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Cardiogenic Shock
Nothing Has Changed

To the Editor:

In the November 2003 issue of CHEST, Lim and colleagues1 suggest that some patients initially presenting with cardiogenic shock actually succumb to a form of distributive shock. The idea of altered gut permeability and bacterial translocation in cardiogenic shock is not new. Brunkhorst et al2 reported patients presenting with cardiogenic shock surviving > 12 h were found to have elevated levels of procalcitonin, neopterin, tumor necrosis factor-α, interleukin-6, and C-reactive protein, suggesting exposure to bacterial endotoxin presumed to be of gut origin. However, evidence for this phenomenon in the patients reported by Lim et al1 is lacking and is not supported by the hemodynamic data. Using data presented in Table 2 of the article by Lim et al, stroke volume index and left ventricular stroke work index can be calculated. In addition, interpretation of the hemodynamic data within a “per-beat” rather than traditional “per-minute” framework using stroke systemic vascular resistance index in place of systemic vascular resistance index allows a more accurate assessment of the state of vasoactivity as the cardiac index used in the systemic vascular resistance index equation already includes chronotropic compensation by the heart rate.3 The true nature of shock in both the low and normalized cardiac index groups is shown in Table 1, and they are the same. All patients are hypodynamic (low stroke volume index), hypocontractile (low left ventricular stroke work index), and vasoconstricted (high stroke systemic vascular resistance index), a state compatible with progressive failure of the left ventricular pump. Not surprisingly, the younger normalized cardiac index group was better chronotropically compensated achieving heart rates of 5 to 17% in excess of their older low cardiac index counterparts. Both groups, however, achieved similar percentages of their age-predicted maximum heart rate, suggesting chronotropic competence appropriate for their age (Table 1). Although the difference in heart rate between groups did not reach statistical significance, the difference is clinically significant as elevated heart rate engenders proportional increases in cardiac index given a constant stroke volume index. Other than cardiac index, no other measure of myocardial function was returned to even near normal despite maximal therapy. Thus, it would appear both the low and normalized cardiac index groups represent one group exhibiting a narrow spectrum of hemodynamic derangements within the

Table 1—Hemodynamics in Nonsurvivors With Low and Normalized Cardiac Index*

<table>
<thead>
<tr>
<th>Variables</th>
<th>0 h</th>
<th>6 to 12 h</th>
<th>12 to 18 h</th>
<th>18 to 24 h</th>
<th>Final</th>
</tr>
</thead>
<tbody>
<tr>
<td>Stroke volume index, mL/beat/m²</td>
<td>19.2</td>
<td>18.4</td>
<td>21.9</td>
<td>19.1</td>
<td>17.5</td>
</tr>
<tr>
<td>Low cardiac index</td>
<td>18</td>
<td>26.3</td>
<td>19.6</td>
<td>23.4</td>
<td>25.5</td>
</tr>
<tr>
<td>Normalized cardiac index</td>
<td>13.6</td>
<td>11.8</td>
<td>15.8</td>
<td>13.5</td>
<td>10.9</td>
</tr>
<tr>
<td>LVSWI, g/beat/m²</td>
<td>14.2</td>
<td>20.7</td>
<td>15.2</td>
<td>18.8</td>
<td>19.8</td>
</tr>
<tr>
<td>Low cardiac index</td>
<td>229</td>
<td>217</td>
<td>216</td>
<td>247</td>
<td>224</td>
</tr>
<tr>
<td>Normalized cardiac index</td>
<td>276</td>
<td>182</td>
<td>249</td>
<td>212</td>
<td>185</td>
</tr>
<tr>
<td>% Vasoconstricted</td>
<td>63</td>
<td>55</td>
<td>54</td>
<td>76</td>
<td>60</td>
</tr>
<tr>
<td>Low cardiac index</td>
<td>97</td>
<td>30</td>
<td>78</td>
<td>57</td>
<td>32</td>
</tr>
<tr>
<td>Normalized cardiac index</td>
<td>99</td>
<td>87</td>
<td>97</td>
<td>89</td>
<td>97</td>
</tr>
<tr>
<td>Heart rate, beats/min</td>
<td>111</td>
<td>107</td>
<td>107</td>
<td>107</td>
<td>102</td>
</tr>
<tr>
<td>Low cardiac index</td>
<td>68</td>
<td>60</td>
<td>67</td>
<td>61</td>
<td>67</td>
</tr>
<tr>
<td>Normalized cardiac index</td>
<td>72</td>
<td>62</td>
<td>69</td>
<td>60</td>
<td>66</td>
</tr>
</tbody>
</table>

*LVSWI = left ventricular stroke work index; SSVRI = stroke systemic vascular resistance index.
same clinical entity, cardiogenic shock, and succumb to the so-called downward spiral after all. Any thought to the contrary would be real “hubris” indeed.

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To the Editor:

We thank Dr. Joffe for his comments on our article. Although interesting, the concept of single-beat analysis should be regarded with caution. We strongly disagree with Dr. Joffe’s hypothesis suggesting that most of the differences can be explained by differences in heart rate. Indeed, this would imply that stroke volume was lower or equal, but surely not higher, in the high cardiac index group. However, we observed just the reverse: stroke index decreased over time by 9% in the low cardiac index group, while it increased by 38% in the normalized cardiac index group. In addition, heart rate decreased over time in the normalized cardiac index group, while it increased by 33% in the normalized cardiac index group, which is more severe in nonsurviving than in surviving patients, including endotoxin and cytokine release, and excessive nitric oxide release leading to plexus nitrate formation may also explain why these patients present pronounced microcirculatory alterations, which are more severe in nonsurviving than in surviving patients, and are remarkably similar to those observed in patients with septic shock.

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Differentiating Asthma and COPD Patients

I read with interest the article by Hanania et al1 in September 2003, where the combination of fluticasone propionate (250 μg)/salmeterol (50 μg bid) was found to improve FEV1 better than salmeterol in COPD. Moreover, fluticasone alone improved FEV1. I am concerned that the article has inadvertently allowed the inclusion of asthmatic patients who were mislabeled as “COPD.”

1. Patients’ ages were ≥ 40 years. Moderate-to-severe COPD is rare below the age of 50 years.2
2. The exclusion criteria removed patients with “current diagnosis of asthma.” This implies that patients who had asthma in the past were recruited. And who decided they were no more asthmatic? Over a hundred doctors at 76 investigative sites across the United States, where the standards of clinical judgment vary.
3. The patients recruited were classified as “reversible” and “nonreversible.” The mean percentage increase of FEV1 in the reversible group—after administering 400 μg of albuterol—was 30% (range not given). As the inclusion criteria included patients with baseline FEV1 < 65%, it is very likely that many patients in the reversible group had achieved an FEV1 of > 80% after albuterol, which takes them out of the definition of COPD.3
4. The patients’ response to medications is typical of asthma in that fluticasone was effective at a small dose. Several previous studies on COPD have failed to demonstrate such effect even though larger doses of inhaled corticosteroids were used.4 In addition, the response to fluticasone was manifest 24 h only after the initiation of treatment. This is too quick a response for patients with COPD outside acute exacerbation.

My concern is highlighted by the fact that differentiation between asthma and COPD can sometimes be difficult.2 Also, a metaanalysis5 of eight articles showed that inhaled corticosteroids had improved FEV1 in patients with COPD. The same analysis—after excluding the articles that included patients with asthma/COPD—showed no such effect.6 The inclusion of a few asthmatic cases can tip the balance towards response.

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References

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