lungs may shift the blood flow to the atelectatic lung by mechanical compression of pulmonary vasculature. When segmental or lobar atelectasis fail to respond to conservative measures and bronchoscopy, short-term specific techniques such as the use of cuffed endobronchial tubes and the ventilating bronchoscope, with inflation by a resuscitator bag should be considered. In patients with unilateral lung disease with severe refractory hypoxemia, CPAP can be selectively administered through a double-lumen tube to the diseased lung thereby preventing the above-mentioned complication.4

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REFERENCES

To the Editor:

We feel that our report of 40 consecutive patients receiving face mask CPAP demonstrated that this modality effectively improved oxygenation in selected patients with early respiratory distress. Included in this group was five patients with refractory atelectasis who also responded with an increase in their PaO2, as well as three patients who had complete reversal or prevention of atelectasis. This report neither substantiated nor advocated CPAP mask therapy for segmental or lobar atelectasis, but suggested its use as a possible "therapeutic alternative when other measures have failed." Certainly other investigators have also observed this response, noting the reversal of lobar atelectasis as well as prophylactically preventing clinically significant atelectasis.4

We do not completely agree with Dr. Venus's concern of the potential dangers of CPAP when treating patients with roentgenographically apparent unilateral lung disease. The majority of patients with significant atelectasis probably have diffuse microatelectasis. Although positive airway pressure directed to the lung with more significant involvement may provide greater improvement in PaO2, the use of a double lumen endotracheal tube or even intubation with resuscitator bag inflation seem to be extremely aggressive measures with even greater potential for danger than CPAP mask therapy.

A recent review of an approach to managing patients with asymmetric lung disease suggests the use of a double lumen endotracheal tube or single lumen intubation as a final measure to be initiated only after more conservative therapy including a CPAP mask trial have failed.4

The overall role of the use of the CPAP mask in treating established atelectasis will hopefully be further defined in future prospective studies.

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REFERENCES

Preinduction Use of Pulmonary Artery Catheters during Cardiac Surgery

To the Editor:

The article by Feikes and Roberts, "Simplified Method of Intraoperative Pulmonary Artery Flow-Directed Catheter Insertion during Cardiac Surgery" (Chest 1982; 81:755), described a modification of a previously utilized technique for insertion of central monitoring catheters via the innominate vein at the time of median sternotomy. The major criticism of this technique is that it leaves the patient with relatively little reliable monitoring for the most critical period of the operation, especially for the patient undergoing coronary bypass surgery: that is, the time of anesthetic induction. It is probable that early and immediate treatment of the signs of cardiac ischemia will forestall many potentially diastolic complications, such as ventricular fibrillation. In our experience the most sensitive indicator of impending ischemia has been a rise in the pulmonary capillary wedge pressure and this has been aggressively treated at our institution with intravenous nitroglycerin. Even the ST segment morphology on the precordial electrocardiogram has lagged behind the rise in wedge pressure in most patients. Insertion of the balloon flotation catheter preoperatively has taken about 10 minutes in our institution and we have had no difficulties in floating the catheter into the wedge position on any occasion. There have been no catheter-related infectious complications using the percutaneous right internal jugular vein approach.

At least some portion of the increased safety with which we can perform open heart surgery on critically ill patients must be attributed to more precise hemodynamic monitoring which has allowed us to rapidly intervene pharmacologically in order to prevent subtle changes in subendocardial perfusion from degenerating into severe subendocardial necrosis, arrhythmias or cardiac arrest. If one subscribes to the suggestion that "the routine use of this technique (use of the balloon flotation catheter) may be associated with a reduction in perioperative myocardial damage," it seems only reasonable to insert the catheter so that the data derived therefrom will be available at the most critical moments of the operation.

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Sexual Dysfunction and Erectile Impotence in Chronic Obstructive Pulmonary Disease

To the Editor:

We read with interest the paper describing sexual dysfunction in chronic obstructive pulmonary disease (COPD) by Fletcher and Martin (Chest 1982; 81:413-21). Of 20 COPD sufferers in their study, six had organogenic erectile impotence (OEI), and the others lesser degrees of sexual impairment. The authors concluded that both
Table 1—Sexual Function in Hypoxic Men with Bronchitis

<table>
<thead>
<tr>
<th>Patient No</th>
<th>Age (Years)</th>
<th>PaO₂ (Normal 80-100 mm Hg)</th>
<th>Testosterone (Normal 11-50 ng/ml)</th>
<th>Diminished Libido?</th>
<th>Time since last sexual intercourse</th>
<th>Erectile difficulty?</th>
<th>Early morning erection?</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>50</td>
<td>36</td>
<td>6 (when well 9)</td>
<td>Yes-18 months</td>
<td>2 months</td>
<td>Yes</td>
<td>No-18 months</td>
</tr>
<tr>
<td>2</td>
<td>36</td>
<td>39</td>
<td>6 (when well 19)</td>
<td>Yes-when chest bad</td>
<td>3 months</td>
<td>Yes</td>
<td>Not when chest bad</td>
</tr>
<tr>
<td>3</td>
<td>53</td>
<td>39</td>
<td>4 (when well 9)</td>
<td>Yes-10 years</td>
<td>4 months</td>
<td>No</td>
<td>No-years</td>
</tr>
<tr>
<td>4</td>
<td>52</td>
<td>52</td>
<td>7 (when well 13)</td>
<td>Yes-3 months</td>
<td>3 months</td>
<td>Yes</td>
<td>No-years</td>
</tr>
<tr>
<td>5</td>
<td>57</td>
<td>39</td>
<td>10</td>
<td>Yes-10 years</td>
<td>3 months</td>
<td>Yes</td>
<td>No-many years</td>
</tr>
<tr>
<td>6</td>
<td>60</td>
<td>48</td>
<td>12</td>
<td>Yes-3 years</td>
<td>3 years</td>
<td>Yes</td>
<td>No-2 years</td>
</tr>
<tr>
<td>7</td>
<td>43</td>
<td>42</td>
<td>22</td>
<td>No</td>
<td>Recent</td>
<td>No</td>
<td>Yes-frequent</td>
</tr>
<tr>
<td>8</td>
<td>54</td>
<td>57</td>
<td>15</td>
<td>Yes-10 years</td>
<td>6 weeks</td>
<td>Yes</td>
<td>Yes-occasional</td>
</tr>
<tr>
<td>9</td>
<td>53</td>
<td>37</td>
<td>15</td>
<td>Yes-3 years</td>
<td>3 years</td>
<td>Yes</td>
<td>No-2 years</td>
</tr>
<tr>
<td>10</td>
<td>50</td>
<td>47</td>
<td>9</td>
<td>Yes-when chest bad</td>
<td>4 months</td>
<td>When chest bad</td>
<td>No-4 months</td>
</tr>
</tbody>
</table>

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Organic and psychological factors contributed, but no physiologic or biochemical factor was incriminated. Degree of sexual dysfunction correlated with severity of hypoxemia and sexual performance improved in two subjects given oxygen therapy. The authors remarked on the paucity of literature in this field, but evidently had not noticed our earlier published work.

We first described reduced serum testosterone in COPD in 1979. Further studies suggested that the degree of testosterone reduction is related to severity of hypoxemia, but hypoxemic hypothalamic-pituitary-testicular (H-P-T) suppression occurs and that such changes are reversible. In a study of ten randomly selected hypoxic COPD men serum testosterone levels were lower than an age-matched control group (patient mean 10.9±5.8; control mean 21.5±5.6, p<0.01) and six had subnormal values. Nine had diminished libido and seven absent early morning erections (EME's). Patients 1-4 were studied while acutely ill and again after recovery. A rise in PaO₂ was associated with serum testosterone rise, improvement in sexual performance in three patients, and return of EME's in two. The three patients with regular EME's were those with highest serum testosterone values. Accordingly, an association between OEL in COPD and testosterone suppression by hypoxemia was postulated.

Although Fletcher and Martin found normal testosterone values, their patients were less hypoxic than ours (mean PaO₂ 60 mm Hg vs PaO₂ 44 mm Hg). Moreover, OEL may not be an "all or none" phenomenon and normal serum testosterone levels do not necessarily reflect a normal H-P-T axis as supported by our finding of H-P-T suppression in COPD patients with low normal serum testosterone levels. Psychiatric disturbances of COPD may be another consequence of hypoxemia and are not necessarily causally related to impotence. Finally, our current projects suggest that similar endocrine and sexual disturbances affect patients with hypoxic restrictive lung disease but apparently not those with cyanotic congenital heart disease.

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References


To the Editor:

Dr. Semple and colleagues suggest that disturbed hypothalamic-pituitary-testicular axis might be a possible cause of decreased libido and impotence in patients with chronic obstructive pulmonary disease (COPD). We did not find a significant correlation between serum testosterone and room air arterial O₂ tension (PaO₂) in our subjects (linear regression analysis). We further analyzed the data by separating subjects into a severely hypoxic group (PaO₂≤50 mm Hg; n=6) and a moderately hypoxic group (PaO₂>60 mm Hg; n=12). The respective mean serum testosterone values of 6.7±2 (SD) and 6.9±2.1 MIU/ml were not significantly different. Likewise, the mean serum testosterone value for the organogenic impotence group was not different from that of the non-impotent group. The lack of difference between moderate and severely hypoxic subjects may in part be due to the fact that four (mean PaO₂=44.5 mm Hg) of the eight severely hypoxic subjects were receiving chronic supplemental home O₂ at the time of study. Thus, while the data presented by Semple et al are interesting, our findings were not similar.

We did suggest a physiologic factor that may have contributed to the organogenic impotence associated with COPD. Increased bulbo cavernosus latency times in the organogenic group suggested the possibility of peripheral autonomic neuropathy. Perhaps both low testosterone and peripheral neuropathy occur in COPD patients and the cause of organogenic impotence is multifactorial.

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