Table 1—Acid-Base Data of Patients Who Developed vs Those Who Did Not Develop Hyperchloremic Metabolic Acidosis During the First 24 h of Resuscitation

<table>
<thead>
<tr>
<th>Acid-Base Data</th>
<th>HMA at 24 h</th>
<th>No HMA at 24 h</th>
<th>P Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Creatinine, mg/dL</td>
<td>2.1</td>
<td>2.7</td>
<td>.6</td>
</tr>
<tr>
<td>HCO₃, mEq/L</td>
<td>16.2</td>
<td>20.1</td>
<td>.01</td>
</tr>
<tr>
<td>Chloride, mEq/L</td>
<td>118.2</td>
<td>108.9</td>
<td>.0001</td>
</tr>
<tr>
<td>pH</td>
<td>7.33</td>
<td>7.35</td>
<td>.46</td>
</tr>
<tr>
<td>AG, mEq/L</td>
<td>11.5</td>
<td>17.0</td>
<td>.0001</td>
</tr>
<tr>
<td>Total administered fluid, L</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Mean</td>
<td>7.9</td>
<td>7</td>
<td>.5</td>
</tr>
<tr>
<td>Median</td>
<td>7.8</td>
<td>5.1</td>
<td>.04</td>
</tr>
<tr>
<td>Administered saline</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Mean, mL</td>
<td>6.6</td>
<td>4.4</td>
<td>.03</td>
</tr>
<tr>
<td>Median, mL</td>
<td>6</td>
<td>3.3</td>
<td>.002</td>
</tr>
<tr>
<td>Rate, mL/min</td>
<td>276.2</td>
<td>183.5</td>
<td>.002</td>
</tr>
</tbody>
</table>

AG = anion gap; HCO₃ = bicarbonate; HMA = hyperchloremic metabolic acidosis.

Age, diuretic use, chronic kidney disease, lactic acidosis, and bicarbonate infusion were not associated with HMA.

HMA has been described in dog models and in human anecdotes since 1900. Stewart and Rourke described the effects of large-volume resuscitation, and Winters et al proposed that HMA may be caused by dilution of bicarbonate. HMA has long been appreciated after resuscitation of patients with diabetic ketoacidosis and was recently reported in children with meningococcal septic shock. Ketoacidosis differs somewhat in that urinary ketone excretion contributes (with dilution) to the development of HMA.

In conclusion, this limited retrospective study suggests that HMA is common during resuscitation of patients with a primary diagnosis of shock, and that HMA is associated with volume of infused saline. The limitations of this retrospective, medical records review preclude precise estimates of frequency and risk of this phenomenon, but the findings suggest hypotheses for a prospective study.

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REFERENCES

Questions in the Role of Chest CT Scanning in TB Outbreak Investigation

To the Editor:

The study by Won Lee and colleagues from South Korea in the recent issue of CHEST (May 2010) has generated a number of important issues and questions that need to be highlighted. The authors have diagnosed active pulmonary TB (PTB) in about 21% of patients, and it is really hard to believe this unusually high rate of active disease, especially when all the patients are young soldiers who are immunocompetent and healthy.

It is quite surprising that none of the 18 cases of active PTB that were diagnosed using high-resolution CT (HRCT) scans showed positive acid-fast staining of the sputum specimens. Among the nine cases of active PTB diagnosed on the basis of CT scanning alone, two patients had neither any symptoms nor a positive sputum test result for acid-fast bacilli culture. Did these soldiers really have active PTB? How often do we see patients with active PTB who have no symptoms? Was administering a full course of antitubercular treatment for 6 months to these patients justified?

The authors have claimed symptomatic or radiographic improvement in all cases of active PTB. Therefore, in those nine patients with normal chest radiographs and active PTB, follow-up HRCT scans of the thorax must have been done to assess radiologic improvement. How do we view this very high dose of radiation exposure in these young soldiers? It is well known that radiation exposure in these young soldiers? It is well known that radiation exposure in these young soldiers?
generates highly reactive free radicals and is carcinogenic, teratogenic, and mutagenic. It has been associated with cancers of the thyroid, bone, lung, breast, and leukocytes. Like lead and asbestos, radiation has no safe threshold. Therefore, performing HRCT scans of the thorax in only those patients who have symptoms suggestive of active PTB seems to be a more pragmatic approach.

The authors have relied heavily on the results of interferon-\( \gamma \) (INF-\( \gamma \)) release assay in a few patients for the diagnosis of active PTB, even in the absence of clinical symptoms. It is well known that higher production of INF-\( \gamma \) correlates to some extent with the activity of *Mycobacterium tuberculosis* infection, but its low sensitivity and specificity for distinguishing active vs latent TB has been demonstrated by various studies worldwide, and therefore it is not recommended as a diagnostic tool for active TB.\(^3\)\(^5\)

We have been demonstrated by various studies worldwide, and therefore the sensitivity and specificity for distinguishing active vs latent TB has been demonstrated by various studies worldwide, and therefore

However, in our opinion, there are some interesting issues to be discussed.

According to the recent European Society of Cardiology guidelines on pulmonary hypertension (PH), the definition of PH on exercise is not supported by published data.\(^3\) Thus, no definition of exercise-induced PH can be provided currently. Moreover, US guidelines are consistent with this statement.\(^3\) However, recently published data indicate that more attention should be paid to an exaggerated increase of systolic PAP during exercise, which is suggested to be a marker of early pulmonary vasculopathy in connective tissue disease. Kovacs et al\(^1\) revealed that EDE showed a systolic PAP >40 mm Hg during exercise in 26 of 52 patients with connective tissue disease. In our unpublished observations, an increase in systolic PAP of >20 mm Hg at EDE was recorded in 11 (17%) of 65 patients with systemic sclerosis. In all of the 11 patients with increased systolic PAP, subsequent right-sided heart catheterization (RHC) confirmed systolic PAP increase during exercise. In our opinion, EDE is a potentially useful test to detect “exercise-induced PH” that may represent an intermediate stage between a physiologic response and manifest PH.

Importantly, an elevated systolic PAP on exercise can be caused not only by pulmonary vasculopathy but also by a significant increase in pulmonary venous pressure. Among patients reported by Kovacs et al,\(^1\) RHC revealed an elevation of pulmonary arterial wedge pressure >20 mm Hg in 33% of subjects with exercise-induced PAP increase. In our group of 16 patients with systemic sclerosis (11 subjects with exercise-induced PH and five with PH at rest), RHC showed an elevated pulmonary arterial wedge pressure in 12 (75%) of them. These findings may suggest a latent left-ventricular filling dysfunction. Interestingly, these observations suggest that left-ventricular diastolic dysfunction in patients with connective tissue disease is more frequent than previously believed. Our data indicate that left-ventricular preload reduction using small doses of diuretics may be beneficial for such patients.

In our opinion, Kovacs et al\(^1\) are perfectly right that EDE is a useful, noninvasive method to detect systolic PAP increase during exercise in patients with connective tissue disease. However, it should be remembered that RHC still remains the golden standard for assessment of PH.