent.

From left to right the record of figure 5 shows the following. During the positive phase flow was greatly reduced, but increased rapidly upon sudden release of the lungs to atmospheric pressure. This was owing to the backlog of blood that rushed from the peripheral veins to the heart. Right atrial pressure was below zero indicating the existence of normal thoracic aspiration when the airway pressure was atmospheric. The flow increase lasted for less than a second (depleting stage) and leveled off (collapsed stage). During the next

positive pressure phase flow was again reduced in the same manner as already described. Then followed the negative phase, during which flow was greatly augmented. It is obvious that this augmentation of flow is the result of the backed up blood *plus* the depleting of the extrathoracic venous reservoir by the greater thoracic aspiration. With the application of negative pressure, the depleting stage lasted longer (about one and a half seconds); it was followed by a leveling off of the still greatly augmented flow (collapsed stage). The next positive phase reduced flow again, as shown on the right side of the record. This flow reduction was slightly greater than that during the positive phases which alternated with atmospheric pressure. The greater flow reduction, however, did not cancel the flow increase which occurred during the preceding negative phase.

On the right side of figure 5 it is noted that the aortic pressure rose from 116 to 131 mm. systolic and from 97 to 104 mm. diastolic. This significant rise of arterial pressure evidently is the result of the increased venous return, and was delayed by about three heart beats since the blood had to circulate through the pulmonary bed. This direct relationship between venous return and arterial pressure emphasizes the importance of venous return for the control and maintenance of cardiac output.

It may be concluded from these findings that intermittent positive-negative pressure respiration with a clinical respirator in the closed chest increases venous return significantly over that measured during positive-atmospheric pressure respiration.

Effect of Positive-Negative Pressure Respiration in Hypovolemia

The intention in this study was to alter the circulatory conditions in various ways in order to investigate the influence of positive-negative pressure respiration on the impaired circulation. For this purpose a hypovolemic state was created by bleeding the animals in a stepwise fashion until the arterial blood pressure was reduced to about 50 mm. of mercury.

Figure 6 depicts a segment of a record illustrating the deleterious effect of positive-atmospheric pressure respiration and the beneficial effect of positive-negative pressure respiration on venous return in the closed chest during hypovolemia. The segment of this record corresponds to that shown in figure 5. On the left side, it is seen that venous return was very small during positive-atmospheric pressure respiration but became greatly augmented by the use of negative pressure as shown on the right side of the figure. Although mean blood flow during positive-atmospheric respiraton amounted to only 133 cc. per minute when measured over two complete respiratory cycles, the respective mean flow during positive-negative respiration increased to 266 cc. per minute. This is a *net* flow increase of 100 per cent.

It should be recalled that the addition of negative pressure in the closed chest of a normovolemic dog resulted in a net flow increase of

only 33 per cent. The absolute flow increase by the use of the negative pressure phase in hypovolemia (133 cc.) was not so great as in normovolemia (297 cc.) since the content of the peripheral reservoirs was greatly reduced. However, flow increase was relatively greater in hypovolemia since the initial flow rate was very low.

The great relative flow increase as expressed in percentage emphasizes three facts derived from the data of figure 6. First, positive pressure results in almost complete cessation of flow. Second, atmospheric pressure does not create a favorable enough pressure-gradient between the peripheral veins and the right atrium to deplete adequately the backed up blood into the thoracic veins. Third, increase of the pressure-gradient from the peripheral veins to the heart not only empties substantially the small peripheral venous reservoirs during

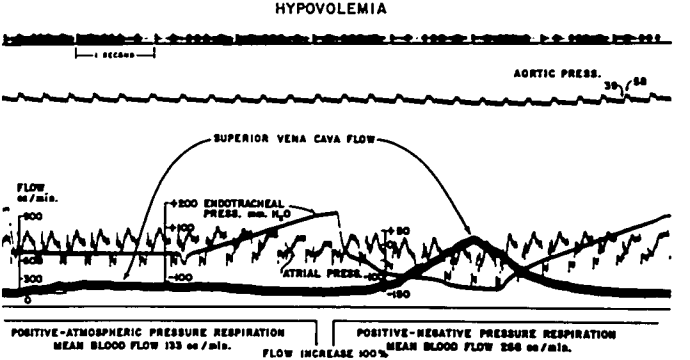

FIG. 6. Segment of an original optical record illustrating the relatively greater net increase of venous return in hypovolemia as compared to normovolemia shown in figure 5 (closed chest). (Tracings as in fig. 4.)

the depleting stage but also maintains a greatly augmented venous return in spite of the partial collapse of the peripheral veins during the "collapsed stage."

As already demonstrated in the case of normovolemia, arterial pressure rose also in hypovolemia as a result of the increase in venous return. In fact, the increase of arterial pressure in hypovolemia was even more striking than in experiments with normal blood volume.

Effect of Positive-Negative Pressure Respiration on Venous Return in the Open Chest

From the results of the experiments presented in figures 3 to 6, it is apparent that increased thoracic aspiration is the mechanism responsible for the augmentation of venous return during positive-nega-

tive pressure respiration. If this is true, it should be expected that after abolition of thoracic aspiration, such as in the open chest, venous return could not be improved by the use of positive-negative pressure respiration.

This is demonstrated by a segment of a record in figure 7. This segment corresponds, again, to those shown in figures 5 and 6. The very end of a positive pressure lung inflation and the sudden release of endotracheal pressure to an atmospheric value is seen at the extreme left of the record. Superior vena caval flow, which was reduced during the lung inflation, increased immediately upon release of the positive pressure in the airway. The next positive pressure lung inflation, seen in the middle of the record, shows the same reduction of flow. This lung inflation was followed by the application of negative pressure to the airway (right side of record). It did not result in a sub-

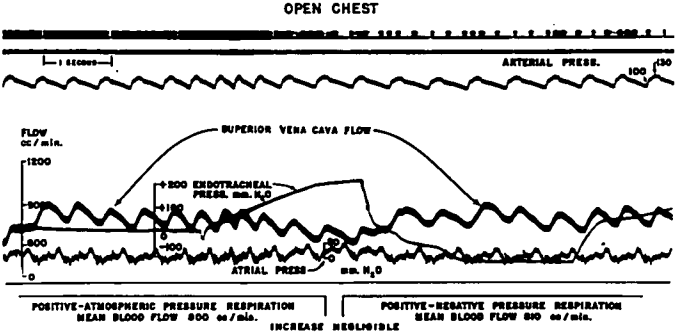

FIG. 7. Segment of an original optical record showing the negligible increase of venous return when negative endotracheal pressure is applied between positive pressure lung inflations in the open chest. (Tracings as in fig. 4.)

stantial increase of superior vena caval flow over that observed during atmospheric endotracheal pressure. By measuring mean flow over a longer period of time, it was found that during two complete positive-atmospheric respiratory cycles, superior vena caval flow amounted to 800 cc. per minute, while during the respective positive-negative respiratory cycles it was 810 cc. per minute. In short, in the open chest the net flow increase by the application of positive-negative pressure respiration is almost negligible.

It should be mentioned that in all experiments there was consistently a very small net flow increase with the use of negative airway pressure, but an analysis of the factors responsible for this small flow improvement is not the purpose of the present investigation. It suffices to say that, contrary to the findings in the closed chest, no sig-

nificant benefit to the circulation can be expected by the application of negative endotracheal pressure in open thoracotomy.

DISCUSSION

The continuous, simultaneous recording of superior vena caval blood flow, endotracheal, aortic and right atrial pressures has made possible a study of the effect of positive and negative pressure respiration on venous return in both the closed and open chest. It may be thought that measurement of only the superior vena caval flow might not be indicative of total venous return, since the increase of superior vena caval flow could be counteracted by a corresponding decrease of flow in the inferior vena cava. Studies (19) with two flowmeters under normal physiological conditions in dogs with closed chests, however, revealed that flow is increased in both cavae simultaneously with each spontaneous inspiration.

Brecher and Mixter (23) in their study of the effect of respiratory movements on superior vena caval flow concluded that the immediate changes in venous return resulting from lung inflation by means of intermittent positive pressure are determined by the pressure-gradient between the peripheral veins and the right atrium, uninfluenced by left cardiac output. The reduction of arterial pressure during positive pressure respiration is caused primarily by a decrease in venous return alone. Baxter and Pearce (24) in studies of pulmonary arterial blood flow observed that lung inflation by positive pressure resulted in an immediate, marked decrease in blood flow. Negative pressure deflation of the lungs, however, caused a decided increase in beat to beat output of the right side of the heart. From these observations and our own measurements using an experimental positive-negative pressure respirator, it would appear that, in the closed chest, intermittent positive pressure respirations have a deleterious effect on venous return and that negative endotracheal pressure deflation of the lung augments venous return.

Wide clinical experience with mechanical respirators has demonstrated the safety and desirability of such an apparatus in patients with relatively normal blood volumes. The detrimental effect of positive pressure in the airway is overcome by a rise in peripheral venous pressure, increased autonomic activity and mobilization of blood reservoirs leading to a more favorable pressure-gradient between the peripheral veins and the right atrium with an adequate blood flow into the central veins. From a clinical standpoint, therefore, intermittent positive pressure respiration has little effect on the circulatory dynamics under normal conditions, although we have shown that there is a net increase of venous return with positive-negative pressure respiration over that with intermittent positive-atmospheric pressure respiration.

Under conditions of blood loss and hypotension, however, the de-

pletion of the peripheral venous reservoir does not allow this compensatory rise in pressure-gradient favoring inflow and the diminished venous return is further lessened with a resultant fall in cardiac output and arterial blood pressure. Our experiments have shown that in the closed chest when negative pressure is added to the respiratory cycle in circulatory inadequacy there is a beneficial effect on venous return. The greater increase in net flow in hypovolemia makes possible a greater turnover of available blood and substantially empties further the already diminished peripheral venous reservoirs toward the heart.

It should be emphasized that the observed changes in venous return in our experiments were the result of relatively small variations in endotracheal pressure. The use of greater and more prolonged positive lung inflations has an even more deleterious effect. Wilhelm (25) pointed out that a distended anesthesia bag creates significant intrapulmonary pressure and therefore sustained positive pressure developed in this way should be avoided in order not to interfere with the circulation. With circulatory inadequacy these effects are magnified. Even in the open chest there is interference with venous return with positive pressure in the airway and Papper (26) has pointed out that increased pulmonary pressure can produce hypotension. Therefore, the clinical practice of *prolonged* positive pressure to aerate atelectatic lung areas should be avoided.

SUMMARY

An experimental positive-negative pressure respirator was evaluated for its effect on blood flow in the superior vena cava in experimental animals. Blood flow in the superior vena cava was directly measured with a low resistance electrically recording stream-bristle flowmeter.

In the closed chest, intermittent positive-negative pressure respiration increases venous return significantly over that measured during positive-atmospheric pressure respiration.

The use of a negative pressure phase, between positive pressure lung inflations in the closed chest, is physiological since the mechanisms of increased venous return correspond to those observed with spontaneous inspiration.

Positive pressure lung inflation in the closed chest decreases venous return by decreasing the pressure-gradient from extrathoracic to thoracic veins.

Intermittent positive-atmospheric pressure respiration has a deleterious effect on venous return in hypovolemia.

In hypovolemia, intermittent positive-negative pressure respiration increases venous return relatively more than in the state of normovolemia.

In the open chest, positive pressure lung inflation impedes venous return, but the interposition of a negative phase between positive pressure lung inflations does not benefit the circulation significantly.

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