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Table 1—Pressure Values in Two Tracheal Models

<table>
<thead>
<tr>
<th>ID of Rusch</th>
<th>LWP at ICP of 25 mm Hg, mm Hg</th>
<th>ICP at LWP of 25 mm Hg, mm Hg</th>
</tr>
</thead>
<tbody>
<tr>
<td>21-mm-ID</td>
<td>Model</td>
<td>Model</td>
</tr>
<tr>
<td>26-mm-ID</td>
<td>Model</td>
<td>Model</td>
</tr>
<tr>
<td>6</td>
<td>0</td>
<td>233</td>
</tr>
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<td>8</td>
<td>23</td>
</tr>
<tr>
<td>9</td>
<td>15</td>
<td>3</td>
</tr>
</tbody>
</table>

*ETT = endotracheal tube; ICP = intracuff pressure; ID = inner diameter; LWP = lateral wall pressure.

Routine Monitoring of Intracuff Pressure

To the Editor:

In an article that appeared in the October 1991 issue of Chest, Guyton et al. once again raised the questions "Is 25 mm Hg the cutoff level for the safe intracuff pressure (ICP)?" and "Is routine ICP monitoring useful or worthwhile?" Even though their findings are not totally new, the authors deserve to be complimented for the impressive demonstrations of the effect of airway pressure on ICP and dynamic self-sealing action. The spikes of ICP during the inspiratory phase secondary to the high airway pressure in poorly compliant lungs may partly explain the continuous problems of tracheal damage despite the use of high-volume, low-pressure cuffs. Unfortunately, the authors did not measure the lateral wall pressure (LWP) exerted upon the tracheal mucosa by the cuff (or cuffed-tracheal [CT] pressure, as Guyton et al referred to it) and seemed to assume that the ICP approximately equaled the LWP if this is the case, apparently an ICP of 25 mm Hg is not always safe.

The pressure that causes ischemic damage to the tracheal mucosa should be the exerting LWP, not the ICP itself. It has been shown that the two pressures are not always equivalent. Recently, we performed a study to evaluate the discrepancy between ICP and LWP. With two tracheal models (inner diameters of 21 and 26 mm), we measured ICP and LWP simultaneously in a series of Rusch endotracheal tubes (inner diameters of 6 to 9 mm). The LWP values were recorded when ICPS were 25 mm Hg and vice versa. The results are summarized in Table 1.

Our study showed that the correlation between LWP and ICP was poor, in general—more so when the cuff diameter was too small or too large for the trachea. The ICP was always higher than the LWP, but the gradient between them was not predictable with endotracheal tubes of different shapes and sizes in tracheal simulators of different sizes. In other words, ICP on many occasions may not appropriately reflect LWP. In vivo, the distensibility of the trachea and the dynamic character of mechanical ventilation surely make the estimation of LWP from ICP even more difficult. It is possible that an ICP greater than 25 mm Hg will not cause tracheal complications as long as the LWP is not in excess of the capillary perfusion pressure. It is important to realize that many patients require excessive ICP (>25 mm Hg) to seal the trachea during mechanical ventilation when lung compliance is very low. Off et al. reported that 22.3 percent of their patients had a high ICP that could not be corrected to a so-called safe range (<25 mm Hg) even by use of minimal leak technique. They concluded that routine ICP monitoring did not benefit the patient. We believe that monitoring ICP is a good and vigilant medical practice even with its shortcomings. However, without understanding and consideration of LWP, overemphasis on monitoring ICP may itself be easily misleading and not really worthwhile after all.

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To the Editor:

Dr Lee's comments are appreciated, hit the mark, and point out some of the difficulties in estimating or preventing potential tracheal damage associated with tracheal-tube cuff overinflation. The ICP and LWP do not always correlate, and a high ICP, as his data show, may not be associated with a correspondingly high LWP. Conversely, however, if the ICP is low (<25 mm Hg) and the cuff is sealing appropriately, a clinician may be reasonably assured that the LWP is within 'safe' limits. Under these conditions, tracheal damage is unlikely. Hence, measurement of ICP is a useful, though not optimal, adjunct to ventilatory monitoring.

In theory, the absolute value of ICP, even when very high, would not be damaging if the cuff was inflated very carefully until it made minimal contact with the tracheal wall. In practice, such care in...
cuff inflation is seldom seen, and an increase of 1 ml or less in cuff volume can markedly increase LWP.

What can be done to minimize the possibility of cuff-induced tracheal damage? First, select the largest endotracheal tube that can be insertedatraumatically. A greater cross-sectional area ratio between the tube and the trachea means less air must be injected into the cuff to effect a seal. Second, use a tube with the greatest cuff length and resting cuff volume. As our report showed, the variability between different cuffs, even from a specific manufacturer, can be significant. Third, measure ICP; even though the absolute pressure may not reflect the true LWP a progressive increase of ICP necessary to maintain a cuff seal, particularly when high peak inflation pressure is necessary, may indicate a need to change the tube, reposition it, or consider alternatives, such as intermittent cuff inflation.1,2 As we stated in our study, the ultimate solution awaits new and improved cuff design.

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Possible Role of Tungsten Oxide Whiskers in Hard-Metal Pneumoconiosis

To the Editor:

One of the key processes in tungsten technology is the reduction of tungsten trioxide with a gas phase to yield the tungsten metal. Whiskers of slightly reduced tungsten trioxide are a characteristic reaction product during the process.1 Hard metal is manufactured by a process of powder metallurgy from tungsten metal and carbon, with cobalt as a binder. Dust generated during the process of manufacturing hard metals causes clinical and pathologic changes in the lungs of workers, which have occasionally proved fatal. Such industrial airborne dust, which can cause severe lung fibrosis, is of a heterogeneous, multielement type.

In a communication to the editor of Chest in April 1985, Dr Cullen2 emphasized that the cause of lung disorders that have been associated with exposure to hard metal (tungsten carbide with binder, including cobalt) remains uncertain. By contrast, Dr Abraham3 argued in the same issue that the causal agent has been clearly identified and that the pathologic condition observed in hard-metal industry employees is produced by cobalt. No previous report has shown or suggested the presence of airborne fibers in the work environment in the hard-metal industry.

In a study conducted as part of a project on optimizing the sample transfer method for analysis of airborne fibers by transmission electron microscopy, airborne tungsten oxide fibers were observed in such an environment. Figure 1 shows a typical photomicrograph of the observed fibers. It is not, however, within the scope of this letter to give more elaborate details of the results. Nevertheless, all fibers were respirable, and about 80 percent of the fibers were 0.3 μm or less in diameter. This suggests that most of these fibers are below the practical resolution limit of the optical microscope.

Inhalation of asbestos fibers has long been associated with a variety of malignant and nonmalignant respiratory diseases. In recent years, however, the category of possibly etiologic fibers has been broadened to include other durable inorganic fibers. Fibers with chemical characteristics, structure, and dimensions different from those of asbestos are suspected to have caused various biologic effects.1,2 In this respect, our observation is compatible with the case report of Dr Cullen, that occupational exposure to such fibers has not been accounted for in discussions of hard-metal pneumoconiosis. It should also be noted that tungsten makes up a major part of hard-metal production, about 95 percent.

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Bronchoscopically Induced Bleeding

To the Editor:

We read with interest in the October 1991 issue of Chest the excellent review by Cordasco et al. on the prevention and management of bleeding induced by bronchoscopic biopsy. We would like to comment on another technical aspect of transbronchial biopsy that seems of importance in our experience.

We have encountered severe bleeding (more than 100 ml) after transbronchial biopsy in three patients with sarcoidosis or diffuse lung disease. On each occasion, the sequence of events had been as follows: After closing the cups, a greater than usual resistance had been felt in withdrawing the biopsy forceps. At the same time,