of Chest, Abroug et al. state that the normal values of systemic blood pressure and vascular resistance observed in their patients contradict the hypothesis attributing a preponderant role to catecholamine discharge. They conclude that the mechanism of "left ventricular dysfunction" remains unclear and speculate that involvement of superimposed alveolar capillary lesions in the genesis of pulmonary edema is not excluded, without pathologic or experimental evidence of the existence of such lesions.

The authors also propose "a primary scorpionic myocarditis" as the possible mechanism of the abnormalities observed. We also suspected a direct myocardial effect of scorpion venom based only on ECG changes, although such direct myocardial involvement is observed only in the isolated working guinea pig heart and in isolated atrial and papillary muscles, but not in the intact animal. We recently observed enzymatic myocardial injury in a group of symptomatic children without heart failure; however, we were unable to substantiate the hypothesis of a direct myocardial effect of scorpion venom.

Furthermore, there is ample evidence in humans of the existence of catecholamine cardiomyopathy due to excessive release of catecholamines, as seen in scorpion envenomation with characteristic myocardial pathology similar to that described in pheochromocytoma. Although the authors believe that the mechanism of pulmonary edema is not clear, there is sufficient evidence of left ventricular diastolic dysfunction, and recent echocardiographic observations emphasized reversible depressed systolic function ("myocardial stunning") after envenomation.

It should be emphasized that hemodynamic monitoring is quite difficult in small children with central nervous system dysfunction with or without respiratory failure. Hypovolemia due to excessive perspiration, increased insensible loss, and vomiting due to pancreatitis may obscure the hemodynamic changes. Echocardiography is quite sensitive in assessing myocardial compromise after envenomation without subjecting the patient to invasive evaluation except in the presence of pulmonary edema and hypovolemia, as seen in our patients, in order to facilitate the regulation of afterload reduction therapy.

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To the Editor:

Our own experience and published studies on scorpion envenomation do not provide the certitudes of Gueron and Sofer about the mechanisms of pulmonary edema (PE) following scorpion envenomation and the underlying cardiac dysfunction.

Although most clinical and experimental studies and even our series give strong evidence of the hemodynamic type of PE following scorpion envenomation, we find we cannot conclude that this is the exclusive mechanism. Indeed, in an experimental model of scorpion envenomation, Rossi et al. found severe structural damage of the alveolocapillary barrier, suggesting a possible role of high permeability in the pathogenesis of PE. Furthermore, Rahav and Weiss reported on one patient suffering from scorpion envenomation-induced PE with a pulmonary capillary wedge pressure within the normal range. Moreover, in some of the patients we recently treated for scorpion envenomation-induced PE, we found bronchial aspiration with an increased albumin content (unpublished data). All of these facts led us to be cautious in the conclusions of a report of five cases. Thus, as long as there are no definitive data, high-permeability PE should be kept in mind, and further studies are needed to search for ARDS indicators in this setting.

We believe that the mechanism of scorpion envenomation-induced cardiac dysfunction is still unclear. The catecholaminergic hypothesis of Gueron et al. needs to be more carefully substantiated in the scorpion envenomation setting. We agree that there is sufficient evidence of the existence of catecholaminergic cardiomyopathy in humans, but we lack precise information about the time course and the reversibility of such cardiomyopathy, and any comparison with scorpion envenomation cardiomyopathy appears hazardous. There is also a documented catecholaminergic discharge following spoid envenomation. But should scorpion envenomation-induced cardiomyopathy be categorized as a catecholaminergic cardiomyopathy? The histopathologic changes observed at autopsy in scorpion envenomation (interstitial edema, increased cellularity, cardiac cell necrosis) should not be considered as specific for catecholaminergic cardiomyopathy.

In our study, we reported on early hemodynamic data neither supporting nor excluding the catecholaminergic hypothesis. We do not exclude other hypotheses, especially a possible "scorpionic myocardiopathy" due to a direct myocardial effect of scorpion venom.

We believe that there are still many dark areas in the comprehensive approach to scorpion envenomation-induced cardiopulmonary disturbances, and any conclusion should be cautious and rigorous.

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Table 1—Pressure Values in Two Tracheal Models*

<table>
<thead>
<tr>
<th>1D of Rusch</th>
<th>ETT, mm</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 Model</td>
<td>6</td>
<td>0</td>
<td>233</td>
</tr>
<tr>
<td>26-mm-ID Model</td>
<td>7</td>
<td>18</td>
<td>58</td>
</tr>
<tr>
<td>21-mm-ID Model</td>
<td>8</td>
<td>8</td>
<td>54</td>
</tr>
<tr>
<td>26-mm-ID Model</td>
<td>9</td>
<td>15</td>
<td>62</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 al1 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.2 Recently, we performed a study to evaluate the discrepancy between ICP and LWP.2 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.3 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 that 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