sneezing, urinating, defecating, or while eating. Fletcher and colleagues have examined a population of patients with moderate COPD who had no evidence on daytime arterial blood gas testing of significant hypoxemia. In all tested patients, arterial Po2 was greater than 60 mm Hg during daytime tests. When monitored by oximetry overnight, 27 percent of these patients showed desaturation below 90 percent for 5 minutes or more reaching a nadir saturation of 85 percent or lower. Do such transient events of hypoxemia warrant treatment with supplemental oxygen? The British MRC trial and multicentre NOTT trial which demonstrated improved survival in hypoxic COPD patients treated with oxygen established their treatment criteria based on arterial blood gas testing during wakeful hours. In our patient, arterial blood gas testing failed to show hypoxemia. When oximetry was used for continuous monitoring of oxygenation at a clinically relevant time, the hypoxemia was evident. Our patient's unusual presenting complaint offered a dramatic illustration of the possible clinical importance of transient hypoxic events. We would not attempt to generalize from this report to the treatment of all such patients. Nonetheless, the findings in the present case should encourage further investigation into the importance of even transient episodes of mild hypoxemia.

In our patient, weight loss was the direct consequence of meal-time hypoxemia. However, dysphagia must be a comparatively rare sequel of impaired oxygenation and it is not clear whether impaired gas exchange contributes to the malnutrition seen so commonly in other forms of chronic lung disease. Malnutrition and cachexia in COPD are generally attributed to the increased caloric needs arising from increased work of breathing. Is it possible that some patients with COPD become malnourished because of impaired oxygenation while attempting to eat? Evidence to support such speculation is scant and indirect. A recent nutritional survey of patients with obstructive lung disease found a closer correlation between weight loss and daytime blood gas values than between weight loss and measures of airways obstruction. As well, the correction of longstanding hypoxemia may result in weight gain. However, Brown and colleagues have examined this hypothesis more directly in a small group of patients with severe COPD. By monitoring oxygenation at meal times with oximetry, they confirmed that oxygen saturation falls when such patients eat. However, the mean decrease in saturation was just 1.8 percent, a decrement of doubtful physiologic importance. Moreover, despite identifying a subgroup of patients with more profound meal time desaturation, they could find no correlation between degree of desaturation and body weight.

In summary, we report what we believe to be the first reported instance of dysphagia presenting as a manifestation of hypoxemia. Continuous noninvasive monitoring by oximeter allowed detection of the transient hypoxic event, treatment with supplemental oxygen abolished the symptom and weight gain ensued. The case illustrates the usefulness of continuous noninvasive monitoring of oxygenation and the clinical importance of at least some transient hypoxic events.

**References**

8. Fletcher EC, Miller J, Divine GW, Fletcher JG, Miller T. Nocturnal oxyhemoglobin saturation in COPD patients with arterial oxygen tensions above 60 mm Hg. Chest 1987; 92:604-08

**Creatine Kinase MB Elevation in Paralytic Shellfish Poisoning**


An outbreak of paralytic shellfish poisoning occurred in southern Taiwan, affecting 116 persons who had consumed purple clams. Two victims died within four hours. Gonyautoxins were identified as causative toxins. During the outbreak, five patients with paralytic shellfish poisoning were seen in our hospital. All recovered following supportive treatments. Serum creatine kinase concentration was elevated in three of the five patients. The levels of the

*From the Department of Medicine, Chang Gung Memorial Hospital and Department of Health, Executive Yuan, Taipei, Taiwan, Republic of China.

Reprint requests: Dr. Hung, Department of Medicine, Chang Gung Memorial Hospital, Taipei, Taiwan, ROC 10550*
enzyme did not seem to correlate with the severity of poisoning. The most significant finding was the previously unreported observation of elevation of the creatine kinase MB level. In all four patients who had creatine kinase MB value determined, it was elevated.

(Chest 1991; 99:1032-33)

PSP = paralytic shellfish poisoning; CK = creatine kinase

Paralytic shellfish poisoning (PSP) is due to the ingestion of shellfish contaminated with one of several potent neurotoxins such as saxitoxin and gonyautoxin. Gonyautoxins are extremely potent, and even at very low concentrations they inhibit the sodium-potassium pump of nerve cells. The toxins are water soluble, heat and acid stable, and do not kill the shellfish. Humans ingesting the contaminated shellfish usually begin to have symptoms within 30 minutes. In severe cases, death occurs as a result of respiratory insufficiency. However, the overall mortality is low (<10 percent) and the recovery is usually complete.

CASE REPORTS

An outbreak of PSP occurred in southern Taiwan on January 1 and 2, 1986, affecting 116 persons who had consumed purple clams (Sanguinolaria rostrata). The clams were bought from the same commercial agriculture pond by retailers. The incubation period ranged from 30 minutes to seven hours (median, 1.5 hours). Illness lasted from one to nine days (median, one day). All but two victims, who died within four hours, recovered. Gonyautoxins types 1, II, III, and IV were identified by laboratories of Tokyo University, Japan, and Food and Drug Bureau, Department of Health of the Republic of China. During the outbreak, five patients were seen in our hospital. Clinical characteristics of the five patients are summarized in Table 1. Patient 1 was admitted to the intensive care unit after endotracheal intubation in the emergency department because of acute respiratory failure; he was discharged from the hospital after eight days. The other four patients (patients 2 through 5) were discharged from the emergency department several hours after supportive treatments. Electrocardiograms were normal in all patients. The results of serum creatine kinase (CK) levels are shown in Table 2. Mild elevation of the total CK level was observed in three of four patients (patients 2 through 5) about seven hours after ingestion of the clams. The CK MB level was elevated in all four patients. However, in the patient with the most severe poisoning, the total CK value was normal 24 hours after ingestion of the clams.

Table 1—Clinical Manifestations in Paralytic Shellfish Poisoning

<table>
<thead>
<tr>
<th>Patient No.</th>
<th>1</th>
<th>2</th>
<th>3</th>
<th>4</th>
<th>5</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age, yr/sex</td>
<td>34/F</td>
<td>37/F</td>
<td>35/M</td>
<td>34/M</td>
<td>32/M</td>
</tr>
<tr>
<td>Symptoms</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Circumoral paresthesia</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>+</td>
</tr>
<tr>
<td>Numbness of fingers and hands</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>+</td>
</tr>
<tr>
<td>Nausea and vomiting</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>+</td>
</tr>
<tr>
<td>Dyspnea</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>+</td>
</tr>
<tr>
<td>Respiratory failure</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>+</td>
</tr>
<tr>
<td>Recovery time, day</td>
<td>8</td>
<td>1</td>
<td>1/2</td>
<td>1/4</td>
<td>1/2</td>
</tr>
</tbody>
</table>

*M = male; F = female; plus sign = presence of symptom; and minus sign = absence of symptom.

Table 2—Serum Creatine Kinase (CK) in Paralytic Shellfish Poisoning

<table>
<thead>
<tr>
<th>Patient No.</th>
<th>1</th>
<th>2</th>
<th>3</th>
<th>4</th>
<th>5</th>
</tr>
</thead>
<tbody>
<tr>
<td>CK, IU/L*</td>
<td>14</td>
<td>190</td>
<td>155</td>
<td>52</td>
<td>227</td>
</tr>
<tr>
<td>BB, %†</td>
<td>-</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>MB, %†</td>
<td>-</td>
<td>41</td>
<td>10</td>
<td>8</td>
<td>13</td>
</tr>
<tr>
<td>MM, %†</td>
<td>-</td>
<td>59</td>
<td>90</td>
<td>92</td>
<td>87</td>
</tr>
</tbody>
</table>

* Determined using a UV kinetic method at 30°C; normal range, 5 to 89 IU/L.
† Analyzed by the electrophoretic method (Helena Laboratories).

The most significant finding in our study was elevation of the CK MB level in four of the five patients. To our knowledge, the elevation of the CK MB fraction has not been reported in PSP. Elevation of the CK MB fraction occurs in many clinical settings, including myocardial infarction, false interpretation of other substances as CK MB, such as "spilled-over" from the MM fraction, chest trauma, and others. Acute myocardial infarction was excluded in our patients by normal electrocardiograms and absence of the symptoms. The mechanism of CK MB release in our patients was not clear, but it could have been related to transient respiratory insufficiency and subsequent tissue hypoxia that led to release of the CK MB fraction from affected skeletal muscles. Several limitations in the study deserve comments. Only one blood sampling was made for CK analysis in each patient. The timing of CK measurements after clam ingestion was not uniform. Lastly, CK isoenzyme characterization was not made in one of the five patients. In future cases of PSP, serial blood samplings should be obtained to define the time course of CK enzyme elevation and its isoenzyme characterization.

ACKNOWLEDGMENTS: The authors wish to thank Shu-Ling Huang and Lu-Hung Chen of the Bureau of Food Sanitation, and Food and Drug Laboratory, Department of Health, the Executive Yuan, Taiwan, Republic of China, for providing us with the result of the toxins isolated from the specimen of the outbreak.

REFERENCES