Blood-pressure and Blood-Gas Changes during Anesthesia for Bronchoscopy Using a Modified Method of Ventilation


In our hospital, diagnostic bronchoscopy has usually been performed using general anesthesia with muscle relaxants and intermittent positive-pressure ventilation via a small-bore orotracheal tube. Although ventilation was considered satisfactory, disturbing features were the almost constant occurrence of elevated blood pressure and the frequent occurrence of cardiac arrhythmias. These were particularly alarming in the elderly and in patients with pre-existing hypertension and cardiovascular disease. The purpose of this study was to compare the arterial blood-gas changes and cardiovascular responses during general anesthesia for bronchoscopy using two methods of ventilation: a) ventilation via an endotracheal tube placed alongside a non-ventilating bronchoscope; b) ventilation via both endotracheal tube and ventilating bronchoscope simultaneously.

PATIENTS AND METHODS

Twenty unselected adult patients who underwent diagnostic bronchoscopy under general anesthesia were studied. The first ten (Group I) were ventilated via an orotracheal tube and the next ten (Group II), via both orotracheal tube and ventilating bronchoscope.

The primary disease in every patient was tumor of the lung, and secondary pulmonary disease when present, was in the nature of chronic bronchitis, emphysema and atelectasis. All patients were anesthetized by the same anesthesiologist (S. H.). The same drugs for premedication (meperidine and promethazine hydrochloride) were given to all patients 45 minutes preoperatively. Anesthesia was induced with methohexitol sodium, followed by succinylcholine to facilitate intubation with a Portex endotracheal tube, 4.5–5.5 mm ID. Anesthesia was maintained with 15–20 l/min of 50 per cent nitrous oxide–oxygen with 0.5–1.0 per cent halothane. Muscle relaxation was maintained with intermittent injections of succinylcholine (total dose 100–300 mg iv) and intermittent positive-pressure ventilation with a 500-ml tidal volume, using a Ruben non-rebreathing valve.

In Group I, a Storz bronchoscope (ID 8–9 mm) was introduced alongside the endotracheal tube, the patients being ventilated through the latter. In Group II, a Foregger-
Table 1. Mean Arterial Values before, during, and after General Anesthesia for Bronchoscopy

<table>
<thead>
<tr>
<th></th>
<th>$P_{aCO_2}$ (mm Hg)</th>
<th>$P_{aO_2}$ (mm Hg)</th>
<th>$P_{aCO_2}$ (mm Hg)</th>
<th>$P_{aO_2}$ (mm Hg)</th>
<th>pH</th>
<th>$P_{aO_2}$ (mm Hg)</th>
<th>$P_{aCO_2}$ (mm Hg)</th>
<th>pH</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Group I</td>
<td>Group II</td>
<td>Group I</td>
<td>Group II</td>
<td>Group I</td>
<td>Group II</td>
<td>Group I</td>
<td>Group II</td>
</tr>
<tr>
<td>Before anesthesia</td>
<td></td>
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<td></td>
<td></td>
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<td></td>
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<tr>
<td>(after premedication)</td>
<td>81.3</td>
<td>82.3</td>
<td>37.4</td>
<td>37.8</td>
<td>7.41</td>
<td>7.42</td>
<td></td>
<td></td>
</tr>
<tr>
<td>During anesthesia</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
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</tr>
<tr>
<td>6-10 min</td>
<td>169.9*</td>
<td>290.2*</td>
<td>51.2</td>
<td>37.1</td>
<td>7.28</td>
<td>7.40</td>
<td></td>
<td></td>
</tr>
<tr>
<td>16-20 min</td>
<td>152.6*</td>
<td>285.6*</td>
<td>55.4</td>
<td>36.0</td>
<td>7.25</td>
<td>7.41</td>
<td></td>
<td></td>
</tr>
<tr>
<td>26-30 min</td>
<td>143.0*</td>
<td>222.3*</td>
<td>53.1</td>
<td>32.2</td>
<td>7.25</td>
<td>7.43</td>
<td></td>
<td></td>
</tr>
<tr>
<td>After extubation, 10 min</td>
<td>83.4</td>
<td>89.1</td>
<td>35.2</td>
<td>33.6</td>
<td>7.35</td>
<td>7.38</td>
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</table>

* Patients ventilated with a 50 per cent $N_2O-O_2$ mixture.

Table 2. Mean Changes in Systolic Blood Pressure before, during, and after General Anesthesia for Bronchoscopy

<table>
<thead>
<tr>
<th></th>
<th>Before Anesthesia (after Premedication)</th>
<th>During Anesthesia</th>
<th>After Extubation 10 min</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>6-10 min</td>
<td>16-20 min</td>
<td>26-30 min</td>
</tr>
<tr>
<td>Group I</td>
<td>146</td>
<td>175</td>
<td>191</td>
</tr>
<tr>
<td>Mean (mm Hg)</td>
<td>100</td>
<td>120.5</td>
<td>130.8</td>
</tr>
<tr>
<td>Per cent of mean</td>
<td>100</td>
<td>103</td>
<td>90</td>
</tr>
</tbody>
</table>

Safar ventilating bronchoscope (ID 8–9 mm) was similarly introduced. In the latter group, however, ventilation was modified by joining the sidearm of the ventilating bronchoscope to the proximal end of the endotracheal tube by means of a Y connection (fig. 1). This enabled us to ventilate both lungs simultaneously during most of the procedure. When the window of the ventilating bronchoscope was opened for the purpose of biopsy, suction, or lavage, limb A, connected to the bronchoscope, was clamped, while ventilation continued uninterruptedly to at least one lung through the endotracheal tube.

ECG monitoring by oscillograph started immediately prior to induction of anesthesia and continued throughout the procedure, with recordings being made frequently. Blood pressure was determined by conventional sphygmomanometer cuff. Arterial blood samples were taken in sealed syringes via an indwelling needle in the brachial artery, cooled in ice, and promptly examined in duplicate by the Radiometer Blood Gas Analyzer for $P_{aO_2}$, $P_{aCO_2}$ and pH. Blood was drawn: 1) immediately prior to the start of anesthesia; 2) when the bronchoscope was resting in the trachea immediately above the carina; 3) at the end of the examination of the right main bronchus, with the bronchoscope still in this position; 4) at the end of the examination of the left main bronchus; 5) after extubation, after the patient had breathed air spontaneously for at least 10 minutes.

RESULTS

Arterial Blood-Gas Changes. The changes in mean arterial $P_{aO_2}$, $P_{aCO_2}$ and pH in the two groups are shown in Table 1. The mean $P_{aO_2}$ in both groups were satisfactory during anesthesia. The mean $P_{aCO_2}$ in Group I was elevated during anesthesia, ranging from 51.2 to 55.4 mm Hg, whereas in Group II the $P_{aCO_2}$ decreased to slightly below initial levels.

The mean changes in pH during anesthesia closely paralleled the $P_{aCO_2}$ changes in the two
groups, being markedly acidotic in Group I, while remaining normal in Group II.

Cardiovascular Changes. Mean changes in systolic blood pressure at the times of drawing blood for analysis are summarized in table 2. In Group I there were progressive increases in mean systolic blood pressures during anesthesia, increasing from an initial 146 to as high as 191 mm Hg. In contrast Group II showed minimal blood pressure changes. The relations between $P_a CO_2$ and systolic blood pressures in the two groups are shown in figure 2.

Various arrhythmias were found and recorded during the procedures with almost equal frequency in both groups; tachycardia, bradycardia, supraventricular extrasystoles, ventricular premature contractions, multifocal extrasystoles, and first-degree heart block. In most cases the arrhythmias and changes in heart rate were associated with manipulation of the bronchoscope or with suction.

DISCUSSION

Techniques of general anesthesia for bronchoscopy have not always provided consistently satisfactory conditions regarding ventilation.\textsuperscript{3,4} In two common methods ventilation is accomplished via a small-bore endotracheal tube,\textsuperscript{3} or via a ventilating bronchoscope.\textsuperscript{3,4} With the former, ventilation is often inadequate due to the small diameter of the endotracheal tube. With the latter, $CO_2$ accumulates when the eyepiece of the ventilating bronchoscope is removed for various periods. In the present study the disadvantages inherent in these two techniques were overcome by joining the proximal orifice of the endotracheal tube and the side vent of the ventilating bronchoscope by means of a Y piece (fig. 1). Continuous ventilation at all times in either both lungs simultaneously or at least in the one lung not being examined was thereby assured.

Most reports on general anesthesia in bronchoscopy have emphasized the problems of ventilation, gas exchange\textsuperscript{3-4} and arrhythmias.\textsuperscript{5,6} To the best of our knowledge, changes in blood pressure have not often been discussed, an interesting oversight in view of the well-known direct relationship between hypercarbia and hypertension.\textsuperscript{5} This relationship was strikingly demonstrated in the patients ventilated via a small-bore tube, in whom the increases in blood pressure closely paralleled the increases in $P_a CO_2$ (fig. 2). This finding is important in view of the high incidences of hypertensive and arteriosclerotic heart disease in patients referred for diagnostic bronchoscopy. Neither hypercarbia nor elevation of blood pressure above the control level occurred with our modified method of ventilation. The hypertension in our cases seems, therefore, to have been related to $CO_2$ accumulation.

There was no difference between the incidences of cardiac arrhythmias in the two groups, a surprising feature in view of the relationship of arrhythmias to elevated $P_a CO_2$.\textsuperscript{8,9} Arrhythmias during bronchoscopy may also be caused by mechanical stimulation of the sympathetic and parasympathetic nerve endings.\textsuperscript{10} This was the most likely cause of the arrhythmias we found with almost equal frequencies in both groups.
CLINICAL WORKSHOP

SUMMARY

Adequate alveolar ventilation including elimination of CO₂ is essential at all stages of the bronchoscopic procedure, especially when it is prolonged, since excessive increases in systolic blood pressure even at moderately elevated P₈CO₂’s may be a source of danger to the elderly or cardiac patient. With our method of ventilation during general anesthesia, involving simultaneous use of a ventilating bronchoscope and an endotracheal tube, we can assure adequate alveolar ventilation, and eliminate abrupt elevations in blood pressure.

REFERENCES


Drugs and Their Actions

SERUM DIGOXIN LEVELS Difficulty in maintaining the well-known narrow range between effective and toxic doses of digitalis has prompted the development of techniques for its assay in serum. Using a tritiated digoxin tracer and scintillation counter or a radioactive-iodine-labeled digoxin analog tracer and gamma-ray counter, reasonably prompt determinations are possible from small samples. Computer assistance in the calculations shortens the assay time to half an hour. Below a digoxin concentration of 2 nanograms/ml, 90 per cent of patients manifested no evidence of toxicity; at higher concentrations 87 per cent of the patients did show evidence of toxicity responsive to drug withdrawal. Interpretation of a serum level requires correlation principally with serum electrolytes and cardiopulmonary function. Radioimmunoassay methods also permit the determination of ouabain levels in plasma and urine. Intestinal absorption rates for digoxin peak in an hour during fasting but are blunted by a prior meal. In malabsorption syndromes absorption is erratic. During cardiopulmonary bypass there is a temporary decline in serum digoxin levels owing to mixing of the patient’s blood with the pump prime. An inverse relationship was found between plasma digoxin level and the ventricular rate during atrial fibrillation. Digoxin levels and resting heart rate did not correlate well in the presence of intrinsic disease of the conduction system. (Smith, T. W.: The Clinical Use of Serum Cardiac Glycoside Concentration Measurements, Am. Heart J. 82: 833–837, 1971.)