I. General changes in postmortem muscle

A. pH
   1. Decreases rapidly early postmortem.
   2. Primarily due to increase in intracellular lactate.

B. ATP (“acid-labile phosphorus”) and creatine phosphate.
   \[ \text{ADP + creatine-phosphate} \xleftrightarrow{\text{creatine kinase}} \text{ATP + creatine} \]
   1. ATP decreases slowly, creatine-phosphate decreases rapidly in postmortem muscle.
   2. Short-term means of regenerating ATP.

C. Glycogenolysis
   1. Elevated in response to decreased ATP, G-6-P and elevated AMP, P_i
   2. Represents the last-ditch attempt of muscle to survive.

D. Extensibility
   1. Decreases early postmortem.
   2. Caused by the formation of rigor bonds.

---

**Figure 4-7.**

Chemical and physical changes in muscle during development of rigor mortis. Values are for beef sternomandibularis muscle held at 37°C (99°F). Extension changes were recorded by an apparatus similar to the one described by Bate-Smith and Bendall (1949) using a load of about 60 g/cm² and a loading-unloading cycle of eight minutes on and eight minutes off. Zero time = 1 hr 45 min post-mortem. (Newbold, 1969.)

II. Fast-glycolyzing versus slow-glycolyzing muscle
A. G-1-P, G-6-P, F-6-P
   1. Greater change in concentration in fast-glycolyzing.
   2. Indicates contribution from glycogen, inhibition at PFK.

B. F-16-P₂, triose-phosphates
   1. Lower change in concentration in fast-glycolyzing.
   2. Indicates more inhibition at PFK in fast-glycolyzing.
C. Crossover diagrams

1. Provide a comparison of two physiological states.
2. Indicate controlling or rate-limiting actions.

![Crossover diagram](image)

**Fig. 35.3.** Crossover plot of glycolytic intermediate levels observed in fast- and slow-glycolyzing muscles at 15 min post mortem. Calculated from mean values of 22 longissimus muscles with “slow” post mortem glycolysis and 15 muscles with “fast” post mortem glycolysis. Values found in slow-glycolyzing muscles are considered to be 100%.

III. Metabolism in non-stimulated versus electrically stimulated muscle

A. Electrical stimulation

1. Passing DC, pulsatile current through a carcass.
2. Increases tenderness of meat unless the meat already is tender.

B. Mechanism of action

1. More rapid pH decline.
2. Indicates more rapid glycolysis.

C. Crossover diagram (ES vs. non-ES)

1. Greater change in concentration of glucose, G-1-P, G-6-P, F-6-P, lactate in ES muscle
2. No true “crossover” at F-1,6-P₂, hence regulation is not at PFK.
3. Elevated glycogenolysis
   a. Elevation of Ca$^{++}$ increased phosphorylase kinase activity.
   b. Decreased ATP increased phosphorylase activity.
   c. Both suggest increased muscle contraction.

Relationship between the amount of lactate and the resulting pH in non-stimulated (O) and electrically stimulated (☐) muscles.

Fig. 5. Cross-over plot of some energy metabolites at 0.25, 6 and 24 h post mortem in electrically stimulated muscles in relation to the non-stimulated counterpart (100%). Abbreviations given in the 'Materials and Methods' section. * Almost significant difference ($P < 0.05$). ** Significant difference ($P < 0.01$).
IV. pH and 6-phosphofructokinase activity

A. 6-PFK activity in mouse muscle

1. Mouse 6-PFK activity declines sharply with pH.
2. The decrease in pH abolishes affinity of PFK for F6P.
3. This causes an apparent reduction in F6P binding sites (i.e., V_{max}).

B. 6-PFK activity in postmortem sternomandibularis muscle

1. Bovine muscle 6-PFK activity declines sharply with pH.
2. 6-PFK activity also is depressed during 4 days of postmortem storage.
3. Both pH and postmortem storage cause an apparent reduction in V_{max}.

Fig. 1. 6-PFK enzyme activity in *M. beef sternocephalicus pars mandibularis* muscle. Activity was measured at 30 min postmortem (d 0) at pH 7.4 or 7.0, and at d 4 at pH 7.4. Data are means of four replicates for each day postmortem and pH. Inset: 6-PFK activity of d 0, pH 7.0 and d 4, pH 7.4 samples. The y-axis has been expanded to indicate the hyperbolic nature of 6-PFK activity in these samples.
C. Glycolytic intermediates in sternomandibularis muscle
1. Glycogen levels decline about 60%.
3. Lactate concentrations increase 30-fold.

D. Crossover diagram
1. Crossover at phosphorylase indicates stimulation of its activity during postmortem storage.
2. A second crossover at 6-PFK reflects the depression of its activity.

E. Glucose metabolism and pH
1. The conversion of $^{14}$C-labeled glucose to lactate is strongly depressed by lowering the pH.
2. Glycogen synthesis and CO$_2$ production were not affected by pH.

Table 1
Least squares means for concentrations of glycolytic metabolites of beef *M. sternocaphealis pars mandibularis* muscles analyzed immediately post-exsanguination or after 4 d postmortem

<table>
<thead>
<tr>
<th>Metabolite</th>
<th>Day postmortem</th>
<th>SEM</th>
<th>$P &gt; F$</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>0 d</td>
<td>4 d</td>
<td></td>
</tr>
<tr>
<td>Glycogen</td>
<td>86.7</td>
<td>39.6</td>
<td>9.64</td>
</tr>
<tr>
<td>Free glucose</td>
<td>0.84</td>
<td>6.54</td>
<td>0.35</td>
</tr>
<tr>
<td>Glucose-6-phosphate</td>
<td>1.65</td>
<td>4.57</td>
<td>0.47</td>
</tr>
<tr>
<td>Fructose-6-phosphate</td>
<td>0.40</td>
<td>1.90</td>
<td>0.21</td>
</tr>
<tr>
<td>Fructose-1,6-bisphosphate</td>
<td>0.06</td>
<td>0.03</td>
<td>0.03</td>
</tr>
<tr>
<td>Glyceraldehyde-3-phosphate + dihydroxyacetone phosphate</td>
<td>0.46</td>
<td>0.43</td>
<td>0.13</td>
</tr>
<tr>
<td>Lactate</td>
<td>3.33</td>
<td>45.9</td>
<td>1.09</td>
</tr>
</tbody>
</table>

Fig. 3. Crossover diagram for glycolytic metabolites of beef *M. sternocaphealis pars mandibularis* muscle sampled at d 0 and d 4 postmortem. Values are the ratios of (concentrations at d 4/concentrations at d 0) $\times$ 100. Data are means of six replicates.

Table 2
Least squares means for rates of conversion of [U-$^{14}$C]glucose to lactate, glycogen, and CO$_2$ in beef *M. sternocaphealis pars mandibularis* muscles incubated at pH 7.4 or 7.0

<table>
<thead>
<tr>
<th>Product</th>
<th>Incubation pH</th>
<th>SEM</th>
<th>$P &gt; F$</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>7.4</td>
<td>7.0</td>
<td></td>
</tr>
<tr>
<td>Lactate</td>
<td>84.7</td>
<td>25.5</td>
<td>12.1</td>
</tr>
<tr>
<td>Glycogen</td>
<td>23.7</td>
<td>16.5</td>
<td>0.8</td>
</tr>
<tr>
<td>CO$_2$</td>
<td>14.7</td>
<td>13.4</td>
<td>0.8</td>
</tr>
<tr>
<td>Total product formation</td>
<td>123.3</td>
<td>55.5</td>
<td>16.8</td>
</tr>
</tbody>
</table>

Percent total product formation

<table>
<thead>
<tr>
<th>Product</th>
<th>SEM</th>
<th>$P &gt; F$</th>
</tr>
</thead>
<tbody>
<tr>
<td>Lactate</td>
<td>66.4</td>
<td>10.9</td>
</tr>
<tr>
<td>Glycogen</td>
<td>20.9</td>
<td>7.3</td>
</tr>
<tr>
<td>CO$_2$</td>
<td>12.5</td>
<td>4.6</td>
</tr>
</tbody>
</table>
II. Postmortem glycolysis in bovine longissimus muscle

A. Muscle glycogen declined to about one-third of initial values.

D. F6P increases, indicating inhibition at 6-PFK.

B. Glucose increases over 5-fold, caused by debranching of glycogen.

E. Lactate increases 3-fold, which causes the decline in pH.

C. G6P increases as F6P increases, which would inhibit hexokinase activity.

F. The pH declines to 6.75 by 4 h postmortem.