EFFECTS OF COMMON RESPIRATORY PHENOMENA DURING GENERAL ANESTHESIA ON ARTERIAL BLOOD PRESSURE AND PULSE

RICHARD E. JONES, M.D., MARTIN HELRICH, M.D.
JAMES E. ECKENHOFF, M.D.

During the course of investigations into the effects of anesthetic and preanesthetic agents on the respiration and the circulation of man, various respiratory problems commonly encountered during general anesthesia were observed. The circulatory responses to these complications were often marked and of concern. The abruptness, the magnitude, and the violent fluctuations of these reactions were frequently a source of surprise to our resident anesthetists. It seemed, therefore, that a presentation of some of the observed changes would be a worth while contribution to teachers of anesthesiology. While the descriptions of the responses are not new, as far as we are aware they have never been consolidated within a single article.

METHODS

Intra-arterial blood pressure was measured with a Lilly capacitance manometer (1) employing a small plastic catheter or needle introduced into the brachial artery. The changes in blood pressure were recorded with a Brush oscillograph (2). Measurements also were made of respiratory rate, tidal and minute volume, end expiratory carbon dioxide concentrations, and electrocardiographic and electroencephalographic changes (3). Since only the measurements of blood pressure are pertinent to the discussion, other data obtained will not be mentioned further in this report.

The studies were conducted, for the most part, on patients in the operating room prior to and during the administration of anesthesia. A few of the records were obtained from resident physicians and medical students who were subjects for study (4, 5).

RESULTS

1. Cyclic Variations in Blood Pressure. This phenomenon has long been recognized. As seen in figure 1, blood pressure rises during expiration and falls during inspiration. The variation observed in the illustration is from 122/100 mm. Hg to 104/86 mm. Hg. Such a rhythmical rise and fall differs from patient to patient and even within

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the same individual under changing circumstances. Ether et al. (2) have observed variations of as much as 70 mm. Hg in systolic pressure during normal cyclic fluctuations.

The explanation for these changes has been sought in the laboratory. Arterial pressure falls when a dog’s lungs are inflated by negative pressure upon their external surface. It rises again when the pressure is removed and the lungs are allowed to collapse (6). Under these conditions, an average increase of 25 cc. has been observed in right ventricular output coincident with inspiration (7). This closely approximated the increase in the capacity of the pulmonary vascular bed occurring on inspiration (8). With expiration, this blood is forced into the left heart, cardiac output increases, and blood pressure rises (9). It would appear that the mechanism of the normal respiratory variations in blood pressure in man are similar.

2. Deep Inspiration and Forced Expiration. Figure 2 demonstrates the fall observed in arterial blood pressure coincident with deep inspiration. This decline from 100/60 mm. Hg to 78/38 mm. Hg resulted from: (1) a decrease in blood flow to the left ventricle as the increased pulmonary vascular bed was filled; and (2) in this patient, bradycardia. Starr and Friedland (9) have pointed out that the slowing in heart rate observed in sinus arrhythmia occurs during that portion of the respiratory cycle when the cardiac output is lowest. They suggest that this is due to changes in cardiac filling produced by respiratory movements.

EFFECT OF DEEP INSPIRATION UPON BLOOD PRESSURE AND PULSE

![Figure 2](http://anesthesiology.pubs.asahq.org/pdfaccess.ashx?url=/data/journals/jasa/931677/)
COMMON RESPIRATORY PHENOMENA

EFFECT OF FORCED EXPIRATION UPON PULSE AND BLOOD PRESSURE

From the above, it would be expected that with forced expiration, arterial blood pressure would rise with the "squeezing out" of the contents of the pulmonary vascular bed into the left ventricle. Figure 3 confirms this.

3. Effects of Raised Airway Pressure. The circulatory responses to the Valsalva maneuver have been described in detail (10). They

EFFECT OF FORCED EXPIRATION AGAINST CLOSED GLOTTIS UPON BLOOD PRESSURE AND PULSE

will be discussed here briefly since they pertain to phenomena seen during the administration of anesthesia. In the conscious subject, forced expiration against a closed glottis usually affects the circulation as follows (fig. 4): (1) A sharp increase in systolic, diastolic, and pulse pressure lasting one to two seconds; (2) a decrease in systolic, diastolic, and pulse pressure which becomes maximal in six to ten seconds; (3) a slow return of all three toward control values; (4) with release

EFFECT OF A COUGH UPON BLOOD PRESSURE AND PULSE

Fig. 3.

Fig. 4.

Fig. 5.
of pressure, a sharp slight decrease in all values; and (5) an "overshoot" above control values which reaches a maximum in four to seven seconds.

Modifications of the Valsalva maneuver commonly are seen during general anesthesia. These include:

(a) Coughing. A cough is a forced expiration against a closed glottis which suddenly opens. A cough is, therefore, a brief Valsalva maneuver. There is an initial sharp rise in systolic, diastolic, and pulse pressure. In the patient whose tracing is reproduced in figure 5, blood pressure rose from 140/80 to 185/106 with a single cough.

(b) Retching. Figure 6 is a tracing from a conscious patient with spinal anesthesia to a sensory level of the sixth thoracic segment. The increase in blood pressure was due to retching which also represents an increase in intrathoracic pressure against a closed glottis. This patient's systolic pressure rose from 180 to 275 mm. Hg.

EFFECT OF A "STORMY INDUCTION" UPON BLOOD PRESSURE AND PULSE

Fig. 7.
The initial rise in systolic, diastolic, and pulse pressures in response to raised airway pressure during a Valsalva maneuver results from the expulsion of blood from the vascular bed of the lung into the left ventricle and systemic circulation, and from compression of the thoracic aorta. This same mechanism probably operates to produce the rise in systemic pressure noted during coughing and retching. It is noteworthy that, after spiking to 275 mm. Hg (fig. 6), the pressure fell precipitously to approximately 100/93 mm. Hg. This represents a period during which the pulmonary vascular reservoir was relatively empty and the pressure on the thoracic vessels was sharply reduced.

(c) Partial Airway Obstruction. Figure 7 presents 4 sections from a tracing made during a period of "stormy induction" of nitrous oxide, oxygen, and ether anesthesia. The patient's airway became partially obstructed, and she coughed at intervals. The increased variations in intrathoracic pressure produced by partial obstruction resulted in an exaggeration of the normal cyclic patterns in arterial pressure (7-B). An oropharyngeal airway was inserted to relieve the obstruction. This led to a paroxysm of coughing and marked fluctuation in blood pressure (7-C). The patient's airway remained partially obstructed and she continued to cough (7-D). In the interval between 7-A and 7-D, the blood pressure rose from 108/80 to 166/128 mm. Hg, and continued to fluctuate rapidly for many minutes.

4. Application of Positive Airway Pressure to Insure Ventilation. Initially, as positive airway pressure is applied, blood is forced from the pulmonary vascular bed by the increased pressure just as in the Valsalva maneuver (9). Figure 8 illustrates a transient rise in brachial arterial pressure produced by assisted respiration and probably due to the increase in left ventricular output.

If positive airway pressure is maintained for more than a few seconds, a different picture ensues. A proportion of the applied pressure is absorbed by the elasticity of the lungs, but the remainder is trans-
mitted to the pulmonary vasculature, the great veins, and the heart. To preserve blood flow to the right heart and from the right to the left heart, the pressure within these vessels must increase to compensate for the raised intrapulmonary pressure (11). If this adjustment fails, cardiac output and arterial pressure fall in proportion to the increased pressure (10). The ability of the conscious subject to withstand raised airway pressure is believed due to the occurrence of a generalized vasoconstriction mediated by the pressoreceptors and activated by the fall in the systemic arterial pressure. However, in anesthetized individuals, this reflex may be depressed or absent. Figure 9 is a tracing from a patient under general anesthesia in whom blood pressure de-

**EFFECT OF SUSTAINED POSITIVE AIRWAY PRESSURE UPON THE PULSE AND BLOOD PRESSURE**

![Graph](image1)

**EFFECT OF CONTROLLED RESPIRATION UPON BLOOD PRESSURE AND PULSE**

![Graph](image2)

clined from 100/76 to 62/55 mm. Hg before the airway pressure was released. Compensatory vasoconstriction failed to function during anesthesia in this instance.

If intermittent positive pressure is substituted for continuous positive pressure, the fall in blood pressure may be obviated or
minimized. Suspension of positive pressure allows for filling of the great veins and the right heart, and helps to maintain right to left heart blood flow. This is demonstrated by figure 10, a tracing made during a brief period of controlled respiration. The arterial pressure dropped from 148/95 to 138/93, but was maintained at that level.

**DISCUSSION**

Some of the blood pressure changes mentioned are brief and may return to normal rapidly if corrective measures are taken. Others are explosive with abrupt, marked alterations with violent fluctuations. In the ordinary practice of anesthesiology, circulatory effects of respiratory difficulties usually are missed because the anesthetist is busy correcting the complication and does not have time to take the blood pressure, or he misses the peak changes because of the rapid fluctuation in the blood pressure.

If these phenomena occur during the induction period of anesthesia, they can be overlooked readily. This is the period during which the anesthetist is least likely to record the blood pressure. It is common to hear an anesthetist remark that there were no changes in the blood pressure and the pulse although he and the patient have just completed thirty minutes of a "stormy induction" during which no attempt has been made to take either blood pressure or pulse. It is important that residents be instructed to record blood pressure and pulse even more frequently in the induction stage than during the maintenance phase of anesthesia. When possible, an assistant or a nurse might be asked to record these vital signs.

The significance of the observed changes is difficult to define. Obviously, from the numbers of these complications encountered daily in an anesthetic clinic, the average human being tolerates them surprisingly well. Nevertheless, the morbidity and the mortality of anesthetics is measured in terms of thousands of anesthesias. Were it possible definitely to define the end result of these respiratory complications on the circulation, our concern might be greater than at present. None will deny that the circulatory effects of these complications discussed are undesirable; the poorer the risk, the more undesirable.

The shorter, more explosive rise and fluctuations of blood pressure may be of less concern than the longer periods of hypotension observed with maintained elevation in airway pressure. Nevertheless, in an elderly patient, sudden and precipitous rises in blood pressure may produce cerebral hemorrhage. In the patient with poor myocardial reserve, sudden blood pressure changes may lead to a sharp reduction in blood supply and myocardial infarction. In both situations mentioned, however, the sharp decline of blood pressure after the upward spike may be the dangerous factor. In patients with auricular fibril-
lation or those with a history of embolization, these violent changes may produce further embolic phenomena. One also cannot overlook the occasional cardiac arrest seen following aspiration of tracheal secretions either in the patient immediately postoperatively as his endotracheal tube is removed, or on the ward as he is being treated prophylactically for atelectasis. These deaths may be connected intimately with sudden elevations in blood pressure due to brief increases in airway pressure.

The influence of improperly applied positive airway pressure can be seen frequently in most anesthetic clinics. We do not decry the use of positive pressure or of controlled respiration. However, if the mechanism of hypotension occurring under these circumstances is understood, difficulties usually can be obviated by a change to assisted respiration or spontaneous respiration. Fortunately, most patients are able to compensate for increases in their airway pressure by peripheral vasoconstriction and elevation in central venous pressure. Occasionally compensation does not occur. If such patients have been made apneic by the injudicious use of anesthetic or relaxant drugs and are unable to return to spontaneous respiratory movements at ambient pressures, the outcome may be disastrous. The fatality may occur at the time of operation or hours to days later from myocardial damage due to prolonged hypotension during anesthesis. The latter reasoning has been our principal reason for avoiding the rapid or "blitz" techniques of inducing anesthesia in all but good risk patients.

The relationship of respiratory complications observed during and following the administration of anesthetics to morbidity or mortality is rarely cleared up. However, an awareness of the mechanisms by which the complications influence the circulation is important in understanding the fundamental principles of anesthesiology.

**Summary**

The effects of certain respiratory complications on the circulation, commonly observed during general anesthesiain, have been described. These have included the response to deep inspiration, forced expiration, the Valsalva maneuver, coughing, retching, partial airway obstruction, and positive airway pressure. The etiology of the responses and their possible clinical significance has been discussed.

**References**


