CIRCULATORY EFFECTS OF INCREASED PRESSURE IN THE AIRWAY *

HENRY K. BEECHER, M.D., H. STANLEY BENNETT, LT. (MC), U. S. N. R. AND DAVID L. BASSETT, M.D. †

Boston, Mass.

THE clinical use of an elevated pressure in the respiratory airway is increasing. In transpleural surgery it is used to aid oxygenation when a blowing pneumothorax is present, to help stabilize the mediastinum, and to inflate collapsed lung tissue. Anesthetists sometimes employ positive pressure in the airway to lower the diaphragm so that the liver or spleen may be well exposed during abdominal operations. Positive pressure is used to combat pulmonary edema; it is believed by many physicians that edema can be minimized or even overcome by safe increase of pressure in the airway.

That the procedure is without danger appears to be a common assumption. From a fairly extensive experience with this unquestionably useful technic in thoracic surgery, our view is that under some circumstances it may be dangerous. At times some patients will not tolerate even moderate increase in pressure; others show signs of deteriorating circulation as the elevated pressure is maintained. The inference to be drawn from the clinical cases is clear enough. Patients in good condition will tolerate a mean pressure of 10 cm. of water (above atmospheric pressure) for twenty or thirty minutes or more, but the circulation of patients in precarious condition often will deteriorate rapidly under such circumstances. Various changes in the circulation which occur on elevating pressure in the airway are described in this paper, and the probable physiologic basis for ill effects encountered clinically in certain cases is presented.

An opportunity for making the necessary observations arose in connection with experiments performed for another purpose. Experimental data are based upon 19 periods of pressure elevation in 6 dogs. Four dogs were under cyclopropane, and two under ether anesthesia. Since the response to the pressure elevation was comparable in all cases, we have considered the data together.

Systolic, diastolic and mean arterial pressure, central venous pressure, and blood flow in the carotid, femoral and mesenteric vascular beds were

* From the Department of Pharmacology and the Anesthesia Laboratory of the Harvard Medical School, Boston, Mass. This study had to be interrupted before we had carried it as far as planned. The consistency of the data obtained and the hazard sometimes present in the use of positive pressure justify, we believe, publication of the data at this time.

† Fellow of the National Research Council. Now located at Stanford University, Calif.

recorded and corresponding hydraulic impedances calculated as described by Bennett, Bassett and Beecher (1943). The pressure of the breathing bag was measured by means of a water manometer. In all cases the pressure was raised to a mean pressure of about 10 cm. of water with spontaneous respiratory fluctuations in pressure oscillating about this figure. Control pressures in the breathing bag and after release were approximately 0.1 to 0.2 cm. of water. The duration of elevation was usually 10 ± 2 minutes. When the animals were in bad condition from hemorrhage or deep anesthesia, the pressure was released much sooner to avoid killing the subject.

RESULTS

The results of the elevation of pressure in the airway under ether and under cyclopropane are alike in so far as can be judged from these experiments. Elevating the pressure of the breathing bag caused: great elevation in central venous pressure; some fall in systolic, diastolic and mean arterial pressure; a narrowing of pulse pressure; a decrease in flow in femoral and carotid arteries and in the superior mesenteric vein; a slowing of respiration; no consistent significant change in hydraulic impedance in the three vascular beds in which flow was recorded, and no consistent changes in heart rate. Release of pressure in the breathing bag tended to restore these factors to their original values, except in certain subjects in bad condition.

Typical records of the effect of elevation and release of pressure in the airway on blood flow and blood pressure are shown in figures 1 and 2. Averages of changes in all dogs are summarized in the table.

The average flow decrease, expressed in percentages in the mesenteric vein on elevation of pressure (see table) was greater than in the case of the carotid and femoral arteries, and the mean of the impedance changes calculated on the basis of these flow changes showed an appreciable increase. Actually, great variability was encountered in the mesenteric flow changes on elevation of the pressure of the bag. In the majority of the trials, the flow decreases in this vessel on elevation were proportionately no greater than those recorded in the femoral or carotid arteries. In two or three instances on elevation of the pressure, very great diminution in flow was encountered in the mesenteric vein. These few exceptional decreases were sufficient in extent to lower greatly the mean value of percentage flow and to elevate the mean of percentage impedance changes and to introduce a large standard error of the mean. It is our belief that these large decreases in flow in the mesenteric vein were not physiologic, but when present were probably due to kinking of the superior mesenteric vein against the flow meter cannula by the lowering of the diaphragm which occurred with positive pressure in the airway. Because of the inconsistencies in degree of the flow changes in the mesenteric vein and of the possibility for mechanical obstruction on lowering the diaphragm, we are not inclined to regard these cases of extreme slowing of flow in the mesenteric vein as significant.

The decrease in arterial pressure, the decrease in flow through the carotid and femoral arteries and the increase in mean central venous pressure were observed with great consistency in all the dogs on all 19 occasions of elevating pressure in the airway. The series included episodes of elevation of

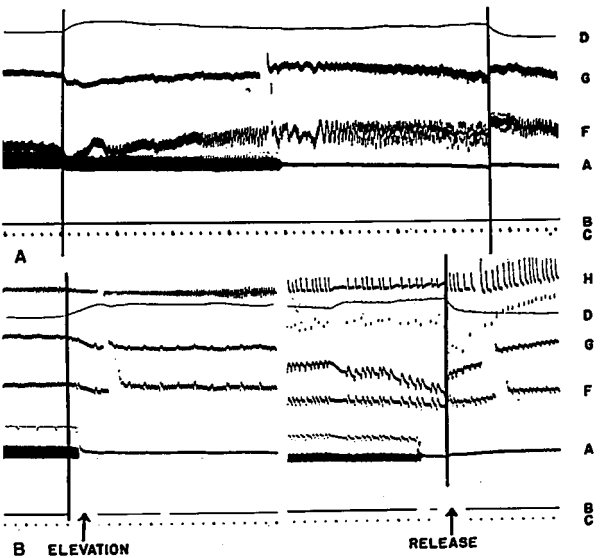

FIG. 1. Effect of elevating pressure of the breathing bag under cyclopropane (top) and under ether (bottom). Duration of ether elevation was 15 minutes, of cyclopropane 7 minutes 40 seconds.

Breaks on flow records denote points at which the flow meter circuits were checked for shifts due to parasitic currents. A shift in the line after the break indicates that a correction was made. Respiratory rate is recorded by the phasic changes in the mesenteric vein flow in the bottom figure. With this type of recording, small changes in the curve may represent significant numerical changes; for example, the carotid flow in the lower figure on elevation of bag pressure changed from 0.64 to 0.45 cc. per second, and at the same time the venous pressure rose from − 0.7 to + 4.0 mm. of mercury.

Key for both figures. A.—arterial blood pressure, either damped (mean) or undamped. B.—arterial pressure base line. C.—time, small marks, 10 seconds; large marks, 1 minute. D.—mean central venous pressure. F.—femoral artery flow. G.—carotid artery flow. H.—superior mesenteric vein flow.

pressure in dogs in good condition and in shock from hemorrhage and under conditions of both light and deep anesthesia.

The series is too small to permit an analysis of all differences between dogs in good condition and dogs in shock, but the following observations

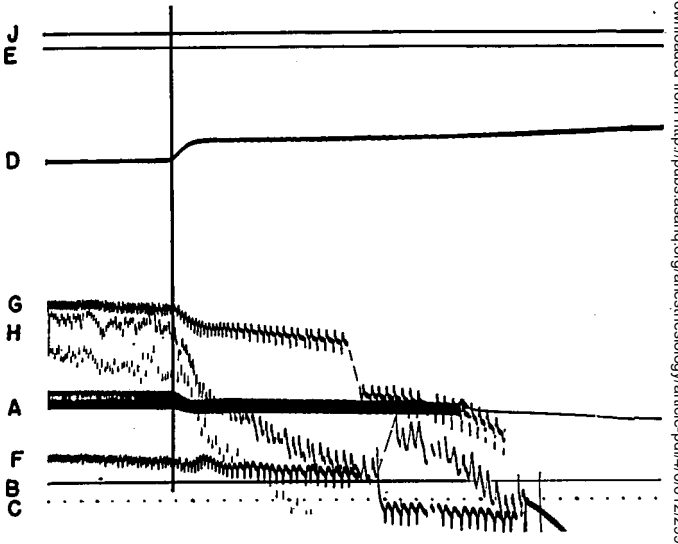

FIG. 2. Effect of elevating pressure in the airway in a dog in severe shock. At the vertical line, mean pressure of the breathing bag was raised to 10 cm. of water. The prompt rise in venous pressure and fall in arterial pressure was followed by a further progressive decline in arterial pressure and rise in venous pressure until both equilibrated at + 5 mm. of mercury at the death of the animal. Flow in mesenteric, femoral and carotid vessels diminished to zero (which records off the figure). A dotted line has been drawn to bridge the breaks in the carotid and mesenteric flow records produced when flow meter circuits were checked and corrected.

are worth recording. Dogs in good condition were greatly affected when pressure in the airway was elevated for as long as fifteen minutes. The same dogs, after going into shock from hemorrhage, particularly when under deep anesthesia, often showed severe circulatory impairment when the mean pressure in the airway was raised to 10 cm. of water. The circulation, as shown by the failing heart beat, by fall in arterial blood pressure and flow, and by acute rise in venous pressure, deteriorated so rapidly in these cases that the pressure in the airway had to be released before the expiration of the planned period of elevation to avoid killing the animal. When the pressure was not released under these circumstances, the animal died quickly (fig. 2).

COMMENT

The probable reason for the difference in tolerance of animals in good condition and of those in shock can be deduced from the pressure changes observed. The difference between mean central arterial and mean central

EFFECTS ON THE CIRCULATION OF ELEVATION AND RELEASE OF PRESSURE IN THE AIRWAY
Mean values based upon 19 pressure elevations in 6 dogs

	Arterial Blood Pressure in mm. Hg			Venous Pressure, mm. Hg. Actual pressure	Fem. Flow	Car. Flow	Mes. Flow	Fem. Imp.	Car. Imp.	Mes. Imp.	Heart Rate
	Systolic	Diastolic	Mean								
	Actual pressure				Control values before elevation in per cent						
Control before elevation	111±8	60±6	73±7	−2.3±0.7	100	100	100	100	100	100	150±17
	Changes in pressure				Percentages of control values						
After elevation	−9±1	−6±1	−7±1	+2.3±0.6	85±8	82±3	64±12	98±2	105±4	159±43	149±16
	Actual pressure				Control value before release in per cent*						
Before release	99±6	54±5	61±7	+2.2±0.9	100	100	100	100	100	100	138±14
	Changes in pressure				Percentage of pre-release values						
After release	+7±2	+2±1	+6±1	−2.4±0.7	124±6	124±6	141±19	100±4	101±5	95±12	132±14

* Since some dogs showed changes in flow because of progressive shock or changing depth of anesthesia during the period of elevation of bag pressure, a second control value for each flow and impedance figure was taken just before the release of pressure and compared with the corresponding figure after release. Thus these values represent acute changes on release, but do not necessarily indicate return of flow or impedance to their original pre-elevation values.

venous pressure provides the motive force which drives the blood through the peripheral vessels. Elevated pressure in the airway causes a rise in venous pressure as well as a fall in arterial pressure; thus the adverse effect on the circulation is greater than if arterial pressure alone were affected. A comparison of actual pressure changes in a dog in good condition with similar changes in a dog in shock serves to illustrate this point, and to bring out differences between them, which are masked by the mean figures in the table.

In a typical instance, a dog in good condition before elevation of pressure had a mean arterial pressure of 133 mm. of mercury and a central venous pressure of 0.0 mm. of mercury, giving an effective perfusion pressure of 133 mm. of mercury. After elevation, the arterial figure was lowered to 126 mm. and the venous raised to + 5.0 mm. yielding an effective perfusion pressure of 121 mm. of mercury. Although the mean arterial pressure was lowered only 7 mm. by elevating pressure in the airway, the effective perfusion pressure was lowered 12 mm. of mercury. It is obvious that the perfusion pressure of 121 mm. of mercury after elevation is adequate to maintain a brisk peripheral flow, and the animal tolerated well this elevation.

In a corresponding typical instance in a dog in shock, mean arterial pressure before elevation was 44 mm. and central venous pressure − 2.7

mm. of mercury, giving an effective perfusion pressure of 46.7 mm. of mercury. After elevation of pressure in the airway, the mean arterial pressure was 41 mm. and the mean venous pressure + 3.2 mm., giving an effective perfusion pressure of 37.8 mm. of mercury. Thus, although the mean arterial pressure fell only 3 mm., or 7 per cent, the effective perfusion pressure fell 8.9 mm., or 19 per cent. In this typical instance, flow through the peripheral vessels showed a marked decrease, the animal progressively declined, and after ten minutes of elevation, the mean arterial pressure had fallen to 29 mm. of mercury, and the mean perfusion pressure to 24.7 mm. of mercury, a 47 per cent fall from the pre-elevation pressure. Presumably, increased pressure in the airway interferes with the flow of blood through the lungs and impedes the emptying of the right side of the heart and the filling of the left, with consequent rises in central venous pressure. Judging by the consistency of the decreased flow in femoral, carotid, and mesenteric veins, it seems safe to assume that cardiac output likewise falls.

These examples offer at least a partial explanation as to why an elevation of pressure in the airway is of little moment in animals in good condition, whereas in animals with arterial pressure already critically low, as in shock, increased pressure in the airway may work harm far beyond that to be expected from the fall in arterial pressure alone.

These observations on dogs provide a physiologic explanation for the clinical observation that patients in shock do not tolerate well increased pressure in the airway. They indicate that this procedure is not innocuous, and suggest that although it is perhaps relatively safe in patients in good condition, it is dangerous and may be lethal in patients in shock or in otherwise poor condition. When the procedure introduces additional stresses in a patient in poor condition, he may be overwhelmed. The slowing of respiration is an additional hazard in patients with respiratory impairment.

In clinical practice, 10 cm. of water is a moderate mean pressure. Higher pressures in the breathing bag are in common clinical use, often for prolonged periods of time. The effects on the circulation of these higher pressures probably would be greater, and in certain cases more deleterious than the effects of pressure of 10 cm. of water. (Pressure for expanding collapsed lung tissue in the presence of open pneumothorax probably should never exceed 20 cm. of water, and this for only a few minutes.)

All observations were made with the subject under anesthesia. Deep anesthesia magnified the seriousness of the effect of pressure increase. It is quite probable that unanesthetized subjects would withstand pressure increase in the airway better than persons under general anesthesia, although it seems clear that any procedure which lowers systemic arterial pressure while elevating central venous pressure will have an adverse effect, especially when the blood pressure is critically low. Our results strongly suggest that badly traumatized subjects or subjects otherwise in

poor circulatory condition should be watched carefully if this technic is to be employed. Such a statement is by no means to be construed as an argument against the use of positive pressure when it may be of value.

SUMMARY

Elevation of pressure in the breathing bag in dogs under anesthesia produced a great rise in venous pressure, a fall in systolic, diastolic and mean blood pressure, a decrease in flow in the femoral and carotid arteries and in the superior mesenteric vein, with a slowing of respiration. Release of the airway pressure tended to restore these factors to normal. The pressure effects appeared to be tolerated well by animals in good condition, but in animals with poor circulation, increased pressure in the airway was deleterious and could cause death.

REFERENCE

Bennett, H. S.; Bassett, D. L., and Beecher, H. K.: Influence of Anesthesia (Ether, Cyclopropane, Sodium Evipal) on the Circulation under Normal and Shock Conditions. In press. J. Clin. Investig., 23: 181–208. 1944.

MEETING OF THE AMERICAN SOCIETY OF ANESTHETISTS, INC.

ROOM 440, NEW YORK ACADEMY OF MEDICINE
2 EAST 103RD STREET, NEW YORK CITY

December 9, 1943—7:30 P.M.

1. "The Role of Anoxia in Gastrointestinal Effects of Anesthesia." 50 minutes.
   By Edward J. Van Liere, Ph.D., M.D., Professor of Physiology and Dean of the Medical School of West Virginia University, Morgantown, W. Va.
   Discussant: To be announced.
2. "Clinical and Laboratory Observations on the Use of Curare for Muscular Relaxation During Inhalation Anesthesia." 40 minutes.
   By Stuart C. Cullen, M.D., Assistant Professor of Surgery (Anes.), University Hospitals, The State University of Iowa, Iowa City, Iowa.
   Discussant: Harold R. Griffith, M.D., Montreal, Quebec, Canada.