The Cost of Respiratory Effort in Postoperative Cardiac Patients

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The incidence of postoperative respiratory insufficiency following major thoracic or abdominal surgery is significant.\(^1\)\(^-\)\(^5\) We have previously emphasized the difficulties involved in the early recognition of respiratory insufficiency,\(^4\)\(^-\)\(^5\) and have pointed out that even a minimal degree will frequently produce a lowered arterial oxygen tension, with an associated serious arrhythmia and a decreased cardiac reserve, and result in a vicious cycle that ends in unnecessary death. Furthermore, assisted ventilation may be necessary to avoid respiratory acidosis, which would further impair myocardial function. Until we can determine the patient’s margin of safety, we advocate the prophylactic use of a volume-controlled respirator following open-heart surgery to tide the patient over the immediate and critical postoperative period. The respirator must have sufficient power to meet the patient’s needs, must be safe, and in no way productive of deleterious changes in the cardiovascular system or the parenchyma of the lung. The Engstrom volume-controlled respirator\(^6\) is the only one we have found that meets these criteria in the severely ill patient.

Routine postoperative use of the respirator with careful monitoring of hemodynamic and respiratory functions has emphasized an important aspect of postoperative physiology. The need for adequate oxygenation and carbon dioxide elimination is well understood. The quantity of the work load placed on the patient to maintain adequate respiratory function has not been sufficiently appreciated, despite the fact that Björk and Engstrom implicated respiratory work load as a major causative factor of respiratory insufficiency.\(^7\)\(^-\)\(^8\) The magnitude of this work load was first recognized by us when the clinical courses of patients who were carried on the respirator for a period of a few hours to several days were compared to other cardiac patients whose ventilation was not assisted. Those with respiratory assistance had a smooth recovery after anesthesia and required very little sedation, despite the continued use of an endotracheal tube. Exhaustion, restlessness, and apprehension were not present unless respiratory insufficiency was severe and intractable. The patients were alert and had an ample reserve of strength. Their appearance contrasted sharply with that of patients who were not assisted. This difference raised the possibility that the mere physical work of breathing contributed significantly to postoperative morbidity, to delayed congestive failure, and to a progressive respiratory insufficiency secondary to exhaustion. Therefore, a study was undertaken to evaluate the work and thus the energy cost of breathing in the postoperative patient.

The usual methods of quantitating the work of breathing as described by Bartlett, McIlroy, Cournand, and others\(^9\)-\(^11\) cannot be applied to the semiconscious patient after thoracic surgery, or to the patient who, although awake and cooperative, cannot increase respiratory movement beyond a limited degree because of pain. Another approach was thus necessary. When ventilation is mechanically taken over, the energy cost of breathing is assumed by the respirator. The difference in oxygen consumption, while on
and off the respirator, should indicate the amount of oxygen required by the respiratory muscles for spontaneous ventilation. Cardiac output determinations with and without the respirator with use of the Fick or dye-dilution methods, would thus reflect the percentage of cardiac output expended by the respiratory muscles, provided other physiologic factors are stable. It is possible, however, that changes in oxygen consumption and cardiac output with and without the respirator may represent potential harmful effects of the respirator itself. There is a definite need for supplemental data to elucidate this possibility. Other methods of evaluating the work of breathing are desirable to confirm current data. Recently a preliminary report by Engstrom et al. described such a method, and their data confirm the validity of our approach.13

Methods

Oxygen consumption studies were carried out in a series of patients immediately after major cardiac surgery. Following the operative procedure, the endotracheal tube was left in position and the patient, while still under light anesthesia or sedation, was placed on the volume-controlled Engstrom respirator. This respirator, with a carbon dioxide absorber in the circuit, can be used as a closed re-breathing system and spirometric determination of oxygen consumption carried out. We have employed both a standard spirometer and one especially designed, by one of us, for use with the Engstrom respirator. The latter permits use while the patient is still receiving nitrous oxide. After arrival in the recovery room, serial determinations of oxygen consumption with and without the respirator were initiated. If positioning of a pulmonary artery cannula was possible, oxygen content measurements were obtained simultaneously from the pulmonary artery and aorta during determinations of oxygen consumption and, from these data, changes in cardiac output with or without the respirator were calculated. In some patients, changes in cardiac output were also determined by the dye-dilution method. Further qualitative data were also obtained, including serial determinations of temperature, pulse rate, central aortic, pulmonary artery or right ventricular, central venous, and in one instance, left atrial pressures.

Results

Oxygen consumption studies on 12 patients

A bar graph to indicate the per cent change from predicted normal oxygen consumption in 12 patients studied while breathing spontaneously and while assisted by the respirator and while breathed for by the respirator. Note the increase in oxygen consumption when breathing spontaneously.

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are summarized in figure 1. The three periods of study corresponded to (1) the time immediately after arrival in the recovery room and while still under anesthesia, (2) the period during which the patient was waking, actively moving or shivering, and (3) a later time after the patient had become stabilized and was quiet. Oxygen consumption in six of the 12 patients was increased more than 10 per cent above predicted levels, even while on the respirator. In only four instances were normal values obtained. Furthermore, oxygen consumption increased still further each time the patient was permitted to breathe spontaneously.

The marked increase observed when the patient was moving and waking can only be ascribed in part to the work of breathing, since the level of consciousness and the degree of movement were continuously changing. The increase during the first and third periods, however, when the patient was either asleep or awake but quiet and cooperative, approximates the oxygen expenditure for the act of breathing. Increases during these two periods ranged from 2 to 29 per cent, the average being 18 per cent, which contrasts strikingly to the values of 1.0 to 2.0 per cent that, in the normal human, have been calculated as the percentage of energy expended for the work of breathing.9

The six patients with the lowest increase (L.B., R.H., D.C., D.B., H.E., W.F.) were with one exception (H.D.) young patients with good myocardial function and without a history of chronic lung disease. In these, the period of respiratory assistance was short, and after coming off the respirator all were able to maintain normal arterial oxygen and carbon dioxide tensions without undue effort. In the remaining six patients respiratory assistance was necessary for a longer period of

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In four of these six (E.S., G.A., W.C., E.H.), congestive failure had been a prominent preoperative finding. In another patient (B.C.), operated upon for mitral insufficiency, breathing 100 per cent oxygen after removal of respirator, the last patient (B.C.) had an episode of severe left atrial hypertension during surgery.

Figure 2 depicts the course of a 19-year-old P.G. (E.H.), in whom extracorporeal circulation was not used, pulmonary hypertension was marked and pulmonary compliance was poor.

Table 1

Physiologic Data in Patient B.C. with Mitral Insufficiency, Breathing 100 Per Cent Oxygen after Removal of Respirator

<table>
<thead>
<tr>
<th>Time P.O.</th>
<th>O2 con*</th>
<th>A, O2</th>
<th>V, O2</th>
<th>A, CO2</th>
<th>V, CO2</th>
<th>T*</th>
<th>CO</th>
<th>Pulse</th>
<th>BP</th>
<th>PAP</th>
<th>CVP</th>
<th>R rate</th>
<th>% Iner.†</th>
<th>Remarks</th>
</tr>
</thead>
<tbody>
<tr>
<td>Second hour</td>
<td>On</td>
<td>265</td>
<td>175</td>
<td>26</td>
<td>37.5</td>
<td>36.0</td>
<td>4.4</td>
<td>85</td>
<td>108/64</td>
<td>37/15</td>
<td>160</td>
<td>20</td>
<td>11</td>
<td>Asleep mild shivering</td>
</tr>
<tr>
<td></td>
<td>Off</td>
<td>293</td>
<td>105</td>
<td>28</td>
<td>44</td>
<td>4.8</td>
<td>94</td>
<td>106/68</td>
<td>52/27</td>
<td>230</td>
<td>48</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Twelfth hour</td>
<td>On</td>
<td>301</td>
<td>300</td>
<td>38</td>
<td>46</td>
<td>58</td>
<td>38.8</td>
<td>96</td>
<td>106/70</td>
<td>37/20</td>
<td>175</td>
<td>20</td>
<td>59</td>
<td>Awake very apprehensive</td>
</tr>
<tr>
<td></td>
<td>Off</td>
<td>485</td>
<td>110</td>
<td>39</td>
<td>58</td>
<td>80</td>
<td>100</td>
<td>130/80</td>
<td>55/32</td>
<td>225</td>
<td>39</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Twenty-second hour</td>
<td>On</td>
<td>254</td>
<td>260</td>
<td>31</td>
<td>42</td>
<td>80</td>
<td>38.4</td>
<td>105</td>
<td>110/78</td>
<td>33/23</td>
<td>125</td>
<td>20</td>
<td>18</td>
<td>Awake, quiet</td>
</tr>
<tr>
<td></td>
<td>Off</td>
<td>301</td>
<td>110</td>
<td>31</td>
<td>68</td>
<td>94</td>
<td>115</td>
<td>114/84</td>
<td>55/33</td>
<td>220</td>
<td>40</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Forty-eighth hour</td>
<td>On</td>
<td>248</td>
<td>270</td>
<td>34</td>
<td>41</td>
<td>50</td>
<td>37.6</td>
<td>4.54</td>
<td>120†</td>
<td>104/78</td>
<td>37/26</td>
<td>110</td>
<td>20</td>
<td>23</td>
</tr>
<tr>
<td></td>
<td>Off</td>
<td>301</td>
<td>160</td>
<td>29</td>
<td>36</td>
<td>40</td>
<td>5.81</td>
<td>126†</td>
<td>124/84</td>
<td>65/44</td>
<td>150</td>
<td>30</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

*Predicted oxygen consumption = 214 ml.
†Per cent increase over value on respirator.
‡Atrial fibrillation.
Blood gas studies in this patient indicated impaired pulmonary function. Despite administration of 100 per cent oxygen, arterial oxygen tensions after the earliest period never rose above 300 mm. Hg. These fell promptly when the patient was breathing spontaneously and until 48 hours after surgery, each trial without the respirator was followed by a rapid accumulation of carbon dioxide. We cannot state positively why this patient’s pulmonary function was so reduced. We know that her pulmonary compliance worsened during an episode of left ventricular failure on the operating table. In addition, areas of atelectasis were present, as they are after any thoracotomy, and these were further increased because of the accumulation of blood in the right chest and because of a very large heart. The patient’s pulmonary insufficiency was slow to clear. She was extubated and the respirator discontinued 48 hours postoperatively. Despite nine liters per minute of nasal oxygen, arterial oxygen tension remained in the range of 90 mm. Hg. Twenty-four hours later, after a period of breathing room air, she developed atrial fibrillation, which persisted during the remainder of her hospital stay.

In contrast to the above patient, in (D.C.), a 12-year-old boy with a ventricular septal defect, normal pulmonary artery pressure, and no history of chronic lung disease or congestive failure, the increase in oxygen consumption was minimal after surgery except during the period of shivering while he was re-warming (fig. 3). There was no evidence of significant pulmonary insufficiency. Arterial oxygen tensions reached 500 mm. Hg at
Postoperative Respiratory Effort

Physiologic Data in Two Patients during and after Use of Respirator

<table>
<thead>
<tr>
<th>Time</th>
<th>O2 Con</th>
<th>%↑</th>
<th>A4O2</th>
<th>VpO2</th>
<th>A4CO2</th>
<th>VpCO2</th>
<th>T</th>
<th>R</th>
<th>R</th>
<th>BP</th>
<th>CVP</th>
<th>F1</th>
<th>D1</th>
<th>% Incr.</th>
<th>Remarks</th>
</tr>
</thead>
<tbody>
<tr>
<td>Patient P, G. with aortic stenosis*</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Fourth hour</td>
<td>On</td>
<td>168</td>
<td>260</td>
<td>32</td>
<td>29</td>
<td>36</td>
<td>97.4</td>
<td>74</td>
<td>20</td>
<td>127/75</td>
<td>150</td>
<td>3.0</td>
<td>3.0</td>
<td>22/22</td>
<td>Shivering, hemithorax</td>
</tr>
<tr>
<td></td>
<td>Off</td>
<td>192</td>
<td>270</td>
<td>32</td>
<td>28</td>
<td>38</td>
<td>97.4</td>
<td>70</td>
<td>33</td>
<td>150/80</td>
<td>3.6</td>
<td>3.7</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Eighteenth hour</td>
<td>On</td>
<td>133</td>
<td>114</td>
<td>40</td>
<td>26</td>
<td>31</td>
<td>98.5</td>
<td>80</td>
<td>20</td>
<td>112/64</td>
<td>155</td>
<td>3.0</td>
<td>3.7</td>
<td>36/19</td>
<td>Quiet, large hemithorax</td>
</tr>
<tr>
<td></td>
<td>Off</td>
<td>186</td>
<td>106</td>
<td>43</td>
<td>29</td>
<td>30</td>
<td></td>
<td>80</td>
<td>30</td>
<td>103/65</td>
<td>180</td>
<td>4.1</td>
<td>4.4</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Patient W, F. with mitral stenosis†</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Third hour</td>
<td>On</td>
<td>136</td>
<td>255</td>
<td>44</td>
<td>30</td>
<td>33</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>3.16 3.26</td>
<td>Quiet, awake</td>
</tr>
<tr>
<td></td>
<td>Off</td>
<td>147</td>
<td>160</td>
<td>46</td>
<td>31</td>
<td>39</td>
<td>No change</td>
<td>3.37 3.2</td>
<td>+7/2.5</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

*Predicted O2 consumption/M2 = 131 ml. B.S.A. = 1.85 M2
†Predicted O2 consumption/M2 = 136 ml. B.S.A. = 1.88 M2
‡Cardiac index by Fick principle.
§Cardiac index by dye-dilution technie.
||% increase = change from on to off respirator.
major divisions: increased demand and decreased efficiency. It has been well established that oxygen consumption is increased after any major operation. The expected increase of 10 to 30 per cent may be further augmented by hyperpyrexia, shivering that occurs in hypothermic patients after surgery, infection, and apprehension. Following extracorporeal circulation there is evidence that an excess carbon dioxide accumulates in the tissues during prolonged perfusion, and this must be eliminated through the lungs. Venous carbon dioxide levels in our patients were uniformly high in the early postoperative period. Respiratory quotient determinations were high, suggesting an increased production of carbon dioxide. We have not been able to take over completely a patient's respiratory function in the early postoperative period unless the volume of gas administered was from 30 to 75 per cent higher than predicted from the Engstrom or other nomograms.

Respiratory insufficiency is nearly always present in patients after cardiac surgery. Respiratory insufficiency, from whatever cause, results in what is in effect a pulmonary arteriovenous shunt, limiting the effectiveness of ventilation and increasing respiratory demand. In addition, many of the factors that produce pulmonary insufficiency also increase the stiffness of the lung, thereby increasing the mechanical work necessary both to inflate and to deflate the lung.

In our patients, the work of breathing, as indicated by the difference between the oxygen consumed with the respirator and while breathing spontaneously was augmented significantly in all but three patients (D.V., H.D., W.F.). In those whose preoperative cardiopulmonary status was relatively good the increase in oxygen cost of breathing associated with surgery was usually significant. In patients whose preoperative status was not so good, a greater degree of postoperative pulmonary insufficiency was noted and associated with an even greater increase in work load. Many patients were thought too ill to warrant trial off the respirator due to the high incidence of severe arrhythmia associated with hypoxia. Thus, our determination of the work of breathing was confined to patients who did not develop significant hypoxia breathing spontaneously during the testing period, and, therefore had lesser degrees of respiratory insufficiency.

The percentage increase in oxygen consumption, in cardiac output, in circulatory pressures, and the frequent appearance of arrhythmias shortly after the discontinuance of respiratory assistance constitute persuasive arguments for the use of a respirator during the critical period of adjustment after open-heart surgery. Even in the absence of significant pulmonary insufficiency, the oxygen cost and hence work of breathing may be increased sufficiently to overburden the cardiopulmonary reserve with a significant increase in morbidity. It is quite likely that similar increases in the work of breathing will be found in other postoperative patients. Such examples may be the critically ill or debilitated patient, the obese patient, and the patient with chronic lung or heart disease, no matter what form of surgery is performed. We plan to apply this method of investigation to a broad spectrum of postoperative patients to determine the role played by the work of breathing in morbidity and mortality.

Summary

The Engstrom volume-controlled respirator is used routinely in patients after open-heart surgery as a short-term prophylaxis to avoid hypoxia, respiratory acidosis, and excessive fatigue. The respirator should be discontinued when pulmonary function is improved sufficiently to the point where the work of breathing is no longer excessive. With a spirometer designed for the Engstrom respirator, we have determined oxygen consumption and cardiac output in the spontaneously breathing patient and while assisted by the respirator. The difference between the two measurements reflects the oxygen cost of breathing and thus the work that can be spared the heart, by proper mechanical ventilation, during the period of acute postoperative stress. We have
demonstrated that the work of breathing, even in good risk patients, is appreciably increased postoperatively, that it is maximal immediately and then falls. These data lend objective support for the prophylactic use of respiratory assistance in patients after cardiac surgery. A similar application to other debilitated surgical and medical patients should be investigated.

References
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