BLOOD LACTATE AS A PROGNOSTICATOR OF SURVIVAL FOLLOWING 1/1
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BLOOD LACTATE AS A PROGNOSTICATOR OF SURVIVAL FOLLOWING HEMORRHAGE IN CONSCIOUS SWINE

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Arterial blood lactate concentration at the end of hemorrhage was evaluated as a predictor of survival following fixed volume hemorrhage in unmedicated chronically instrumented immature swine. Following hemorrhage lactate was elevated compared to basal values, $8.4 \pm 4.5$ mg/dL ($\mu$+SD) (n=52). Animals lived (n=71) with a lactate of $43.9 \pm 5.1$ mg/dL compared to $186.5 \pm 40.4$ mg/dL for animals that died (n=60). Lactate concentration at the end of hemorrhage successfully predicted survival (80.3%).

Hypovolemia and resultant hypotension, whether induced by fixed pressure (Wiggers model) or fixed volume hemorrhage, can lead to death. The incidence of death, however, can be highly variable, both within and between studies. This variability, furthermore, occurs in spite of apparent consistency in the design of experiments, unless the experiments are designed to assure either 100% survival or 100% lethality (1). Presumably, the diversity of response reflects differences in the capacity of individual animals to compensate for the functional consequences of hypovolemia.

Although a vast amount of information is available on the pathophysiologic changes that might contribute to death, a definitive explanation has not been identified (2). At the present time, therefore, no functional change has been identified as a predictor of survival or nonsurvival. In this report, we have pooled the arterial lactate values collected in a series of experiments with conscious pigs subjected to various degrees of fixed volume hemorrhage (1-5). Some of the animals survived the insult, others died. The intent of this effort was to determine whether arterial lactate level, measured at the end of hemorrhage, was an accurate predictor of outcome.

METHOD

Immature swine (18 to 30 kg) of either sex were studied, (n=52 controls; n=137 hemorrhaged; total n=189). Five to ten days before the study the animals were surgically prepared with arterial catheters to facilitate hemorrhage and to obtain blood samples. Some animals were splenectomized and others had additional catheters or flow probes implanted. Hemorrhage was initiated when the animals (n=137) were in the conscious resting state, and blood volume losses ranged from 15 to 54 ml/kg over periods of 10 to 60 minutes. Animals were enrolled in the study only if they received no medications and
### TABLE 1

Blood lactate concentration for various groups of swine

<table>
<thead>
<tr>
<th></th>
<th>Control</th>
<th>Hemorrhaged Lived</th>
<th>Hemorrhaged Died</th>
</tr>
</thead>
<tbody>
<tr>
<td>n</td>
<td>52</td>
<td>71</td>
<td>66</td>
</tr>
<tr>
<td>Lactate (mg/dl)</td>
<td>x</td>
<td>43.98*</td>
<td>106.54+</td>
</tr>
<tr>
<td>SD</td>
<td>4.52</td>
<td>37.14</td>
<td>40.43</td>
</tr>
<tr>
<td>range</td>
<td>1.2-24.9</td>
<td>2.6-161.0</td>
<td>25.8-230.4</td>
</tr>
</tbody>
</table>

*Significantly different (P<0.05) from control animals.

+Significantly different (P<0.05) from control and hemorrhage-lived animals.

### TABLE 2

Evaluation of predictive outcome by classification

<table>
<thead>
<tr>
<th>Predicted Die</th>
<th>Predicted Live</th>
<th>Percent Correct</th>
</tr>
</thead>
<tbody>
<tr>
<td>Died</td>
<td>55</td>
<td>83.3</td>
</tr>
<tr>
<td>Lived</td>
<td>16</td>
<td>77.5</td>
</tr>
<tr>
<td>Total</td>
<td>71</td>
<td>80.3</td>
</tr>
</tbody>
</table>
blood lactate level has been suggested as a prognosticator for survival in trauma patients (6-9). However, the prognostic significance of lactate concentration has had to be determined specifically for each disease (9). We have evaluated plasma lactate concentrations obtained at the end of hemorrhage as a predictor of survival or death in conscious swine. While 80% effective in predicting survival in the swine hemorrhage model, blood lactate may not be the sole factor determining outcome. The use of blood lactate may in part replace the 100% lethal exsanguination swine model allowing a more mechanistic evaluation of shock therapies.
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