# *The Ventilatory Response of Infants to Airway Resistance*

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**The respiratory response of anesthetized infants to acute increases in airway resistance was studied. Measured were parameters of ventilation, ventilatory mechanics and acid-base balance. Results showed that the infant responds to sudden increases in airway resistance by decreasing ventilatory rate, increasing tidal volume, decreasing airflow rate and decreasing minute volume. It has been shown that the infant's reflex compensation for increased airway resistance was well maintained by an increase of over 200 per cent in work of breathing. Adequate alveolar ventilation was carried out as indicated by the absence of carbon dioxide retention and acidosis. Of interest was the finding that pulmonary compliance during the recovery period was generally greater than found during the control period. It is stressed that carbon dioxide retention and acidosis are not early signs of abnormal airway resistance but late signs that indicate fatigue and respiratory decompensation.**

Received from the Division of Anesthesiology, Department of Surgery, The Johns Hopkins University School of Medicine and Hospital, Baltimore, Maryland. Presented at the Annual Meeting of the American Society of Anesthesiologists, Denver, Colorado, October 26, 1965; accepted for publication November 18, 1965. Supported by a grant from the Joseph P. Kennedy, Jr. Memorial Fluid Research Fund.

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IN the design of anesthetic techniques and equipment for pediatric patients, considerable attention is directed toward minimizing ventilatory resistance. Little information is available, however, on how pediatric patients react to changes in airway resistance. The purpose of this paper is to describe the way in which infants respond to acute increases in airway resistance with regard to ventilation, ventilatory mechanics, and acid-base balance.

## Material and Methods

Ten infants were studied whose ages ranged from 2 weeks to 11 months and weights from 3.6 to 7.1 kg. All underwent elective operation for uncomplicated inguinal hernia, and were considered to be in good health by history, physical examination, and admission laboratory data. The studies were performed under light halothane anesthesia before the start of surgery in 4 infants and after surgery in 6 infants.

After preanesthetic medication with 0.1 mg. of scopolamine, and 2 mg. per kilogram body weight of pentobarbital, anesthesia was induced with nitrous oxide, oxygen and halothane. Following induction, nitrous oxide was discontinued, and a maximal sized, tapered (Cole), endotracheal tube was inserted without the use of neuromuscular blocking drugs. Anesthesia was maintained with halothane and oxygen administered through an Ayre's tube. A side-arm portal was incorporated in the endotracheal adaptor through which inhaled halothane concentration was determined by an infra-red analyzer. The concentration of the halothane was kept between 1 and 1.5%; total flow of oxygen was 6 liters per minute. To measure respiratory air flow rate, a Fleisch (double O) pneumotachometer head was interposed between the endotracheal adaptor and the Y-piece. The dead space of the pneu-

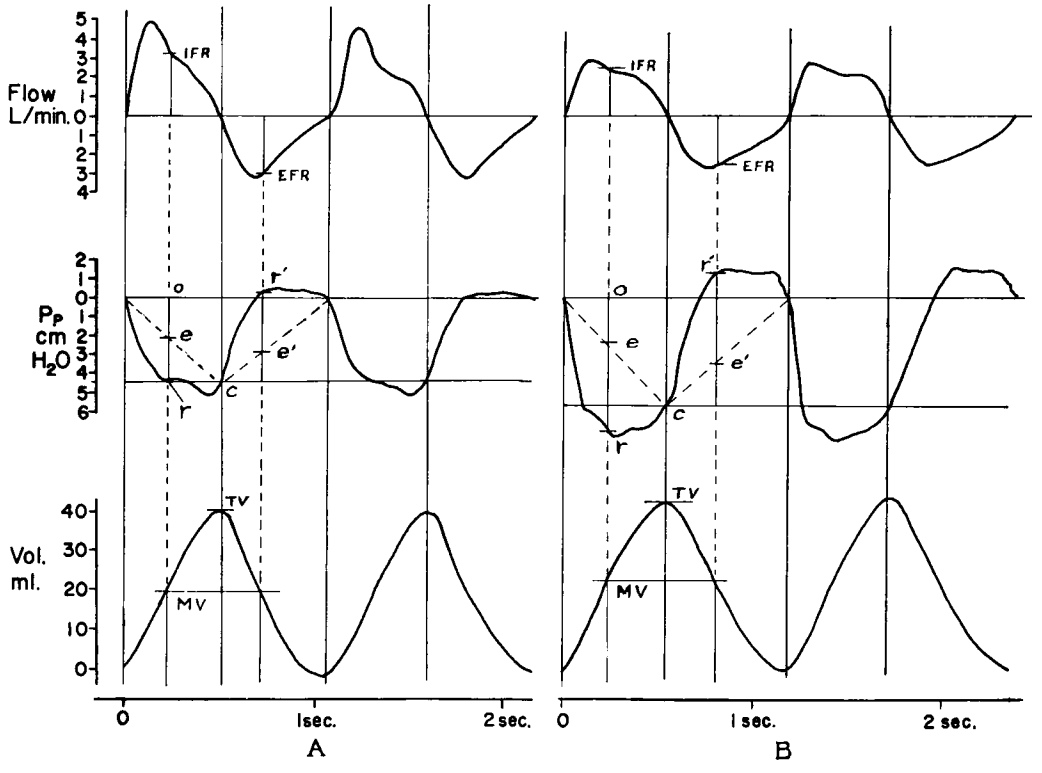


FIG. 1. T. G., 3 mo. Tracings of airflow rate, intraesophageal pressure, and tidal volume during control period (A) and period of increased airway resistance (B). Total resistive pressure ($\Delta$ *P*dyn) at mid-tidal volume equals *o-r* during inspiration and *c-r'* during expiration. Elastic resistive pressure ($\Delta$ *Pe*) is represented by *o-e* and *c-e'* for inspiration and expiration, respectively. Total frictional resistance in g. cm. per liter per second is measured by the formula:

$$R\ (I) \text{ or } (E) = \frac{\Delta\ P\text{dyn} - \Delta\ Pe}{\dot{V}_I \text{ or } \dot{V}_E \text{ at } V/2}$$

motachometer and Y-piece assembly was 4.2 ml. The proximal and distal leads of the pneumotachometer were led into an electronic integrator; the electrical outputs from the pneumotachograph and integrator were in turn amplified and recorded on a multiple channel recorder to give a continuous tracing of respiratory airflow rate and tidal volume. Airflow calibrations were carried out with a variable orifice flowmeter. Galvanic deflections were linear for flow rates from 0 to 170 ml. per second. Volume was calibrated with an oiled 50 ml. syringe which likewise gave a deflection that was linear from 0 to 50 ml.

Changes in intrapleural pressure were measured indirectly by an intra-esophageal air-filled balloon. The balloon was made of three dipped layers of latex and measured 0.5 cm. in diameter and 4 to 5 cm. in length. The balloon was affixed to a perforated polyethylene (1.4 mm.) catheter. Following tracheal intubation, the balloon was inserted into the mid-thoracic esophagus and connected to a strain gauge previously calibrated in centimeters of water. Intrapleural pressure was electronically recorded simultaneously with the other data. The optimum position of the balloon was determined by making the infants apneic by means of hyperventilation and then positioning the catheter tip where there was minimal recording of the cardiac impulse.

Pulmonary compliance was calculated during control and recovery periods from the ratio of tidal volume to the change in intra-eso-

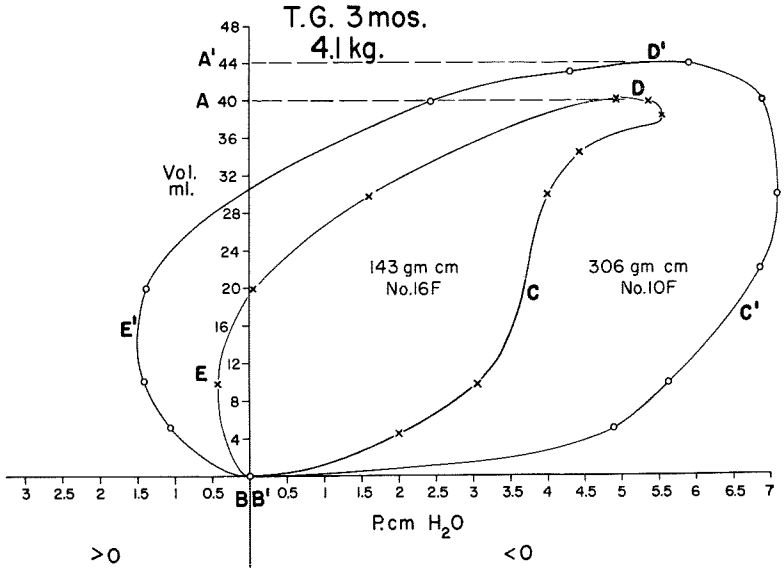


FIG. 2. T. G., 3 mo. Pressure-volume diagram graphically representing respiratory work during control period (ABCDE) and period of increased airway resistance (A'B'C' D'E'). Work in g. cm. determined by measuring these areas. BCD—inspiratory phase, DEB—expiratory phase of respiratory cycle. Area to left of ordinate enclosed by portion of expiratory phase of loop represents expiratory work.

phageal pressure measured between points of zero flow at the extremes of tidal volume. Inspiratory and expiratory resistance were measured at points of mid-ventilatory volume (fig. 1). The formula [1] used was:

$$R(I) \text{ or } R(E) = \frac{\Delta P \text{ dyn} - \Delta Pe}{\dot{V}}$$

$\dot{V}$ = airflow rate (liters/second) at mid-inspiratory or expiratory volume. $\Delta$ $P$dyn = total esophageal pressure change (cm. of water) from the beginning of inspiration or expiration to midpoint of the given respiratory phase. $\Delta$ $Pe$ = elastic portion of the resistive pressure that is subtracted from the total pressure change in calculating respiratory resistance. Thus the resistance measured was the sum of the frictional airway resistance and the frictional tissue resistance.[2]

TABLE 1. Internal diameters and cross-sectional areas of endotracheal catheters

| French Size | Diameter (mm.) | Area mm.$^2$ |
|---|---|---|
| 18 | 4.0 | 12.6 |
| 16 | 3.6 | 10.2 |
| 14 | 3.0 | 7.1 |
| 12 | 2.5 | 4.9 |
| 10 | 2.2 | 3.8 |
| 8 | 1.5 | 1.8 |

From the simultaneous recordings of esophageal pressure changes and ventilatory volume, pressure-volume loops for the respiratory cycle were plotted, and the average work done on the lungs per breath was determined from the graphic representation [3] (fig. 2).

The $p$H, $P_{CO_2}$, and standard bicarbonate were measured on samples of arterialized capillary blood. Blood was taken from the finger tip of a hand that had been warmed for a period of 3 minutes. Free capillary flow was obtained by puncturing the finger tip with an 18 gauge needle. The blood was drawn into heparinized capillary tubes, and immediately analyzed utilizing Astrup AME-1 Micro Equipment according to the technique described by Astrup.[4]

Body temperature was monitored with a rectal thermocouple and maintained within a normal range by means of a heating blanket. In addition to constant auscultation of precordial heart sounds, blood pressure was frequently measured with a 2.5 cm. rubber arm cuff.

## Procedure

With the establishment of a stable level of halothane anesthesia, a rapid tracing was obtained (25 or 50 mm. per second) of ventilatory airflow rate, tidal volume, and esophageal pressure; a blood sample was taken for the

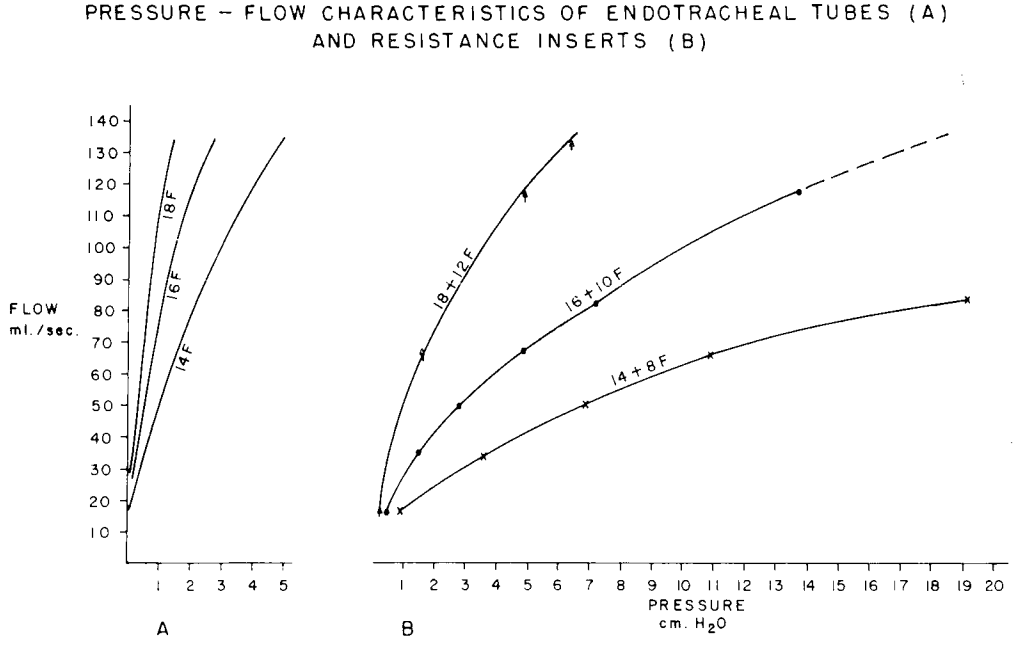


FIG. 3. Pressure-flow characteristics of endotracheal tubes (*A*); and resistance inserts (*B*).

determination of acid-base chemistries. The endotracheal adaptor was then disconnected, and a second endotracheal tube with an outer diameter 6 French sizes smaller than the original endotracheal tube was inserted into the lumen of the first tube. The adaptor was replaced and the infant allowed to breathe through the lumen of the smaller tube for a period of 10 minutes. At the end of the resistance period, a *rapid* tracing was again obtained (tracings were taken continuously), and a second sample of blood drawn for the determination of *p*H, $P_{CO_2}$ and standard bimarbonate. The resistance was then removed. The infant was allowed to breathe through the original endotracheal tube for a 10 minute recovery period after which a final ventilatory and pressure tracing was obtained, and a third sample of blood drawn for chemistries. The internal diameters and cross-sectional areas of the endotracheal tubes used are given in table 1, and their pressure-flow characteristics meas-

TABLE 2. Ventilation Changes with Airway Resistance

| Infant | Age (mo.) | Wt. (kg.) | Airway French + | | V (ml.) | | | f | | | M.V. (ml.) | | | $\dot{V}_I$* (ml./sec.) | | | $\dot{V}_E$* (ml./sec.) | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | | | C | R | C | R | % | C | R | % | C | R | % | C | R | % | C | R | % |
| 1 | 3 | 4.1 | 16 | 10 | 40 | 44 | 10.0 | 55 | 50 | −09.1 | 2200 | 2200 | 00.0 | 56 | 43 | −23.2 | 52 | 43 | −17.3 |
| 2 | 3 | 4.2 | 16 | 10 | 38 | 34 | −10.5 | 78 | 75 | −03.8 | 2964 | 2550 | −13.8 | 75 | 56 | −25.3 | 63 | 42 | −33.3 |
| 3 | 5 | 7.3 | 16 | 10 | 32 | 40 | 18.8 | 88 | 58 | −34.1 | 2816 | 2320 | −17.7 | 80 | 67 | −16.3 | 66 | 57 | −13.7 |
| 4 | 7 | 6.2 | 18 | 12 | 46 | 50 | −08.7 | 50 | 48 | −04.0 | 2350 | 2400 | 02.1 | 96 | 92 | −04.2 | 93 | 90 | −03.2 |
| 5 | 11 | 6.9 | 18 | 12 | 58 | 65 | 12.1 | 52 | 44 | −15.4 | 3016 | 2860 | −05.2 | 106 | 100 | −05.7 | 110 | 106 | −03.7 |
| 6 | 6 | 6.6 | 18 | 12 | 24 | 28 | 16.7 | 54 | 44 | −18.5 | 1296 | 1232 | −04.9 | 70 | 55 | −21.4 | 58 | 56 | −03.4 |
| 7 | 2 | 3.7 | 14 | 8 | 20 | 22 | 10.0 | 42 | 36 | −14.3 | 840 | 802 | −04.5 | 50 | 40 | −20.0 | 48 | 46 | −04.2 |
| 8 | 2 wks. | 3.6 | 14 | 8 | 14 | 14 | 27.3 | 57 | 42 | −08.8 | 627 | 588 | −06.2 | 28 | 26 | −07.2 | 30 | 24 | −20.0 |
| 9 | 4 | 5.7 | 16 | 10 | 34 | 37 | 08.8 | 68 | 60 | −11.7 | 2312 | 2220 | −04.0 | 47 | 45 | −04.3 | 46 | 45 | −02.2 |
| 10 | 4 | 7.1 | 16 | 10 | 24 | 30 | 25.0 | 90 | 54 | −10.0 | 2160 | 1620 | −24.6 | 46 | 32 | −13.0 | 40 | 34 | −15.0 |
| Mean percentile change | | | | | | +12.3 | | | −16.0 | | | −7.9 | | | −14.1 | | | −11.6 | |

C = Control; R = Resistance; % = Percentage Change.
* Inspiratory and expiratory flow rates measured at mid-tidal volume.
† Luminal diameters and areas given in table 1.

TABLE 3. Changes in Ventilation Mechanics with Airway Resistance*

| No. | Airway French | | RI† (cm. $H_2O$/l/sec.) | | | RE† (cm. $H_2O$/l/sec.) | | | Work/Breath (gm.cm.) | | | Exp. Work per Breath | | | Compliance‡ (ml./cm. $H_2O$) | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | C | R | C | R | % | C | R | % | C | R | % | C | R | % | C | Recovery | % |
| 1 | 16 | 10 | 39 | 109 | 180 | 57 | 123 | 116 | 143 | 306 | 114 | 5 | 33 | 560 | 9 | 11 | 22 |
| 2 | 16 | 10 | 21 | 72 | 243 | 43 | 86 | 100 | 151 | 235 | 56 | 9 | 37 | 311 | 7 | 8 | 14 |
| 3 | 16 | 10 | 27 | 90 | 233 | 38 | 77 | 103 | 101 | 304 | 201 | 9 | 31 | 244 | 9 | 11 | 22 |
| 4 | 18 | 12 | 28 | 77 | 173 | 27 | 66 | 144 | 169 | 471 | 179 | 16 | 82 | 412 | 12 | 14 | 17 |
| 5 | 18 | 12 | 30 | 82 | 173 | 29 | 69 | 138 | 325 | 695 | 114 | 24 | 93 | 288 | 11 | 12 | 9 |
| 6 | 18 | 12 | 28 | 115 | 311 | 32 | 135 | 322 | 68 | 201 | 195 | 3 | 26 | 766 | 5 | 5 | 0 |
| 7 | 14 | 8 | 50 | 160 | 220 | 41 | 152 | 271 | 47 | 230 | 389 | 2 | 34 | 1600 | 4 | 6 | 50 |
| 8 | 14 | 8 | 50 | 230 | 360 | 52 | 234 | 350 | 57 | 258 | 353 | 2 | 57 | 2750 | 5 | 6 | 20 |
| 9 | 16 | 10 | 47 | 155 | 229 | 35 | 132 | 277 | 92 | 373 | 305 | 9 | 49 | 444 | 10 | 13 | 30 |
| 10 | 16 | 10 | 65 | 275 | 323 | 60 | 236 | 293 | 135 | 367 | 172 | 15 | 64 | 326 | 6 | 10 | 66 |
| Mean percentile change | | | +241 | | | +211 | | | +205 | | | +770 | | | +25 | | |

C – Control; R – Resistance; % – Percent Change.

* Ages and weights given in table 2.

† Inspiratory and expiratory frictional resistance measured at mid-tidal volume.

‡ Dynamic complicance represents the mean values measured from 6 to 10 breaths during control and recovery period.

ured under steady state conditions are illustrated in figure 3.

## Results

The results of the individual studies on the 10 infants are summarized in tables 2, 3, and 4. Each of the 10 infants served as his own control, and per cent change of the various factors are shown.

The tidal volumes increased an average of 12.3 per cent with the introduction of the airway resistances. Only one infant failed to increase depth of breathing. Respiratory rates decreased in all infants with an average decrease of 16 per cent. Minute volumes were generally less after introduction of airway resistance. Both inspiratory and expiratory flow rates measured midway between extremes of tidal exchange invariably decreased (a fall of 14.1 and 11.6 per cent, respectively).

Calculated respiratory resistances increased an average of 241 per cent during inspiration and 211 per cent during expiration in response to the smaller airway (table 3). The average increase in work performed on the lungs with the added airway resistance was 205 per cent as calculated from pressure-volume loops constructed from representative respiratory cycles. Figure 2 shows a typical pressure-volume diagram which demonstrates the increase in total work performed on the lungs in response to the imposed airway resistance. Shown here, and seen in similarly constructed pressure-volume diagrams for all the other infants, is an area to the left of the volume ordinate enclosed by a portion of the expiratory phase of the loop. Such areas represent active work performed during the expiratory phase of the respiratory cycle and were seen invariably during both the control periods and the periods of increased airway resistance. From table 3, it is seen that this expiratory work increased an average of 770 per cent with the added resistance to breathing. Dynamic compliance, calculated from 6 to 10 representative breaths during the control and recovery periods, showed an average increase of 25 per cent with the removal of the resistance when compared to the control values. Only one infant failed to increase his compliance. Table 4 shows that there were minimal changes in acid-base balance with the increase in airway resistance. The average decrease in *p*H was less than 0.01 units, the average increase in

TABLE 4. Arterial Acid-Base Balance Changes with Airway Resistance

| Infant* | Airway (French) | | pH | | $P_{CO_2}$ (mm.Hg) | | Stand. Bicarb. mdg/l. | |
|---|---|---|---|---|---|---|---|---|
| | C | R | C | R | C | R | C | R |
| 1 | 16 | 10 | 7.32 | 7.33 | 42 | 41 | 20 | 20 |
| 2 | 16 | 10 | 7.31 | 7.29 | 40 | 46 | 20 | 21 |
| 3 | 16 | 10 | 7.34 | 7.33 | 40 | 41 | 21 | 21 |
| 4 | 18 | 12 | 7.32 | 7.31 | 42 | 44 | 21 | 21 |
| 5 | 18 | 12 | 7.27 | 7.25 | 58 | 60 | 22 | 22 |
| 6 | 18 | 12 | 7.32 | 7.33 | 46 | 45 | 22 | 21 |
| 7 | 14 | 8 | 7.30 | 7.29 | 52 | 51 | 22 | 21 |
| 8 | 14 | 8 | 7.34 | 7.34 | 44 | 45 | 23 | 23 |
| 9 | 16 | 10 | 7.35 | 7.36 | 47 | 49 | 23 | 24 |
| 10 | 16 | 10 | 7.29 | 7.30 | 55 | 52 | 21 | 21 |
| Mean change | | | $<-0.01$ | | $<+2$mm.Hg | | none | |

* Ages and weights given in table 2.

carbon dioxide tension was less than 2 mm. of mercury, and there was no change in the derived standard bicarbonate.

## Discussion

Several investigators [5, 6, 7] have shown that an awake adult human subjected to increased airway resistance increases depth and slows the rate of breathing. The infants reported in this study responded to added airway resistance by increasing tidal volume, slowing respiratory rate, decreasing inspiratory and expiratory airflow rate, and decreasing minute volume. Although most of the infants had developed mild respiratory and metabolic acidosis by the time control samples had been taken, the addition of an airway resistance did not result in significantly greater carbon dioxide retention. In spite of the fall in minute volume, it would appear that an equally effective ventilation was obtained with a slower respiratory rate and a slightly larger tidal volume because of the effect of moving less dead space gas.

The maintenance of the control acid-base balance values during the period of increased airway resistance is accomplished by a marked increase in respiratory work, both during inspiration and expiration. McIlroy [8] and his coworkers showed that with added elastic resistance, the respiratory rate increases and the tidal volume decreases, and that with added nonelastic resistance the rate decreases and the tidal volume increases. They suggested an optimal respiratory rate at which the work of breathing is least, which depends on a balance between the elastic and nonelastic resistive factors. When airway resistance increases, more energy is needed to overcome the resistance. Slow, deep breathing keeps airflow rates and therefore turbulence low, thereby avoiding excessive resistive work. By contrast, Cook and his group [9] pointed out that infants with hyaline membrane disease, with marked decrease in lung compliance, increase respiratory rate and thereby minimize the amount of work done against elastic resistance. Mead [10] has subsequently demonstrated that expenditure of muscular force rather than work is the important factor in regulating patterns of breathing.

Average work done on the lungs was arrived at graphically in this study rather than by utilizing the formula of Otis, Fenn, and Rahn [11] or the simplified formula of McIlroy.[9] The formula of Otis assumes that the second order resistive factor of turbulence is negligible, and McIlroy's formula is based on the fact that normally 70 per cent of pulmonary work is elastic. Neither of these assumptions hold true when studying the effects of increased airway resistance. In the present study, an additional element of turbulence was undoubtedly caused by the side-arm port incorporated in the endotracheal adaptor used for sampling

the halothane concentration. This element of turbulence is of little importance in the study since it was a factor during both the control and resistance periods. The resistance caused by the adaptor was not measured when diagramming the pressure-flow characteristics of the endotracheal tubes shown in figure 3.

Dynamic compliance was measured during the control and recovery periods and was found to have increased during the interval in all but one of the infants (table 3, case 6). Compliance was not calculated during the resistance period since increased airway resistance does not allow complete filling of the air spaces in the time allowed. The increased pulmonary compliance is probably the result of a greater functional residual capacity which Whittenberger [12] has shown to increase in response to airway resistance.

Since there was no significant change in arterial carbon dioxide tension, what is the mechanism that explains the marked increase in respiratory work in response to added airway resistance? It is significant that the infants in this study responded to the increased resistance by an immediate elevation in intrapleural pressure and calculated ventilatory work as shown on continuous tracings of ventilation and intra-esophageal pressure. There was almost certainly inadequate time for the respiratory center to respond to changes in arterial *p*H or carbon dioxide concentration. Campbell and Howell [13] have described the rapid restoration of the tidal volume following the addition of a mechanical load to ventilation. They explain this prompt reaction as being due to a reflex similar to the muscle or tendon stretch reflex. The sensing elements are the muscle spindles closely associated with voluntary muscle fibers throughout the body including the respiratory skeletal muscles. When there is inadequate muscle contraction due to resistance, the spindles sense the inadequacy and operate as a servo mechanism to drive the main muscle fibers to develop additional tension and quickly restore ventilation to normal.

This study shows that reflex compensation for increased airway resistance is well maintained in small infants during halothane anesthesia. Nunn [14] noted a similar compensatory ability for anesthetized adults and pointed out that under anesthesia, conditions are most favorable for tolerating a narrowed airway, since the fear and anxiety that lead to increased oxygen consumption are eliminated. What must be stressed from the results of this study is that carbon dioxide retention and respiratory acidosis are relatively late sequellae of increased airway resistance. They are seen only after varying periods of ventilatory compensation brought about by strenuous muscular effort and pulmonary work. As late sequellae, they indicate muscular fatigue and respiratory decompensation.

## Summary

The respiratory response of anesthetized infants to acute increases in airway resistance was studied. Ventilatory rate decreased, tidal volume increased, airflow rate decreased, and minute volume decreased. Reflex compensation for increased airway resistance was well maintained. By an increase of over two hundred per cent in their work of breathing, adequate alveolar ventilation was carried out as indicated by the absence of carbon dioxide retention and acidosis. Of interest was the finding that pulmonary compliance during the recovery period was generally greater than found during the control period. It is stressed that carbon dioxide retention and acidosis are not early signs of abnormal airway resistance, but late signs that indicate fatigue and respiratory decompensation.

## References

1. Krieger, I.: Studies on mechanics of respiration in infancy, Amer. J. Dis. Child. **105**: 439, 1963.
2. Swyer, P. R., Reiman, R. C., and Wright, J. J.: Ventilation and ventilatory mechanics in the newborn, J. Pediat. **56**: 612, 1960.
3. McIlroy, M. B., and Tomlinson, E. S.: The mechanics of breathing in newly born babies, Thorax **10**: 58, 1955.
4. Astrup, P.: A new approach to acid-base metabolism, Clin. Chem. **7**: 1, 1961.
5. Davies, H. W., Haldane, J. S., and Priestley, J. G.: The response to respiratory resistance, J. Physiol. **60**: 43, 1919.
6. Killick, E. M.: Resistance to inspiration—its effect on respiration in man, J. Physiol. **84**: 162, 1935.
7. Cain, C. C., and Otis, A. B.: Some physiologic effects resulting from added resistance to respiration, J. Aviat. Med. **20**: 149, 1949.

8. McIlroy, M. B., Eldridge, F. L., Thomas, J. P. and Christie, R. V.: Effect of added elastic and non-elastic resistance on pattern of breathing in normal subjects, Clin. Sci. **15**: 337, 1956.
9. Cook, C. D., Sutherland, J. M., Segal, S., Cherry R. B., Mead, J., McIlroy, M. B., and Smith, C. A.: Studies of respiratory physiology in the newborn infant, J. Clin. Invest. **36**: 440, 1957.
10. Mead, J.: Control of respiratory frequency, J. Appl. Physiol. **15**: 325, 1960.
11. Otis, A. B., Fenn, W. O., and Rahn, H.: Mechanics of breathing in man, J. Appl. Physiol. **2**: 592, 1950.
12. Whittenberger, J. L.: Lung volume and air flow characteristics in asthma. *In:* Abramson, H. A.: Somatic and Psychiatric Treatment of Asthma. Baltimore, Williams & Wilkins Co., 1951, pp. 50–61.
13. Campbell, E. J. M., and Howell, J. B. L., as quoted by Comroe, J. H.: Physiology of Respiration. Chicago, Year Book Medical Publishers, 1965, p. 88.
14. Nunn, J. F., and Ezi-Ashi, T. I.: The respiratory effects of resistance to breathing in anesthetized man, ANESTHESIOLOGY, **22**: 174, 1961.

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**JEHOVAH'S WITNESSES** Recent court decisions have clarified what is possible and not possible in cases involving the refusal of members of the Jehovah's Witness sect to accept blood for transfusion purposes, even in instances where such blood would be life saving. Rulings in New York and Illinois have upheld the right of adults to refuse a blood transfusion "even though we may consider the patient's beliefs unwise, foolish, or ridiculous." On the other hand courts have frequently ordered blood transfusion or medical services for minors even though parents have objected. These latter court orders have been supported when brought to higher courts on appeal. (*News and Announcements Column: Blood Transfusions and Jehovah's Witnesses, Transfusion 5: 392 (July) 1965.*)

**LOCAL ANESTHESIA** Successive administration of anesthetics yielded a deeper and longer lasting effect than a single injection. Successive injections of dicaine (amethocaine) and sovocaine (cinchocaine) into a novocaine infiltrate provided greater prolongation of anesthesia than other combinations. The mechanism of action of successive anesthesia is based on the synergistic action of novocaine, sovocaine and dicaine, and in the extended action of the latter two agents. (*Muratov, S. N.: Administration of Successive Local Anesthetics in Bone and Soft Tissue Injuries (Russian), Kemerov Ortop. Travm. Protez 26: 37, 1965.*)

**LOCAL HEMOSTASIS** A new synthetic posthypophysial hormone-like substance, phenyllysyl-vasopressin (PLV-2) was used for topical and local vasoconstriction. Applied topically to the nasal mucosa it was as effective as cocaine 4 per cent in providing nasal decongestion prior to nasotracheal intubation. PLV-2 is considered to be a vasoconstrictor that may be used with greater safety than can be obtained with epinephrine or controlled hypotension. (*Green, H. D., and Blumberg, J. B.: Use of a Synthetic Analogue of Posthypophysial Vasopressin (PVL-2) for Local Hemostasis, Surgery 58: 524 (Sept.) 1965.*)