Platypnea Related to Constrictive Pericarditis

To the Editor:

In the February 1994 issue of Chest, Mashman et al\(^1\) describe an interesting case of platypnea due to pericarditis. As part of their report, the authors list a variety of other disease states associated with platypnea. We are puzzled as to why they omitted chronic liver disease from their list.

Platypnea, which is common and well described in chronic liver disease, is thought to be related to orthodeoxia, deoxygenation when the patient is upright.\(^2\) Two possible mechanisms for this deoxygenation have been suggested, both of which include the presence of abnormally dilated intrapulmonary vessels at the lung bases leading to venous admixture in the upright position.\(^3,4\)

In cirrhosis, true anatomic intrapulmonary arteriovenous shunts can occur at the lung bases. In the upright position, blood flow follows gravity to the bases, increases intrapulmonary shunt, and creates hypoxemia unresponsive to supplemental oxygen. Alternatively, due to the basilar dilatation of intrapulmonary precapillary and capillary blood vessels, a central stream of venous blood may be created. This central blood may remain unoxygenated due to the increased distance from the alveolar epithelium in the setting of a decreased transit time. Since supplemental oxygen will partially correct this hypoxemia, it is not considered to be a true shunt.\(^5\)

Clinically, platypnea and orthodeoxia in chronic liver disease are often associated with spider nevi of the skin. Diagnostically, chest radiographs may suggest bibasilar interstitial infiltrates, and pulmonary angiograms may show “spongy” vasculature within the lung parenchyma. Both $\text{Te}$-99m labeled macroaggregated albumin whole body scans and contrast enhanced echocardiography have been used to detect dilated pulmonary vessels by exploiting that these vessels allow large particles to enter the systemic circulation. Finally, the platypnea and orthodeoxia caused by these intrapulmonary shunts may reverse after liver transplantation.\(^2\)

Although these authors chose to place COPD first on their list of causes of platypnea, we suspect it probably occurs less frequently in those patients than in those patients who with what has recently been referred to as the “hepato-pulmonary syndrome”\(^2\) and who they inadvertently overlooked.

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REFERENCES


To the Editor:

We appreciate the comments of Drs. Vora and Nierman regarding our recent report (Chest 1994; 105:636-37). They correctly point out that chronic liver disease may be associated with the symptom of platypnea and review two possible mechanisms. Both mechanisms are related to shunting that develops in the pulmonary circulation. In our table, we list shunts but do not specify that chronic liver disease can be the cause. Perhaps we could have, as Drs. Vora and Nierman suggest, listed chronic liver disease as a separate entry.

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Unplanned Extubation

To the Editor:

We read with interest three recent studies on unplanned extubations in a variety of critical care settings.\(^1,3\) Listello and Sessler\(^1\) reviewed retrospectively 81 instances of unplanned extubation. Reintubation was required in 45% of the cases, and it was associated with a number of clinical parameters. Tindol et al\(^2\) studied prospectively 13 episodes of unplanned extubation in 400 patients and also noted a 50% reintubation rate. The low rate of unplanned extubation (3%) in the latter study differed from that reported previously (range, 8.5% through 16%).\(^3\) Whelan and coworkers\(^3\) related ventilatory parameters prior to unplanned extubation and the need for reintubation.

We also monitored unplanned extubation in our medical ICU during 1991 to 1993 to identify risk factors for its occurrence. Thirty-five instances (14.4%) of unplanned extubation occurred among 243 intubated patients at risk. The only apparent risk factor for unplanned extubation was a prior episode of self-extubation. One third of all unplanned extubations were associated with a prior episode of self-extubation. Eighteen (51%) of the 35 episodes required reintubation, consistent with the findings of both Listello and Sessler\(^1\) and Tindol.\(^2\) When related to time of day, unplanned extubation was least likely to occur during the night shift (11 PM to 8 AM), which accounted for 21% of all episodes. Thirty-seven percent and 42% of all unplanned extubations occurred during the 8 AM to 5 PM and 5 PM to 11 PM shifts, respectively. Tindol et al\(^2\) observed a similar time distribution for unplanned extubation and attributed it to increased activity in the ICU during daytime hours, which may be distracting to staff.\(^2\)

We commend the studies by Listello and Sessler, Tindol et al, and Whelan et al for highlighting an important problem in critical care medicine. On the one hand, the review of clinical parameters that characterize successful unplanned extubations may add insight to discussions of weaning and patient readiness for planned extubation. On the other hand, because unplanned