Anesthesia and Atelectasis

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The effects of pentobarbital, halothane and methoxyflurane anesthesia on FRC, lung compliance and A-ADO₂ were determined in dogs during unassisted spontaneous respiration. Similar measurements were made in patients free of cardiopulmonary disease, immediately before extremity surgery, during surgery under halothane anesthesia and again postoperatively. No significant change in FRC, lung compliance or A-ADO₂ occurred throughout the anesthetic experience in the dog or in man. We conclude that progressive atelectasis is not a predictable consequence of unassisted respiration during anesthesia in the healthy dog and in man.

INCREASED PHYSIOLOGIC SHUNTING during anesthesia and spontaneous respiration has been reported and is assumed to represent atelectasis. Reports of gradual reductions in lung compliance or increases in shunting during anesthesia also suggest that progressive atelectasis may occur unless respiration is assisted or supplemented with periodic passive hyperinflations. The present study, designed to test these concepts, approaches the assessment of atelectasis by measuring the resting state of lung inflation (FRC) and its relation to changes in compliance and physiologic shunting during anesthesia.

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Methods

EFFECTS OF ANESTHETICS ON COMPLIANCE, FRC AND A-ADO₂ IN DOGS

Unpremedicated dogs weighing 16 to 31 kg., placed in the supine position, underwent induction of anesthesia with either 20 mg./kg. intravenous pentobarbital or open-drop halothane or methoxyflurane. The forelegs were loosely restrained in the flexed position. Five dogs received all three anesthetics, with intervals of at least a week between them. The mean weight of dogs receiving pentobarbital was 19.8 kg.; halothane, 21.1 kg.; methoxyflurane, 18.0 kg. The tracheas of all dogs were intubated with a #38 endotracheal tube which then was attached to a T piece. Oxygen was admitted at the side port at a flow rate in excess of twice the minute volume. The expiratory limb of the T piece was connected to a length of corrugated tubing to prevent contamination of inspired gas with room air. An esophageal balloon was placed in the lower third of the esophagus, filled with 0.5 ml. air, and the pressure balanced against airway pressure to record transpulmonary pressure. Air flow was recorded from a Fleisch #1 pneumotachometer and tidal volume obtained from electrical integration of the flow signal. Following induction, enough additional anesthesia was given to just suppress spontaneous limb movements. This required an occasional 100 mg. of pentobarbital and maintenance of inspired halothane at 0.5-1.0 per cent or methoxyflurane at approximately 0.5 per cent utilizing a Fluotec or Pentec vaporizer. Within 30 minutes following induction, dynamic lung compliance (C₅₅), functional residual capacity (FRC), respiratory rate (FR), tidal volume (Vₜ), inspired and end-expired oxygen tension, and arterial pH, P CO₂ and P O₂ were measured. Anesthesia was

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then deepened and maintained at that level for an hour; inspired halothane concentration was 1.8 per cent; methoxyflurane, 1.5 per cent. After the measurements were repeated, no further anesthetic was given. When spontaneous limb movements occurred the measurements were made a third time, one to two hours following termination of anesthesia. Blood for gas analyses was drawn from the femoral artery in heparinized glass syringes. Analyses of pH, $P_{CO_2}$ and $P_{O_2}$ were performed immediately, or the sample was iced and analyzed later. A Beckman 160 Physiological Gas Analyzer was employed, utilizing a Severinghaus $CO_2$ electrode and a modified Clark electrode for $O_2$. Inspired and end-expired oxygen were measured with a Beckman C-2 or 160 gas analyzer.

FRC was measured by the closed-circuit helium-dilution technique, employing corrections for changes in oxygen and halothane concentrations.$^5$ Methoxyflurane and halothane were found to reduce the galvanometer deflection resulting from the helium. The presence of 1 per cent methoxyflurane led to underestimation of the helium concentration by 0.63 per cent. This relationship was found to be linear over a range of 0–1.5 per cent. Gas was collected with a Hamilton syringe at the time of helium meter readings and analyzed for methoxyflurane employing a Microtex GC-2500R gas chromatograph. Corrections of helium meter readings for methoxyflurane were made employing standard formulas.$^5$

Dynamic lung compliance was expressed as the ratio of inspired volume to the difference between transpulmonary pressure at the beginning of inspiration and the moment of maximum inspiration (no flow), and expressed as liters per cm. water.

Alveolar $O_2$ tension was determined from the following formula:

$$P_{A_{O_2}} = P_{I_{O_2}} - P_{A_{CO_2}} \quad (1)$$

based on the assumption that $P_{A_{CO_2}} = P_{A_{CO_2}}$. In addition, it is assumed that alveolar and arterial tensions of either halothane or methoxyflurane were equal at the time of $P_{A_{O_2}}$ determinations. If the alveolar–blood anesthetic concentrations were to differ by 0.5 per cent, the 3.5 mm. Hg gradient would have negligible influence on the calculation of total physiologic shunt.

During $O_2$ inhalation, the difference between observed arterial oxygen tension and that predicted from assuming equilibration of pulmonary venous blood at the alveolar oxygen tension is due to a reduction in the amount of oxygen physically dissolved in whole blood. This difference between observed and predicted arterial tensions is due to the physiologic shunt.

The total physiologic shunt is defined as that portion of the cardiac output ($Q_t$) which collectively perfuses collapsed lung, perfuses relatively over-perfused areas, and passes through anatomical shunts. Anatomical shunting probably accounts for no more than 1–2 per cent of the total shunt. In addition, a very small portion of the total shunt may be mixtures inhaled. The quantity of shunted due to a diffusion gradient, should hypoxic blood ($Q_s$) during $O_2$ inhalation is determined from the formula:

$$Q_s (\text{vol. %}) = (P_{A_{O_2}} - P_{A_{O_2}}) 0.0031 \quad (2)$$

where 0.0031 represents the Bunsen solubility coefficient of oxygen in blood.

An estimate of the total cardiac output ($Q_t$) shunted is made from the equation:

$$\frac{Q_s}{Q_t} \times 100 = \frac{(P_{A_{O_2}} - P_{A_{O_2}}) 0.0031}{(P_{A_{O_2}} - P_{A_{O_2}}) 0.0031 + C_{O_2} - C_{V_{O_2}}} \quad (3)$$

when $C_{O_2} - C_{V_{O_2}}$ represents the arterial–mixed venous oxygen content difference. This difference was assumed to be 4.5 volumes per cent as measured in dogs lightly anesthetized with pentobarbital,$^6$ breathing spontaneously, and in man at rest.$^7$

**Effects of Halothane Anesthesia on Respiratory Mechanics and A–ADO$_2$ in Man**

Eight patients free of cardiopulmonary disease and premedicated with 100 mg. pentobarbital and 0.6 mg. atropine were studied immediately before surgery done on the periphery. Preoperative measurements the same as those done in experiments on dogs were made at FRC, $C_l$, pH, $P_{CO_2}$, $P_{O_2}$, $V_t$, FR, $P_{O_2}$,
TABLE 1. Effects of Pentobarbital, Halothane, and Methoxyflurane on Respiratory Mechanics and Blood Gases in Dogs*

<table>
<thead>
<tr>
<th>Agent</th>
<th>30 min. after induction of light anesthesia</th>
<th>After 60 minutes of deep anesthesia</th>
<th>During emergence from anesthesia</th>
</tr>
</thead>
<tbody>
<tr>
<td>Vt (ml.)</td>
<td>Pentobarbital 255 ± 50, Halothane 180 ± 48, Methoxyflurane 192 ± 80</td>
<td>250 ± 61, 125 ± 38, 169 ± 77</td>
<td>238 ± 53, 151 ± 46, 180 ± 72</td>
</tr>
<tr>
<td>FR (min.)</td>
<td>Pentobarbital 8 ± 3, Halothane 21 ± 14, Methoxyflurane 12 ± 7</td>
<td>7 ± 3, 21 ± 10, 10 ± 5</td>
<td>7 ± 2, 25 ± 15, 11 ± 4</td>
</tr>
<tr>
<td>FRC (ml.)</td>
<td>Pentobarbital 490 ± 100, Halothane 782 ± 273, Methoxyflurane 903 ± 375</td>
<td>483 ± 157, 874 ± 313, 736 ± 129</td>
<td>452 ± 180, 734 ± 261, 700 ± 237</td>
</tr>
<tr>
<td>Cl (l./cm. H2O)</td>
<td>Pentobarbital 0.063 ± 0.02, Halothane 0.079 ± 0.03, Methoxyflurane 0.060 ± 0.01</td>
<td>0.056 ± 0.01, 0.062 ± 0.05, 0.062 ± 0.02</td>
<td>0.055 ± 0.01, 0.075 ± 0.03, 0.067 ± 0.03</td>
</tr>
<tr>
<td>Po2 (mm. Hg)</td>
<td>Pentobarbital 461 ± 102, Halothane 485 ± 65, Methoxyflurane 504 ± 48</td>
<td>471 ± 108, 450 ± 135, 539 ± 56</td>
<td>499 ± 66, 549 ± 86, 530 ± 68</td>
</tr>
<tr>
<td>Paco2 (mm. Hg)</td>
<td>Pentobarbital 40 ± 7, Halothane 33 ± 5, Methoxyflurane 42 ± 6</td>
<td>55 ± 4, 49 ± 8, 55 ± 8</td>
<td>51 ± 5, 35 ± 6, 41 ± 7</td>
</tr>
<tr>
<td>pH</td>
<td>Pentobarbital 7.35 ± 0.08, Halothane 7.37 ± 0.07, Methoxyflurane 7.31 ± 0.07</td>
<td>7.28 ± 0.07, 7.24 ± 0.08, 7.25 ± 0.05</td>
<td>7.31 ± 0.04, 7.35 ± 0.05, 7.34 ± 0.04</td>
</tr>
<tr>
<td>A-aDO2 (mm. Hg)</td>
<td>Pentobarbital 190 ± 104, Halothane 163 ± 62, Methoxyflurane 151 ± 48</td>
<td>171 ± 102, 129 ± 74, 102 ± 52</td>
<td>146 ± 68, 105 ± 81, 125 ± 65</td>
</tr>
<tr>
<td>Qs/Qt (per cent)</td>
<td>Pentobarbital 11.4 ± 4.2, Halothane 10.9 ± 3.3, Methoxyflurane 9.3 ± 2.2</td>
<td>10.3 ± 4.7, 8.0 ± 4.1, 6.5 ± 3.0</td>
<td>9.0 ± 3.8, 6.5 ± 4.5, 8.7 ± 2.0</td>
</tr>
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</table>

* Mean values ± S.D. (n = 7).

P<sub>EO</sub>2, with the patient supine on the operating table, and after 15 minutes of denitrogenation utilizing a Sierra nonrebreathing valve and face mask. These measurements were repeated approximately an hour after the start of operation, using the same valve and face mask while breathing oxygen and 2 per cent halothane and again in the recovery room following emergence from anesthesia (table 2).

Results

EFFECTS OF ANESTHETICS ON COMPLIANCE, FRC, AND A-aDO<sub>2</sub> IN DOGS

Deep anesthesia sufficient to induce hypventilation and respiratory acidosis had virtually no effect on FRC, Cl, or A-aDO<sub>2</sub>, regardless of anesthetic used (table 1). When anesthetized with pentobarbital, however, dogs had a persistently lower FRC than when inhaling halothane or methoxyflurane, yet this difference appeared to have little influence on the A-aDO<sub>2</sub>.

EFFECTS OF HALOTHANE ANESTHESIA ON RESPIRATORY MECHANICS AND SHUNTING IN MAN

Lightly medicated patients lying supine had appreciable A-aDO<sub>2</sub> values immediately prior to induction of anesthesia (table 2). Lung volumes and compliance, however, were
within normal limits. No significant change in the A-aDO₂ or in the estimated shunt occurred during or following anesthesia (and surgery) in spite of marked alveolar hypventilation and respiratory acidosis.

Discussion

No fully reliable or direct method for the detection of atelectasis is known. Since atelectasis cannot be observed directly except during thoracic surgery, recourse has been made to indirect methods of detection. Clinical examination and X-ray are helpful in localized or gross atelectasis, but are of little help during the early stages of development or diffuse collapse.⁶ ⁷ ⁹ ¹⁰ A frequently-used method of assessment is measurement of the alveolararterial O₂ difference during inhalation of oxygen.¹¹ ¹₂ ¹₃ Normal a gradient of less than 25 mm. Hg exists in man while awake and supine, probably because of blood flowing through anatomical shunts.¹⁴ A gradient in excess of this suggests atelectasis. A gradient of less than 100 mm. Hg exists in supine dogs anesthetized with pentobarbital.⁶ In the present study supine dogs while lightly anesthetized had gradients between 150 and 190 mm. Hg, or an estimated shunt of approximately 10 per cent. This gradient, although it suggested a slight amount of atelectasis, did not increase when anesthesia was deepened with pentobarbital, halothane or methoxyflurane, indicating that spontaneous unassisted respiration during anesthesia does not necessarily lead to progressive atelectasis. Spontaneous deep breaths, noted to occur during administration of all three anesthetics, may have had some influence in preventing an increase in the A-aDO₂.

The results of the present study of patients free of cardiopulmonary disease indicate that the administration of halothane anesthesia with unassisted respiration does not lead to a significant increase in the A-aDO₂, and confirm the findings of Marshall.¹³ Other studies ¹¹ ¹₂ have reported significant shunting during halothane anesthesia, but did not include measurements immediately prior to anesthesia (table 3). Pentobarbital and atropine premedication in the doses employed in the present study have been shown not to alter blood gas tensions.¹⁵

While the stability of the A-aDO₂ throughout the anesthetic experiences in both the dog and man strongly suggests that anesthesia does not lead to progressive atelectasis, changes in cardiac output, were they to occur, could affect the calculated shunt. If the cardiac output were to decrease 25 per cent during anesthesia, a shunt of 10 per cent would be overestimated by about 3 per cent. The error of estimation would be larger in the presence of larger shunts. Reductions in cardiac output of less than 15 per cent are known to occur in man during halothane anesthesia and spontaneous respiration, however.¹⁶ The error in estimating shunt introduced by such a change is well within the limits of observed variance in shunt, and can be ignored.

Changes in A-aDO₂ must be interpreted with caution in situations other than a reasonably steady state. Changes in A-aDO₂ may represent changes in atelectasis if continued perfusion of atelestatic areas of the lung occur. There is some evidence that the distribution of blood flow is gradually shifted away from affected areas of the lung,¹⁷ leading to a reduction in shunt. Moreover, this redistribution of

<table>
<thead>
<tr>
<th>Reference</th>
<th>Before</th>
<th>During</th>
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<tbody>
<tr>
<td>Bergman²</td>
<td>—</td>
<td>196 ± 84</td>
</tr>
<tr>
<td>Marshall¹¹</td>
<td>142 ± 100</td>
<td>154 ± 127</td>
</tr>
<tr>
<td>Nunn¹</td>
<td>—</td>
<td>184 ± 73</td>
</tr>
<tr>
<td>Colgan and Whang</td>
<td>164 ± 57</td>
<td>196 ± 93</td>
</tr>
</tbody>
</table>

**Table 2.** Respiratory and Blood Gas Changes with Halothane in Oxygen Anesthesia*

<table>
<thead>
<tr>
<th></th>
<th>Preoperative</th>
<th>During Surgery</th>
<th>Postoperative</th>
</tr>
</thead>
<tbody>
<tr>
<td>V₀ (ml.)</td>
<td>579 ± 315</td>
<td>250 ± 100</td>
<td>470 ± 227</td>
</tr>
<tr>
<td>FR (min.)</td>
<td>13 ± 4</td>
<td>25 ± 7</td>
<td>16 ± 6</td>
</tr>
<tr>
<td>PICO₃ (L/cm. H₂O)</td>
<td>2.96 ± 0.55</td>
<td>2.13 ± 0.73</td>
<td>2.57 ± 0.87</td>
</tr>
<tr>
<td>PO₂ (mm. Hg)</td>
<td>414 ± 106</td>
<td>438 ± 82</td>
<td>435 ± 116</td>
</tr>
<tr>
<td>PCO₂ (mm. Hg)</td>
<td>25.2 ± 4</td>
<td>24.7 ± 6</td>
<td>28.6 ± 6</td>
</tr>
<tr>
<td>pH</td>
<td>7.44 ± 0.04</td>
<td>7.33 ± 0.07</td>
<td>7.40 ± 0.05</td>
</tr>
<tr>
<td>A-aDO₂ (mm. Hg)</td>
<td>164 ± 57</td>
<td>156 ± 53</td>
<td>163 ± 53</td>
</tr>
<tr>
<td>Q₀/Q₁ X 100</td>
<td>8.8 ± 2.9</td>
<td>10.5 ± 5.1</td>
<td>8.3 ± 4.3</td>
</tr>
</tbody>
</table>

* Mean values ± S.D. (n = 8).

**Table 3.** A-aDO₂ Immediately before and during Halothane Anesthesia

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Footnotes and references are not included in the natural text representation.
blood is greater the higher the concentration of inspired oxygen.\textsuperscript{18} Distribution of pulmonary blood flow and shunting also are influenced by changes in position, cardiac output,\textsuperscript{19} peak inspiratory inflation pressures,\textsuperscript{20} and hyperinflation.\textsuperscript{21} The importance of the pattern of ventilation in influencing shunting during artificial ventilation has been stressed by Bendixen and Hedley-Whyte.\textsuperscript{2,13} During spontaneous respiration, shunting can be reduced during hyperventilation induced by breathing hypoxic mixtures.\textsuperscript{22,23} Thus, interpreting changes in physiologic shunting during changing clinical conditions may be misleading in assessment of atelectasis.

It has long been appreciated that a decrease in resting lung volume (FRC) can be used as a measure for assessing atelectasis.\textsuperscript{9} It is significant that the FRC remained remarkably stable during and following anesthesia in man, in spite of a 50 per cent reduction in tidal volume and a doubling of the respiratory rate during surgery. Nor did a significant change in FRC occur during the course of anesthesia in dogs. However, the FRC in dogs receiving pentobarbital was noticeably lower than in those receiving halothane or methoxyflurane. Since the respiratory rate was much lower during pentobarbital anesthesia, presumably the lungs contracted to a smaller resting lung volume during the longer expiratory pause between breaths. The low FRC, however, had minimal effects on A-aDO\textsubscript{2} and estimated shunt (table 1).

Lung compliance did not change significantly during or following anesthesia in the dog or man. Recent evidence indicates that changes in lung compliance relate to changes in underlying atelectasis more faithfully than changes in either FRC or A-aDO\textsubscript{2} during oxygen inhalation and spontaneous respiration.\textsuperscript{24} Lung compliance, measured over the tidal range, varies little, but with complete collapse of alveoli strong surface forces oppose inflation. Completely atelectatic portions of the lung are thus effectively inactive when volume-pressure measurements are made; the resulting reduction in compliance reflects the amount of collapsed lung tissue. Factors other than the amount of atelectasis, however, affect lung compliance: respiratory rate, mechanical ventilation, and autonomic tone.\textsuperscript{25,29} Interpretation of changes in lung compliance or A-aDO\textsubscript{2} as representing changes in underlying atelectasis should be made with due regard to all factors besides atelectasis known to influence them.

Conclusions

The effects of anesthesia on resting lung volume (FRC), lung compliance, and A-aDO\textsubscript{2} were determined in the dog and in man during spontaneous breathing. Evidence is presented that the anesthetic state does not alter FRC, lung compliance, A-aDO\textsubscript{2} or estimated physiologic shunt; and that progressive atelectasis \textsuperscript{7} is not a predictable consequence of unassisted ventilation during anesthesia in healthy dogs and in man.

Grateful acknowledgment is made to Miss Catherine P. Vangeloff for technical assistance.

References


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Drugs

LOCAL ANESTHETICS The effects of four local anesthetics, procaine, lidocaine, tetracaine and dibucaine, on the soleus neuromuscular preparation of the cat in vivo were studied. All anesthetic agents, when injected intra-arterially or intravenously, depressed the posttetanic potentiation of the soleus muscle and abolished the neural repetitive afterdischarge of the motor nerve terminals. Lidocaine was 1.5, tetracaine 10, and dibucaine 15 times more potent than procaine in depressing posttetanic potentiation. Recovery of posttetanic potentiation was rapid following procaine and lidocaine, but it was prolonged after tetracaine and dibucaine. Since posttetanic potentiation and posttetanic repetitive activity are neural events and local anesthetics depressed them without depressing the transmission of single twitches, it was concluded that local anesthetics act by selective depression of the motor nerve terminal. The possibility of a postjunctional effect of local anesthetics is considered, but, according to the dose—response relationship, it occurs only after injection of large doses. (Usubiaga, J. E., and Standaert, F.: The Effects of Local Anesthetics on Motor Nerve Terminals, J. Pharmacol. Exper. Therap. 159: 353 (Feb.) 1968.)