OBSERVATIONS ON SWAYBACK DISEASE OF LAMBS.

VOLUME II.

R. M. BARLOW.

CONTENTS

TABLES

GRAPHS

FIGURES

APPENDIX A

APPENDIX B

REFERENCES

REPRINTS
## Table I

### General Survey Data on Affected Farms

<table>
<thead>
<tr>
<th>Farm</th>
<th>Average Height (ft.)</th>
<th>Type</th>
<th>Aspect</th>
<th>Geology</th>
<th>Pasture Improvements</th>
<th>Breeds of lambs</th>
<th>Incidence of Swayback</th>
<th>Supplement Cu</th>
<th>Mean Ewe Blood Cu μgms/ 100 mls.</th>
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<tr>
<td>A</td>
<td>700</td>
<td>Marginal</td>
<td>S – SE</td>
<td>Carboniferous Limestone</td>
<td>Ploughed, reseeded and limed</td>
<td>Blackface, Greyface, Other crosses</td>
<td>2–3% 50% &lt;1%</td>
<td>Nil</td>
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<tr>
<td>B</td>
<td>500</td>
<td>Marginal</td>
<td>NW</td>
<td>Do.</td>
<td>Limed 5 tons/acre every 5 years</td>
<td>Greyface</td>
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<td>C</td>
<td>550</td>
<td>Marginal/ Hill</td>
<td>NE</td>
<td>Do. Quartz dolerite</td>
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<td>Do.</td>
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<td>600</td>
<td>Do.</td>
<td>S</td>
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<td>Do.</td>
<td>&lt;0.5% 25% 0%</td>
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<td>E</td>
<td>1000</td>
<td>Do.</td>
<td>S</td>
<td>Do. Quartz dolerite</td>
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<td>Sandstone</td>
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<td>Greyface</td>
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<td>Nil</td>
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<td>S</td>
<td>Clay and Peat</td>
<td>–</td>
<td>Greyface</td>
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<td>Breeds of lambs</td>
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<td>H</td>
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<td>S</td>
<td>Pyroxine-andesite</td>
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<td>0%</td>
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<td>O.R. sandstone</td>
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<td>O.R. sandstone</td>
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<td>L</td>
<td>825</td>
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<td>-</td>
<td>Cornstone</td>
<td>Gradual ploughing and reseeding Limed 3 tons/acre as necessary</td>
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<td>M</td>
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<td>Lowther shales and clay</td>
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<td>0</td>
<td>Hill</td>
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<td>O.R. sandstone</td>
<td>Reseeded and limed 4 tons/acre 1953 2 tons/acre 1955</td>
<td>Cheviot</td>
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<td>S</td>
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<td>Q</td>
<td>Do.</td>
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<td>Do.</td>
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<td>0%</td>
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<td>S</td>
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<td>12</td>
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<td>Carboniferous Limestone Basalt</td>
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<td>10-12</td>
<td>-</td>
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The figures in parenthesis in column 12 refer to the number of ewes sampled. The sign ± is used to denote those farms where mineral licks were available which contained only traces of copper.
The broken line represents the ground temperature 1 foot below the surface.

**TABLE II**

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TABLE III
TABLE IV

Spectrographic Analysis of Farms in Swayback Survey

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<th>Farm &amp; Field No.</th>
<th>Soil pH</th>
<th>Date of Sampling</th>
<th>SOIL (p.p.m.)</th>
<th>HERBAGE (p.p.m.)</th>
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TABLE IV (contd.)

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<th>Farm &amp; Field No. and Date of Sampling</th>
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<tr>
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Farms G, K, N, O, P, Q, R, S, and T were not sampled.
### TABLE V

Blood and Liver Copper Values: Swayback Lambs and their Mothers

<table>
<thead>
<tr>
<th>FARM CASE NO.</th>
<th>AGE of LAMB</th>
<th>Lamb Liver Cu (p.p.m.)</th>
<th>Lamb Blood Cu μgms/100 ml.</th>
<th>Ewe Blood Cu μgms/100 ml</th>
<th>Bottle Fed</th>
</tr>
</thead>
<tbody>
<tr>
<td>A. 465/4/57</td>
<td>24 hrs.</td>
<td>3.2</td>
<td>-</td>
<td>4.6</td>
<td>-</td>
</tr>
<tr>
<td></td>
<td>480/1/57</td>
<td>4.8</td>
<td>-</td>
<td>10.7</td>
<td>-</td>
</tr>
<tr>
<td>B. 832/1/57</td>
<td>4-7 days</td>
<td>4.0</td>
<td>-</td>
<td>-</td>
<td>+</td>
</tr>
<tr>
<td></td>
<td>832/2/57</td>
<td>2.5</td>
<td>-</td>
<td>-</td>
<td>+</td>
</tr>
<tr>
<td></td>
<td>832/3/57</td>
<td>3.1</td>
<td>-</td>
<td>-</td>
<td>+</td>
</tr>
<tr>
<td>C. 903/1/57</td>
<td>10 days</td>
<td>-</td>
<td>-</td>
<td>9.1</td>
<td>+</td>
</tr>
<tr>
<td></td>
<td>966/1/57</td>
<td>2.8</td>
<td>-</td>
<td>6.8</td>
<td>-</td>
</tr>
<tr>
<td>D. 860/2/57</td>
<td>24 hrs.</td>
<td>7.1</td>
<td>-</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td></td>
<td>1218/1/57</td>
<td>2 wks.</td>
<td>17.5</td>
<td>65.5</td>
<td>-</td>
</tr>
<tr>
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<td>7.9</td>
<td>23.2</td>
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</tr>
<tr>
<td>E. 859/1/57</td>
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<td>4.13</td>
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<td>-</td>
<td>-</td>
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<td>6.2</td>
<td>-</td>
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<td>929/3/57</td>
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<td>-</td>
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<td>929/4/57</td>
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<td>-</td>
<td>7.64</td>
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<td>984/1/57</td>
<td>24 hrs.</td>
<td>7.72</td>
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<td>984/2/57</td>
<td>24 hrs.</td>
<td>6.7</td>
<td>-</td>
<td>(?) contaminated</td>
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<td>984/3/57</td>
<td>5-6 days</td>
<td>8.5</td>
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<td>1201/1/57</td>
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<td>1217/1/57</td>
<td>3-4 wks.</td>
<td>1.8</td>
<td>35.9</td>
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<tr>
<td>F. 1026/1/57</td>
<td>2 days</td>
<td>7.84</td>
<td>-</td>
<td>32.3</td>
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<tr>
<td></td>
<td>1026/2/57</td>
<td>1 wk.</td>
<td>6.96</td>
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<td>G. 1058/1/57</td>
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<td>-</td>
<td>-</td>
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<td>H. 1110/1/57</td>
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<td>8.18</td>
<td>-</td>
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<tr>
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<td>1129/1/57</td>
<td>3 wks.</td>
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<td>6.8</td>
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TABLE V (contd.)

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<td>I. 1109/1/57</td>
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<td>540</td>
<td>-</td>
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<td>+</td>
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<td>-</td>
<td>17.7</td>
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<td>K. 1234/1/57</td>
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<tr>
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<td>17.7</td>
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<td>-</td>
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<td>(130)</td>
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<td></td>
<td></td>
<td>at different centre</td>
<td></td>
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<tr>
<td>N. 1520/1/57</td>
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<td>106</td>
<td>-</td>
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<td>1520/2/57</td>
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<td>-</td>
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<td>Q.</td>
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<td>15.3</td>
<td>-</td>
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<td>R. 1360/1/58</td>
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<td>8 wks.</td>
<td>76.8</td>
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+ Indicates bottle feeding.
TABLE VI
Farm E - Blood Copper Values of Ewes 1956-57

<table>
<thead>
<tr>
<th>No.</th>
<th>When Bought</th>
<th>Lamb Crop (1957)</th>
<th>Blood Cu (μg/m/100 ml.)</th>
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<td>10/12/57</td>
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<td>2</td>
<td></td>
<td>1</td>
<td>91.4</td>
</tr>
<tr>
<td>3D</td>
<td></td>
<td>1</td>
<td>74.1</td>
</tr>
<tr>
<td>4/39</td>
<td></td>
<td>1</td>
<td>10.9</td>
</tr>
<tr>
<td>5D</td>
<td>Oct. 1956</td>
<td>1</td>
<td>66.4</td>
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<td>7D</td>
<td></td>
<td>1</td>
<td>82.3</td>
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<td>6</td>
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<td>10D</td>
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<td>10.5</td>
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<td>13</td>
<td></td>
<td>3 or 4</td>
<td>59.1</td>
</tr>
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<td>14D</td>
<td></td>
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<td>5.0</td>
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<td>15</td>
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<td>5</td>
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<td>28.2</td>
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<td>45.0</td>
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<tr>
<td>25</td>
<td>1955</td>
<td>6</td>
<td>25.9</td>
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</table>

D - Dosed 1 gm. CuSO₄·SH₂O on 10/12/56, 5/2/57 and 22/3/57.

* - Gave birth to swayback lamb 1957.
<table>
<thead>
<tr>
<th>No.</th>
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<th>3</th>
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<td>27.7</td>
<td>39.1</td>
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<td>54.1</td>
<td>64.1</td>
<td>18.2</td>
<td>41.2</td>
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</table>

Farm X - Blood copper values of random group of Ewes - 5.2.57

Blood Cu (μgms./100mls.)
TABLE VIII

Seasonal variations of Blood copper levels and the effect of dosing with CuSO₄ or injecting Copper '45'

(μgms/100 mls.)

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### TABLE VIII (contd.)

**FARM D (contd.)**

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<tbody>
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<td>14</td>
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<td>38.1</td>
</tr>
<tr>
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<td>1953</td>
<td>32.1</td>
<td>21.7</td>
<td>49.1</td>
<td>34.5</td>
</tr>
<tr>
<td>19</td>
<td>1954</td>
<td>28.9</td>
<td>25.5</td>
<td>49.1</td>
<td>47.0</td>
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<td>36</td>
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<td>28.5</td>
<td>Died</td>
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<tr>
<td>10</td>
<td>1955</td>
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<td>32.3</td>
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<td>65.5</td>
<td>75.9</td>
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**Untreated**

- Bought June 1956
- Died June 1957

**Mean**

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<tbody>
<tr>
<td></td>
<td></td>
<td></td>
<td>50.8</td>
<td>36.4</td>
<td>54.2</td>
</tr>
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</table>

**Dosed group** - dosed 1.0 gm. CuSO$_4$ 5H$_2$O

- Dec. 5th, Jan. 16th,
- Feb. 18th, Mar. 20th.

**Injected group** - injected Copper '45' Jan. 16th.

### FARM E

<table>
<thead>
<tr>
<th>No.</th>
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<td>5^E</td>
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<td>74.2</td>
<td>55.5</td>
<td>71.9</td>
</tr>
<tr>
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<td>1954</td>
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<td>72.4</td>
<td>74.2</td>
<td>62.1</td>
<td>68.7</td>
</tr>
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<td>1953</td>
<td>61.1</td>
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<td>-</td>
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<td>58.5</td>
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<tr>
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<td>1952</td>
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<td>64.0</td>
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<td>-</td>
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**Mean**

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**FARM E (contd.)**

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* - Dosed with 3 x 1.0 gm. CuSO₄ 5H₂O 1956/57.

S - Swayback lamb 904/58 on 10.4.58 - blood value then 2.3 µgms/100 mls.

Injected Group - Injected Copper '45' Jan. 16th and Mar. 20th.
<table>
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<td>Changes in blood copper levels following introduction to a swayback farm</td>
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**FARM D** - Gimmers bought October 1957 (untreated)  
(μgms/100 mls.)

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**FARM E** - Gimmers bought October 1957 (untreated)

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TABLE X
Selective Grazing Experiment

FARM M.  Initial blood copper values of sheep gms/100 mls.

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SKETCH MAP SHOWING DISTRIBUTION OF SWAYBACK FARMS.

Figure 1
Seasonal Variations in Blood Copper—Ewes

(Farms D, and E)

(Figure 2.)
Seasonal Variations in Blood Copper—
Untreated Gimmers (Farms D, and E)

(Figure 3)
DRAWING OF SHEEP BRAIN

to show sites from which standard blocks were taken.

Only the solid lines represent incisions.

(Figure 4)
Portion of cerebral cortex of a foetus, crown-rump length 46 cms. Note the degree of cellular differentiation. Occasional neurons show vacuolation and chromatolysis. (H & E x 440).

Figure 5(a).
Cortex adjacent to a gelatinous lesion. The "cell density" is less than in the 46 cm. foetus (Fig. 5a). There is little differentiation of cell types. Compare with Fig. 8(b).

(H & E x 440)

Figure 5(b).
357/58 Cerebral white matter of a foetus crown-rump length 46 cms. The oedematous nature of the tissue is apparent. (H & E x 440).

Figure 6(a).
1026/57 Gelatinous lesion. There is a relative elimination in the numbers of small, darkly-staining nuclei. Compare with Figure 6(a).

(H & E x 180).

Figure 6(b).
357/58 Subcortex of 46 cms. foetus showing the glial stroma, and occasional axis cylinders.
(Peters x 440).

Figure 7(a).
Cerebral white matter of 46 cms. foetus stained for myelin. Fine myelin tubes, and globules may be seen. There is progressive diminution in the numbers of stained fibre towards the cortex.

(Frozen section Spielmeyer x 150).

Figure 7(b).
1026/57 Gelatinous lesion, cerebral white matter. Some axis cylinders present but many appear fragmented. The clear spaces contain the swollen cell bodies of astrocytes. Compare with Figure 7(a).

(Peters x 440).

Figure 7(c).
Subcortex of normal lamb, 18 hours old. Cells with small, darkly-staining nuclei and little apparent cytoplasm (microglia) are very numerous. Astrocytes are present, and "paired cells" are not uncommon.

(H & E x 140).

*Figure 8(a).*
2390/57 Portion of cortex of normal lamb 18 hours old. The tissue is cellular and shows a high degree of differentiation. Satellite cells are numerous.

(H & E x 247).

Figure 8(b).
L.S. lateral funiculus normal animal. Tissue obtained by bulk fixation. A chain of Marchi positive globules is seen crossing the field. Compare with Figures 9(b) and (c).

(Glass x 90).

Figure 9(a).
791/58  L.S. spinal cord of severe case. Tissue obtained by bulk fixation. Frequently Marchi positive fibres were much less numerous. (Glees x 90)

*Figure 9(b).*
611/58  L.S. Normal spinal cord. Tissue obtained by routine methods. Showing some of the artifacts which may be produced. The focal accumulation beneath the meninges probably represents a group of stretched fibres in a nerve root. (Glees x 90).

Figure 9(c).
791/58 T.S. Spinal cord Swayback. Same case as 9(b). Marchi positive fibres are very numerous in the dorsal part of the lateral funiculus. See also Figures 22 and 23.

(Glees x 30).

Figure 9(d).
1058/57 Transverse section of cerebrum showing gross cavitation. The fine strands are composed of glia, axons, a few myelinated fibres and occasional small blood vessels.

(Actual size).

Figure 10(a).
1026/57 Transverse section of cerebrum showing the gelatinous lesion in the white matter.  

(Actual Size).

*Figure 10(b).*
1218/2/57 Renal cortex showing early glomerular lesion. Epithelialisation of Bowmans capsule with stratified cuboidal cells. Little periglomerular fibrosis is present in this instance. (H & E x 440).

Figure 11.
2369/57 Late stage glomerular lesion showing pronounced atrophy of the tuft. Dilatation of the juxtaglomerular tubule was not uncommonly associated with lesions of this type.

(H & E x 440).

Figure 12.
2023/57 Gluteal muscle left side. Small size of fibres and proliferation of nuclei is evident. The muscle from the right side is shown in Figure 13(b) for comparison.

(H & E x 240).

Figure 13(a).
2023/57  Gluteal muscle right side. 
Compare with Figure 13(a). 
(H & E x 240). 

Figure 13(b).
Oedema of the pia-arachnoid of the cerebrum with slight cellular proliferation.

(H & E x 60).

Figure 14.
Proliferating feltwork of fibrous glia at the periphery of a cavity; exceptional case. It was unusual to find such an active repair process. More commonly the peripheral glia appeared to be in the form of a compression capsule. See Figure 15(b).

(Frozen Section Cajal x 240).

Figure 15(a).
791/58 Gelatinous lesion exceptional case. Paired nuclei indicating glial proliferation are not uncommon. (Phosphotungstic acid haematoxylin x 440).

Figure 15(b).
Peripheral region of a cavity. The glial fibres tend to run parallel to the surface giving a laminated appearance, suggestive of a compression capsule. This was the type most commonly found.

(Phosphotungstic acid haematoxylin x 247).

Figure 15 (c).
795/58 Gelatinous lesion; exceptional case. Numerous histiocytes are present together with proliferating glia. (H & E x 440).

Figure 16.
Edge of a gelatinous lesion showing a gradual increase in the number of myelinated fibres towards the periphery. A dilated perivascular space is present in the top right hand corner.
(Frozen section Spielmeyer x 240).

Figure 17.
Severe chromatolysis, thalamus, The affected cell is grossly swollen, the cytoplasm having an eosinophilic hyaline appearance. A remnant of the nucleus is situated peripherally. A thin rim of Nissl substance persists. (H & E x 440).

Figure 18.
Chromatolysis of large neurons; thalamus. The affected cell is swollen and contains no Nissl granules. (Spoerri x 440).

Figure 19.
Chromatolysis of large neurons; thalamus. Affected cells show disruption of the neuro fibrillary apparatus which undergoes a dust-like degeneration or forms short rods, peripherally situated. (Holmes x 440).

Figure 20.
Diagram to show the Areas of the Medulla in which severe Chromatolysis has been observed

a) Region of the Pons.

- Vestibular Nuclei.
- Facial Nucleus.
- Ambine Nucleus.
- Reticular Formation.
- Trapezoid Nucleus?

b) Just Caudad to the Obex

- Hypoglossal Nucleus.
- Reticular Formation.
- Nucleus Ambigus.

Generalised distribution pattern of Marchi positive fibres in a T. S. of the Spinal Cord

Figure 21.
795/58  T.S. spinal cord showing demyelination in the lateral funiculus. (Frozen section Spielmeyer x 30).

*Figure 22(a).*
795/58 L.S. spinal cord. Degenerating myelin tubes in dorsal part of lateral funiculus.
(Frozen Section Spielmeyer x 440).

Figure 22 (b).
795/58  T.S. Thoracic cord showing distribution of Marchi positive fibres. Bulk fixation. (Frozen section Glees x 12).

*Figure 23(a).*
984/57  T.S. Lumbar cord showing distribution of Marchi positive fibres. Bulk fixation.
(Frozen section 3 lees x 16);

Figure 23(b).
T.S. Thoracic cord, Normal lamb, showing distribution of Marchi positive material. Routine fixation. In transverse section it is not possible to differentiate between myelin degeneration and artifact. Compare with 23(a) and (b).

(Frozen section Glees x 14).

Figure 23(c).
L.S. Lateral funiculus of spinal cord. Case with moderately severe cerebral lesions. Stained to show axons. Some myelin tubes also stain. Compare with 24(b) and (c).

(Holmes x 247).

Figure 24(a).
984/2/58 Lateral funiculus of spinal cord from a case with no evident cerebral lesions. Compare with Figure 24(a). (Holmes x 247).

Figure 24(b).
2390/2/58 Lateral funiculus of spinal cord from a normal lamb. Compare with Figures 24(a) and (b). (Holmes x 247).

Figure 24(c).
1110/57 Chromatolysis, ventral horn cells, spinal cord. (H & E x 440).

Figure 25(a).
Chromatolysis, ventral horn cells, spinal cord.

(Holmes x 440).

Figure 25(b).
APPENDIX A

Being a summary of data collected from the farms in the survey. The positions of the farms are marked on the map (Fig. 4). Geological data was abstracted from the maps of the Geological Survey Scotland, (Solid and Drift Editions, Scale 1 inch = 1 mile).

Farm A (1957)

650-750' above sea level on a southern extension of the Riccarton Hills.

Higher pasture is on microporphytic olivine basalt (of carboniferous limestone age). Below the house the formation is sedimentary carboniferous limestone.

Pasture grazed has a S. - S.E. aspect.

Management: Land poor and heavy, much ploughed and reseeded, limed 2 tons/acre in last 2-3 years. Greyface ewes x Suffolk. Rotational grazing over whole farm in winter.

History of Swayback: Occasional cases prior to 1957, in which year 17/20 of first lambs affected at birth.

Average Incidence: 2-3%.
Incidence 1957: 50%.

Maternal blood Cu values (random): 4.6, 21.5, 14.1, 5.5, 3.5, 47.5, 5.6, 31.3 μgms/100 mls.
Farm B (1957 and 1958)

500' above sea level. S.S.E. Cowdenbeath. Lower limestone group of carboniferous limestone series.

Pasture has slight N.W. aspect.


History of Swayback: Isolated cases prior to 1957, in which year 50 lambs lost. Complicated by necrobacillosis.

Average Incidence: 0.3%.
Incidence 1957: 20%. (1958 - 2 cases.)
Maternal blood Cu values (random): 27.7, 15.0, 8.6, 38.0, 69.0 gms/100 mls.

Farm C (1957)

400-500' above sea level. N.W. Riccarton Hills.

High pasture on contemporaneous basalt; lower ground quartz dolerite and boulder clay.

Pastures grazed had N.E. aspect.


History of Swayback: None. 6 cases 1957.

Average Incidence: Nil.
Incidence 1957: 20%.
Maternal bloods (swayback mothers): 9.13, 6.18 gms Cu/100 mls.

Farm D (Experimental farm 1957-58)
600' above sea level. N. of Leslie.
Mainly quartz dolerite. Easterly fields, carboniferous limestone.
S. aspect.
Management: Marginal/hill, greatly improved.

Dressed annually with artificials. Limed 5 tons/acre every 5 years. Good B.F. stock ewes (purchases as gimmers). Tupped B.L.

History of Swayback: A few cases every year for six or seven years. 1957: 18/20 first born lambs affected.
Average Incidence: 0.5%.
Incidence 1957: 25%.
Maternal blood Cu values: 49.1, 35.5, 16.8 gms/100 mls.

Farm E (Experimental farm 1956-58)
1000' above sea level. N. Leslie.
Mainly quartz-dolerite, patches of carboniferous limestone.
S. aspect.
Management: Marginal/hill, little arable ground.
Intensive improvement - reseeding, artificials. Limed 10 tons/acre every 3-5 years for last 10 years.

History of Swayback: Gradually increasing incidence over last 10 years. (25% - 1956.)

Average Incidence: 4-5%.
Incidence 1957: 25% (control group).
Maternal blood Cu values: 14.5, 7.64, 10.0, 35.9 \( \mu \text{gms} / 100 \text{ mls} \).

Farm F (1957)

600-900'. E. of Humbie.

Mainly old red sandstone; high ground analcite basalts and lower ground calciferous sandstone.
N.W. aspect.


History of Swayback: Nil.
Average Incidence: Nil.
Incidence 1957: 2%.
Maternal blood Cu values: 32.3 (3 years on farm), 5.5 (8 months on farm) \( \mu \text{gms} / 100 \text{ mls} \).

Farm G (1957)

500-600' above sea level. 8 miles S. Bathgate.

Boulder clay and peat.
S. aspect.
Management: Poor marginal/arable land. Cast B.F. ewes bought annually. Tupped B.L.

History of Swayback: Occasional cases some years.
Average Incidence: 1%.
Incidence 1957: 2.3%.
No ewe blood samples obtained.

Farm H (1957)
800-900' above sea level. 2½ miles N. Biggar.
Pyroxine-andesite of old red sandstone age.
Gentle southerly aspect.

History of Swayback: None until 1957 when 6 cases occurred - 2 at birth, 4 in first 3 weeks of life.
Average Incidence: Nil.
Incidence 1957: 4%.
Maternal blood Cu values (swayback mothers):
8.18, 6.8 μgms/100 mls.

Farm I (1957)
800-1150' above sea level. 3½ miles S.E. Biggar.
Hill; lowther shales; lower ground boulder clay and radiolarian cherts.
General northerly aspect.
Management: Parkland/hill. Good hill flock and two park hirsels of Blackfaces.

History of Swayback: None until 1957 when 3 cases occurred in one park hirsel (grazing land not limed for 3 years). Sheep fed a mineral mixture containing 900 p.p.m. Cu mixed 5 lbs./cwt. of ration. Ration fed at rate of 1 lb/day during last 6 weeks of pregnancy.

Average Incidence: Nil.

Incidence 1957: 1%.

Maternal blood copper value: 10.9 µgms/100 mls.

Farm J (1957)

800' above sea level. 2 mls. S.W. West Linton. Lower old red sandstone conglomerates, peat and gravel.

General southwesterly aspect.


History of Swayback: None prior to 1957 though hypocuprosis in calves in 1956. Offspring of 5 ewes affected. Developed signs at 2-3 weeks old.

Average Incidence: Nil.

Incidence 1957: 1.5%.

Maternal blood Cu levels (swayback mother): 17.7 µgms/100 mls.
Farm K (1957)

1000-1260' above sea level. 2½ mls. N. Lauder.
Upper old red sandstone.
Generally S. aspect.
Management: Good hill. Hirsels of 200 B.F. ewes.

History of Swayback: None prior to 1957 when 4 lambs affected in one hirsel limed at 3 tons/acre 1956. 2 cases were mild and appeared at 6 weeks after lambing.

Average Incidence: Nil.
Incidence 1957: 1.5%.
Maternal blood Cu value (swayback mother): 7.3 µgms/100 mls.

Farm L (1957)

800-850' above sea level. 1 ml. W. of Walston.
Cornstone and boulder clay.
Management: Marginal/arable. Progressive removal of heather and bracken with reseeding since 1941. Limed every 3-4 years at 3 tons/acre. Salt licks provided but containing insignificant Cu.
Blackface.

History of Swayback: One or two cases 1956. 20 lambs affected 1957, 10 dying in first 48 hrs. Others acutely affected at 3-4 weeks of age.

Average Incidence: 0.3%.
Incidence 1957: 7%.
No maternal blood samples obtained.

Farm M (1957)
700-1334' above sea level. 3 miles W. of Peebles.
Lowther shales, boulder clay in valley.
S. aspect.
Management: Hill ground in 2 hirsels, one of which is claimed to be "bad" (cattle don’t thrive, lambs affected with swayback). Limed 6-7 years ago.
Blackface ewes x B.F. and B.L.

History of Swayback: Occasional cases prior to 1957, when 4-5 lambs affected on "bad" hirsel.

Average Incidence: 1%.
Incidence 1957: 3%.
Maternal blood Cu values (random): 21.8, 7.3, 16.3 gms/100 mls.

Farm N (1957)
100-200' above sea level.
Clay and sandstone, cement, stones.
Management: Arable rotation, some hill grazing.
Blackface ewes tupped. Blackface for replacement stock, B.L. for fat lambs.

History of Swayback: Occasional cases. 4 in 1957, affected 8-10 weeks old (mild).

Average Incidence: 1%.
Incidence 1957: 1%.
Maternal blood copper values not obtained.

Farm O (1957)
630-730' above sea level. W. of Greenlaw.
Old red sandstone with some porphryrite.
"Greyspeck" in oats, due to Mn deficiency, had been cleared up; but since a proportion of "deaf ears" (? Cu deficiency) have been observed.

History of Swayback: 5-6 cases annually. 1957: 30 cases, a few at birth but mainly after docking.

Mineral mixture containing 1,800 p.p.m. Cu fed throughout pregnancy at recommended levels.

Average Incidence: 1%.
Incidence 1957: 9%.
Maternal blood Cu values: Not obtained.

Farm P (1957)
800' above sea level.
Pyroxine-andesite.
Management: Arable rotation. Greyface ewes x B.L. on Suffolk. Pasture limed 4 tons/acre every 7 years.

History of Swayback: None prior to 1957 when 17
cases occurred, one or two at birth but mainly 8-12 weeks. (Pasture limed after first cases 4 tons/acre).

Average Incidence: Nil.

Incidence 1957: 10%.

No maternal blood values obtained.

Farm Q (1958)

850-950' above sea level. 3 miles N. Biggar.

Pyroxine-andesite (old red sandstone age).

Management: High arable rotation. B.F. gimmers bought in annually. Tupped B.F.

History of Swayback: Occasional cases. Rather more in 1957 and 1958. Usually normal at birth but develop moderately severe signs at 1-2 weeks. Licks containing 900 p.p.m. Cu available throughout the winter.

Average Incidence: 1%.

Incidence 1957 and 1958: 1-2%.

Maternal blood copper values: 15.3 µgms/100 mls.

Farm R (1958)

800-900' N.W. Lamington.

Clays and shales.


History of Swayback: Occasional cases. None in
1957. 4 in 1958. First noticed at 1 month of age - moderately severe cases.

Average Incidence: 0.5 - 1%.
Incidence 1958: 4%.

Maternal blood Cu values (swayback mothers): 11.7 and 15.3 µgms/100 mls.

Farm S (1957)

700' above sea level. 1½ mls. E. of Broughton.
Lowther shales, boulder clay and some radiolarian cherts.

Southerly aspect.


History of Swayback: Occasional cases. In 1957 about 12 lambs were mildly affected. Licks containing 900 p.p.m. Cu available during pregnancy.

Average Incidence: 1%.
Incidence 1957: 4%.
Maternal blood Cu values not obtained.

Farm T (1957)

600-800' above sea level. 2½ mls. N. Kennoway.
On a suspected fault dividing basalt in the South from carboniferous limestone.

Southerly aspect.

History of Swayback: None prior to 1957 when 10-12 mild cases occurred.

Maternal blood copper values were not obtained.
APPENDIX B

Being a summary of the protocols of cases in the 1957-58 survey.

465/1/57

Farm A. Greyface, 24 hrs. old. ☠ Natural death.
Liver not analysed.

465/2/57

Farm A. Greyface, 24 hrs. old. ☞ Natural death.
Liver not analysed.
Brain shrunken and congested. No macroscopic lesions. Microscopic haemorrhages in meninges and cerebral white matter. Myelin degeneration in corpus medullare of cerebellum. Early chromatolysis of large neurons in brain stem. Occasional shrunken and necrotic neurons in grey matter of the spinal cord. Myelin degeneration
in dorso-lateral and sulcomarginal funiculi.
Sciatic nerves show swelling of axis cylinders and sheaths.

465/3/57
Farm A. Greyface. ♀ 24 hrs. old. Chloroformed.
Liver not analysed.
Brain shrunken and congested. Gelatinous cerebral lesions extending from occipital pole to superior frontal gyrus. Subarachnoid space distended.
Thinning of cerebral myelin (Spielmeyer).
Capillary haemorrhages and slight vascular mural swelling. Chromatolysis and necrosis in red nucleus and medial and lateral vestibular nuclei.
Swelling of sheaths of oculomotor nerves. Cord shows myelin degeneration in dorso-lateral and sulcomarginal funiculi.

465/4/57
Farm A. Greyface, 24 hrs. old. ♀ Decapitated.
Liver Cu = 3.2 p.p.m. D.M. (Maternal blood 4.6 μgms/100 mls.)
Brain shrunken, congested and oedematos. No
macroscopic lesions, though white matter shrunken. Slight rarefaction of myelin in parietal lobe. Perivascular accumulations of gitter cells. Some precapillaries show separation of coats of the wall - space contains structureless eosinophilic material. Occasional cells of dentate nucleus show lysis and dustlike degeneration of neuro fibrils. Severe chromatolysis in lateral vestibular nuclei and myelin degeneration in cerebellar peduncles. Cord shows axon swelling and myelin degeneration dorso-laterally and ventromedially, which extends into peripheral nerve roots.

480/1/57

Farm A. Greyface x Suffolk. 36 hrs. old. Decapitated.
Liver Cu = 4.8 p.p.m. D.M. (Maternal blood Cu = 10.7 µgms/100 mls.)
This lamb was moribund and unable to stand. Brain flabby and shrunken. Small gelatinous lesions in white matter of occipital lobes. Tiny haemorrhages in sciatic nerves. Microscopic thickening of the pia-arachnoid. Chromatolysis, shrinkage and chronic cell changes in the superficial pyramidal cells of the parietal lobe. Chromatolysis and necrosis of the large neurons of the brain stem and cervical cord. Scattered
myelin degeneration in the brain stem, becoming localised into the dorsal, lateral and sulco-marginal funiculi. Also extends into the dorsal nerve roots.


Slight bilateral cavitation in hemispheres. C.N.S. congested and oedematous. Both grey and white matter very cellular - many gitter cells around the lesions and in periventricular white matter. Occasional necrosis in cells of the frontal lobe. Local marked thinning of cerebellar granular layer and occasional vacuolation or necrosis of Purkinje cells. Swelling of sheaths and loss of myelin in lateral and ventral regions of the cord and corpus medullare of cerebellum. Necrosis of large neurones in lumbar cord. Peripheral nerve roots show marked axon swelling and the myelin has a granular appearance. Slight focal fibroglial proliferation beneath the posterior corpora quadrigemina.
17.

Farm B. Greyface, 5 days old. ♂ Decapitated. Liver Cu = 4.0 p.p.m. D.M.
Just able to stand, collapses on attempted movement. Front legs appear more severely affected. Reflex activity appears weak. Brain shrunken especially in frontal lobes, but no macroscopic lesions. Microscopically there is congestion and oedema dilatation of perivascular spaces which frequently contain numbers of gitter cells. Occasional fibres are swollen and basophilic. Chromatolytic changes in lateral, vestibular, facial and dorsal paramedian nuclei. Severe chromatolysis of large neurons of the cord in the lower cervical segments.

Farm B. Greyface, 6 days old. ♂ Decapitated. Liver Cu = 2.5 p.p.m.
Farm E. Greyface, 24 hrs. old. Decapitated.
Liver Cu = 4.13 p.p.m. (Control group.)
Very severe case, unable to stand, nystagmus reflexes feeble.
Brain shrunken and haemorrhagic, marked unilateral gelatinous lesion.
Microscopically there is congestion and oedema.

Farm E. Greyface, 24 hrs. old. Decapitated.
Control group.
Able to stand but collapses on attempting to move.
Reflexes brisk but delayed.
No macroscopic cerebral lesions.
Periventricular white matter contains numbers of glitter cells. Chromatolysis and necrosis of neurons in thalamic and red nuclei and also in lateral vestibular, praepositus, and reticular formation. Marchi positive fibres in corpus medullare of cerebellum. Scattered positive
fibres in brain stem, dorso-medial, dorsal part of lateral, and ventrolateral funiculi of the cord. Some axon swelling.

860/2/57
Farm D. Greyface. ♂ 24 hrs. old. Decapitated.
Liver Cu = 7.1 p.p.m.
Very weak, moribund.
Brain shrunken and wet. Gelatinous softening right occipital lobe.
Slight cellular infiltration right occipital lobe (gitter cells and fibrous glia). Ornmitolysis of neurones in thalamic, pontine, and medullary nuclei. Thickening of walls of the small vessels of the brain stem. Mild myelin degeneration dorsal part of lateral, and ventro-lateral funiculi of cord.
Necrosis of large neurones, in lumbar expansion.

860/3/57
Farm D. Greyface, 24 hrs. old. Decapitated.
Unable to rise.
Brain very congested and oedematous.
Bilateral cavitation from occipital pole to level of hippocampus.
Lesions show peripheral collections of gitter cells.
Perivascular rarefaction of myelin ventrally and laterally in brain stem. Necrosis in nuclei of
pons. Gitter cells present around the bases of the Purkinje cells and occasionally in perivascular spaces of the cerebellar white matter. The cord shows a marked diffuse Marchi reaction anteriorly becoming localised to the dorso-lateral and ventral regions caudad. Chromatolysis extending to necrosis in the lumbar segments.


Brain shrunken, gelatinous lesion left centrum ovale extending forward as cavitation in the parietal lobe. Ventricular dilatation.

Chronic cell changes in deep pyramidal cells. Left centrum semiovale has a finely fenestrated appearance which fails to show any Marchi positive fibres, though minute positive granules are present in perivascular phagocytes. A few Marchi positive fibres in the internal capsule and diffusely scattered through the midbrain, pons and medulla mainly in the ventral regions. Large neurons show chromatolysis extending to necrosis in the lower cervical cord.
Farm E. Greyface, 24 hrs. old. Decapitated.
Liver Cu = 6.5 p.p.m. (Maternal blood Cu = 14.5 μgms/100 mls. Dosed group No. 12.)
Moribund and completely flaccid.
Bilateral cavitation.
Brain oedematous, slight malacia of grey matter around the lesions, especially alongside the small vessels. Normal myelin appears to extend right up to the lesion caudally, but anteriorly fades into a loose glial network containing a few fat phagocytes. Chromatolysis of cortical neurones. Chromatolysis in dentate nucleus, Marchi positive fibres and swollen axons in the cerebellar medulla. Severe chromatolysis and necrosis in red vestibular, abducent nuclei and reticular formation nuclei and in the cord especially the lower cervical segments. Vascular mural swelling with separation of the coats in the pons and medulla.

Farm E. Greyface, 24 hrs. old. Decapitated.
Undosed group. Liver Cu = 4.0 p.p.m.
Its twin was unaffected up to 6 weeks of age.
This lamb unable to stand.
Bilateral cavitation.
The cerebrum is congested and oedematous with
retraction spaces (pericellular oedema) and marked cavitation of the white matter. The margins of the lesion show a "felted" network of glial fibres. Chromatolysis of large neurons of brain stem and spinal cord extending to necrosis in the red nucleus, pons, and ventral horns of the lumbar expansion of the cord.

929/2/57
Greyface. ♂ 24 hrs. old. Decapitated.
Liver Cu = 6.2 p.p.m. (Maternal blood Cu = 14.5 μgms/100 mls. Dosed group No. 12.)
This lamb just able to stand but exhibited marked ataxia, especially of the fore legs.
Bilateral cavitation.
Diffuse infiltration of gitter cells in right frontal lobe. Vacuolation of cells of thalamic nuclei. Chromatolysis of large neurones of the brain stem and cord is not marked, though diffuse myelin degeneration is present.

929/4/57
Farm E. Greyface, 24 hrs. old. Decapitated.
Liver Cu = 6.6 p.p.m.
This lamb was unable to stand, though its twin was only mildly affected.
No macroscopic lesions other than cerebral
congestion.

Oedema, chromatolysis of pyramidal cells. Tiny areas near the occipital pole show slight rarefaction of myelin. Swollen axons and sheaths in the cerebellum. Chromatolytic neurones in vestibular nuclei reticular formation, but necrosis only observed in the medulla and large cells of the lumbar expansion.

966/1/57

Farm C. Greyface, 3 weeks old. Decapitated.
Liver Cu = 2.8 p.p.m. (bottle fed). Maternal blood Cu = 6.8 µgms/100 mls.
Unable to stand. Severe hind leg ataxia. Weakness on the right side. Reflexes delayed but strong.
No macroscopic lesions.
No recognisable rarefaction of myelin, though fat phagocytes frequent throughout cerebral white matter. Chromatolysis and necrosis of the large nerve cells of the thalamus. Myelin degeneration in lateral and ventral columns of the cord. Neurosis in large ventral horn cells of the cervical and lumbo-sacral expansions.

984/1/57

Farm E. Greyface, 24 hrs. old. Decapitated.
Liver Cu = 7.72 p.p.m.  (Dosed group.)
Unable to stand. Reflexes sluggish, slight nystagmus.
Slight unilateral cavitation.
Normal myelin appears to extend right up to the edge of the lesion. Cerebellar white matter, oculomotor nerve roots and brain stem contain Marchi positive fibres. Sulcomarginal and dorso-lateral, and septomarginal funiculi of cord contain many blackened fibres. Chromatolysis only severe in lumbar cord.

984/2/57
Farm E. Greyface, 24 hrs. old. Decapitated.
Liver Cu = 6.7 p.p.m. Dosed group.
Twin to 984/1. Not so severely affected, able to stand. Reflex activity apparently normal.
No macroscopic lesions. Fat phagocytes in periventricular white matter and in perivascular spaces of the occipital lobe. Chromatolysis and necrosis of cells of the dentate nucleus. Scattered Marchi positive material in brain stem becoming localised to dorso-medial, dorso-lateral and ventral columns caudally. Mild chromatolysis at all levels with necrosis of large neurons in thoracic and lumbar segments. Occasional swollen axons and Marchi positive fibres in the sciatic nerves.
Farm E. Greyface. 5-6 days old. Decapitated.
Liver Cu = 8.5 p.p.m. (Control group.)
Severely affected, unable to stand, and bottle fed.
Reflexes apparently normal.
Gelatinous cerebral lesions with some cavitation
(more severe on right side). Terminal B. coli
septicaemia. "Chronic cell change" in a proportion
of pyramidal cells. Normal myelin extends
right up to the lesions. Occasional fat phagocytes at the periphery. Purulent meningitis round
base of brain. Widespread myelin degeneration in
brain stem becoming localised into dorso-lateral
and ventral funiculi of the cord. Necrosis of
cells of red nucleus and ventral horn cells of the
cervical and lumbar expansions of the cord.
Spinal meningitis and neuritis. Occasional
swollen nerve fibres in the peripheral nerve roots.

Farm F. Greyface, 1-2 days old. Natural death.
Liver Cu = 7.84 p.p.m. (Maternal blood Cu = 32.3
/dl. gms/100 mls.)
Rather decomposed. Bilateral cerebral cavitation.
No histological examination was undertaken.
Farm F. Blackface, 1 week old. Decapitated.
Liver Cu = 6.9 p.p.m. (Bottle fed.) (Maternal blood Cu = 5.5/mgms/100 mls.)
Very feeble movements, unable to stand unassisted.
Reflex activity apparently normal.
Slight bilateral gelatinous lesions. Loose glial network thinning of myelin. Deep pyramidal cells around the lesions show early chromatolysis.
Occasional necrotic neurons in pons, medulla, and ventral horns of the spinal cord. Myelin degenerations in dorso-lateral and ventral-lateral funiculi.

Farm G. Greyface, 2-3 weeks old. Decapitated.
Liver Cu = 3.1 p.p.m. (Bottle fed.)
Bilateral cavitation.
Cerebral and meningeal vessels congested.
Occasional chromatolysis of cortical neurons.
Severe malacia of white matter, the periphery of the lesions being circumscribed and formed of compressed glial fibres. Small malacic areas
also present in adjacent cortex. Residual white matter contains a proportion of Marchi positive fibres. Chromatolysis of the large neurons of the brain stem with necrosis in the pons and occasionally also among the large neurons of the lateral and ventral horns of the cord. Marchi positive fibres in the septo-marginal, sulco-marginal, dorsal and ventral parts of the lateral funiculi.

1109/57

Farm I. Blackface, 2-3 days old. Decapitated. Liver Cu = 540 p.p.m. (Bottle fed.) (Maternal blood Cu = 10.9 μgms/100 mls.)

Reflexes normal. Hind legs ataxic, swaying particularly to the left. Kidneys pale, clear ascitic fluid. Brain shrunken with jelly-like leptomeninges.(?) External hydrocephalus, α haemolytic streptococci cultured from C.S.F. and heart blood.

Bilateral gelatinous lesions and ventricular dilatation. White matter replaced by a loose glial network containing small numbers of fat phagocytes. Occasional fragmentation and chromatolysis of cortical neurons. Scattered degenerate fibres throughout the brain stem and in dorsal and ventral parts of the lateral funiculus of the cord.
Chromatolysis of large neurons with necrosis in red nucleus and vestibular nuclei. The kidney shows occasional atrophic glomerular tufts and mild tubular degeneration.

Farm H. Greyface, 4 weeks old. Natural death. Blood Cu = 15 µgms/100 mls. (Maternal blood Cu = 8.18 µgms/100 mls.) No clinical information. Slight flattening of gyri of right cerebral hemisphere. Very pale kidneys. Occasional deep pyramidal cells show fragmentation or foamy cytoplasm. Gitter cells present between nerve fibres. Chromatolysis and occasionally necrosis in thalamic nuclei and widespread chromatolysis in vestibular and hypoglossal nuclei and reticular formation. The inferior olivary nucleus shows many shrunken hyperchromic cells. Very extensive myelin change in lateral funiculi of the spinal cord and severe chromatolysis of large neurons in the cervical and lumbar expansions. The sciatic nerves show occasional degenerate fibres with gitter cells in the interstices.

Farm H. Greyface, 3 weeks old. Decapitated.
Liver Cu = 3.1 p.p.m.  (Maternal blood Cu = 6.8 \( \mu g/m\) /100 mls.

Signs only evident on exercise - ataxia of hind limbs.


Chromatolysis and necrosis of large thalamic nerve cells.  Occasional Marchi positive fibres scattered through the corpus striatum but most numerous in lateral and ventral regions of the brain stem.  Necrotic neurons in pons and cervical expansion of the cord.  Numerous Marchi positive fibres in dorsal and ventral parts of the lateral funiculi.

Farm E.  GreyFace.  3 weeks old.  Natural death.

Liver Cu = 2.1 p.p.m.  (Control group No. 5.

Maternal blood Cu = 10.0 \( \mu g/m\) /100 mls.)

No macroscopic lesions, but cerebrum is congested.

Gitter cells in perivascular spaces of parietal lobe.  Extensive necrosis in thalamic and vestibular nuclei and also in the cervical expansion of the cord.  Scattered Marchi positive fibres become
localised to dorsal and ventral parts of the lateral funiculus in the cord.

1205/58
Farm J. Greyface, 1 month old. Decapitated.
Liver Cu = 10.1 p.p.m. (Maternal blood Cu = 17.7 
\(\mu\)gms/100 mls.)
Normal at birth, developed progressive hind leg ataxia.
Congestion of C.N.S. but no macroscopic lesions.
Slight microscopic oedema and cortical chromatolysis.
Scattered sinter cells in white matter.
Thickening of arteriolar walls of the brain stem.
Necrosis in reticular formation, and ventral horn cells of the cervical and lumbar expansions of the cord. Degeneration of fibres in dorso-lateral and ventral funiculi.

1218/1/57
Farm D. Greyface. \(\Phi\) 2 weeks old. Decapitated.
Liver Cu = 17.5 p.p.m. (Maternal blood Cu = 65.5 
\(\mu\)gms/100 mls.)
Reflexes normal, but severely ataxic. False bursae both carpi.
Brain congested and oedematous with slight bilateral cavitation. Small numbers of Marchi positive fibres in centrum ovale and corpus
striatum, through the brain stem becoming localised into the ventro-lateral and dorso-medial regions in the medulla. Also present in cerebellar white matter and a few blackened fibres in the sciatic nerves. Red, medial vestibular, and hypoglossal nuclei and cells of the reticular formation show severe chromatolysis. Shrinkage and hyperchromasiam of occasional neurons of the inferior olivary nucleus is evident.

12/13/2/57

Farm D. Greyface, 5 weeks old. Decapitated.
Liver Cu = 7.9 p.p.m. (Maternal blood Cu = 23.2 μgms/100 mls.)

Very severe ataxia but able to suckle.
No macroscopic lesions.
Congestion, distention of V-R spaces. Grey matter has a foamy appearance. White matter shows occasional Marchi positive fibres. Chromatolysis and necrosis of neurons in the medial vestibular nuclei, and reticular formation. Considerable number of Marchi positive fibres in medullary body of the cerebellum. Classical distribution of positive fibres in the cord. Kidney shows proliferation of the lining cells of Bowman’s capsule.
Farm E. Blackface, 2-3 weeks old. Decapitated.
Liver Cu = 7.6 p.p.m.
Blood Cu = 35.3 μgms/100 mls. (Maternal blood Cu = 16.4 μgms/100 mls.)
Normal at birth, progressive development of ataxia.
C.N.S. wet and congested. Brain shrunken.
Arteriolar mural thickening in brain stem.
Necrosis of multipolar nerve cells, pons, medulla and ventral horns of the spinal cord. Classic myelin degeneration in the cord.

Farm L. Greyface, 3 weeks old. Decapitated.
Liver Cu = 5.6 p.p.m.
Affected from birth. Unable to rise unassisted.
Collapse on movement.
No macroscopic lesions.

Farm L. Greyface, 2 weeks old. Decapitated.
Liver Cu = 1.8 p.p.m.
"Weakness" of hind legs.
Brain very shrunken. Cellular thickening (? fibroblastic) of pia arachnoid in frontal region. Chromatolysis of the large neurons of the brain stem and cord but necrosis seen only in region of pons, cervical and lumbar enlargements. Classical mild myelin degeneration.

1271/57

1278/1/57
Farm K. Blackface. 3 weeks. Decapitated. Liver Cu = 4.5 p.p.m. Blood Cu = 27.5 μgms/100 mls. (Maternal blood Cu = 10.9 μgms/100 mls.)

1331/57
Farm M. Greyface, 3 weeks old. Decapitated.
Liver Cu = 11.3 p.p.m. Blood Cu = 48.6 μgms/100 mils. (Maternal blood Cu = 21.8 μgms/100 mils.)
"Weakness" of hind legs very marked.
No macroscopic or microscopic cerebral lesions.
Chromatolysis of large neurons with necrosis in pons, cervical and lumbar cord enlargements.
Degenerate fibres in the brain stem, cerebellar peduncles, dorso-lateral and ventral funiculi of the cord.

1429/57
Farm E. Greyface, 8-10 weeks old. Decapitated.
Control group. Liver not analysed.
Necrosis only found in ventral horn cells of lower cervical segments. Very slight degenerative changes in lateral and ventral regions of the cord.

1520/57
Farm N. Greyface, 10 weeks old. Decapitated.
Liver Cu = 106 p.p.m.
Progressive deterioration of irregular foreleg action with exercise.
No macroscopic lesions.
Chromatolysis necrosis in pons, cervical and lumbar enlargements. Myelin degeneration (mild) in dorso-lateral and ventral funiculi.

1520/2/57
Farm N. Blackface, 10 weeks old. Decapitated.
Liver Cu = 19.5 p.p.m.
Moderate/severe hind leg ataxia.
No macroscopic or microscopic cerebral lesions.
Mild chromatolysis in thalamic nuclei, occasional necrotic cells in dentate and medial vestibular nuclei. Marked myelin degeneration of the cord but no evidence of gliosis.

1615/57
Farm O. Cheviot. 3 months old. Decapitated.
Severe ataxia, frequent collapse. Tremors of
head and jaw. Hyperactive pedal reflexes.

Marasmus.

Brain shrunken and dry.

Cerebral white matter contains perivascular Marchi positive material. Chromatolysis of large neurons of thalamus red nucleus, pontine and medullary nuclei. Decussating fibres in the medulla strongly Marchi positive. In the cord they are situated ventrally and laterally, and ventrally only in the lower segments. Necrosis of large neurons in lumbar cord. Heart muscle contains isolated foci of round cell myositis.

2023/57

A.M.W. Greyface. 5-7 months old. Decapitated.

Liver Cu = 55.4 p.p.m. Blood Cu = 57.4 μgms/100 mls.

Well grown and bright lamb. "Stronghalt" action with left hind - increases with exercise.

Reflexes strong but delayed. "Capped knees."


Chronic cell changes in deep pyramidal cells.

Perivascular accumulations of Marchi positive material in cerebrum and brain stem and also
occasional Marchi positive fibres scattered in brain stem and lateral and ventral areas of the cord. Chromatolytic changes in ventral horn cells of lumbar cord.

2359/1/57

Farm N. Greyface, 7 months old. Decapitated. Poor ill-thriven lamb. Only ataxic with exercise (showed clinical improvement). No lesions other than marasmus at P.M. Occasional Marchi positive fibres scattered through cerebellar peduncles and ventrally and laterally in the cord. Shrunken necrotic neurons in ventral horns.

891/58

Cainiean. Bilateral cavitation, residual white matter shrunken. Marked Marchi positive reaction in diencephalon becoming progressively localised to ventral areas caudally. Pons and medulla contain dorso-medial and lateral accumulations also (? from cerebellum). Aggregations in lateral and ventral areas of the cord and also sulcomarginal funiculi in cervical cord.

791/58

Farm U. Blackface, 2 days old. Natural death.
B. coli isolated from all organs.
Unilateral cerebral cavitation.
Cerebrum congested. Masses of gitter cells at periphery of the lesions. Necrosis in red and lateral vestibular nuclei and throughout the ventral horns of the cord. Classic Marchi reaction in the cord.

Farm B. Greyface, 2 days old. Decapitated.
Blood Cu = 21 μ gms/100 mls. Liver Cu = 5.6 μμμ μμμ μμμ μμμ
Unable to stand alone, all limbs affected. Reflexes brisk. Apparently blind. Slight bilateral cavitation. Numerous gitter cells around lesions and along the blood vessels into the adjacent cortex. Chromatolysis in the vestibular nuclei. Marchi positive fibres were not observed in the cerebrum, but were numerous in the brain stem, lateral and ventral fasciculi of the cord. They were not observed in peripheral nerves.

Farm E. Greyface. ♂ 24 hours old. Natural death. Control group. (Maternal blood Cu = 2.3 μ gms/100 mls. No. 39.) Massive bilateral cavitation.
Many gitter cells around the cerebral lesion and between the fibres of the brain stem, especially in areas containing Marchi positive fibres. Similar fibres in *corpus medullare*, pons, and medulla becoming localised to the lateral and ventral sites of the cord. In addition many are present in the septomarginal funiculus. Necrotic neurons only observed in the lumbar cord.

**1321/58**

A.S. Greyface. ♂ 3-4 weeks old. Decapitated.

Moderate - severe ataxia.

No gross lesions. Marchi positive fibres in the internal capsule, but more numerous in the pons and medulla and becoming localised to the lateral and ventral funiculi of the cord. Necrosis of large neurons present in the thalamic nuclei.

**1360/58**

Farm R. Greyface, 10 weeks old. Decapitated.

Blood Cu = 56.8 µgms/100 mls. Liver Cu = 7.5 µg/ml.

(Maternal blood Cu = 11.7 µgms/100 mls.)

Gelatinous oedema of the pia arachnoid. Pyramidal cells show some "chronic cell change". Chromatolysis in the dentate nucleus and large neurons of the ventral horns of the spinal cord. No evidence of gliosis was observed in the cord.
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In the spring of this year the veterinary department of this college was asked to assist in the diagnosis of a condition of young lambs clinically similar to swayback. Two affected lambs were examined without finding any evidence of demyelinating central nervous disease, and it was decided to investigate the matter further.

History
The condition occurred on a sheep farm situated 1,400 feet above sea level on the southern aspect of a hill in the east of Scotland. The ewe flock was entirely of Blackfaces, which either were bred on the farm or bought in as gimmers. The rams were, with one exception, Border Leicesters. A single Blackface ram was kept to replenish the breeding stock.

In recent years a policy of hill improvement had been followed, the ground being ploughed and reseeded, and repeatedly and heavily limed. Small but increasing losses of lambs from an ataxic condition had occurred during the past two or three years, but this year 50 out of a total of 230 Greyface lambs (22 per cent.) and a single Blackface lamb were affected. The Blackface ram had not been intended for use, but had broken out and tupped a single ewe of the home flock, which gave birth to an affected lamb, and several on a neighbouring farm. His offspring on this second farm were unaffected.

Clinical findings
The principal signs exhibited were hyperaesthesia, nystagmus, inco-ordination of gait, ataxia, and intermittent paralysis chiefly affecting the hind but occasionally the fore limbs. At rest, affected lambs frequently held their heads very high in mild episthenotonus. In a large proportion of them the clinical picture became manifest only during, or immediately after, exercise. On driving the flock occasional lambs appeared to lose their balance completely, and with a strong thrust of the hind limbs performed an alarming spiral projectile movement before coming back to earth and rolling over repeatedly. A considerable number were affected from birth, but fresh cases became apparent throughout the first six or seven weeks of life. Both members of each affected pair of twins developed symptoms, but not always at the same time or with equal severity. Lambs which were able to follow their dams and graze throw remarkably well, but others, not so fortunate, developed skin wounds and secondary infections and died or were destroyed. The disease in general appeared to be slowly progressive.

Investigations
In all, 11 lambs were removed from the flock and killed for examination. The method of destruction varied from animal to animal. Two were chloroformed, one was given saturated magnesium sulphate intravenously, six were electrically stunned and then bled, and two killed by severing the vessels in the neck.

Post-mortem examination revealed no macroscopic abnormalities. The central nervous systems of all the lambs and other organs of two of them revealed no significant organisms on culture in glucose broth, meat piece broth or on blood agar.

The brains and spinal cords of all were removed and fixed in Bouin, and representative blocks of the other tissues from two lambs were taken and fixed in formal- corrosive. No abnormalities were recognised in H. & E. stained sections of the latter.

In the study of the C.N.S. H. & E., Mahon’s myelin method, the Busch modification of Marchi, Kurnick (1955) and Holmes (1956) were the chief techniques applied. In the H. & E. stained sections necrotic neurons were observed diffusely scattered through the hind brain and cord, but were most numerous in the pons and dentate nucleus. No evidence of encephalitis or generalised toxæmia which might account for these changes has so far been observed. The supplementary techniques revealed structural disturbances in a much greater proportion of cells. In a large number, the neurofibrils appear to degenerate into a finely granular dust, whilst the cytoplasm of the cell becomes swollen particularly round the exit of the axis cylinder. In others the neurofibrils appear to form short rods disposed irregularly towards the periphery of the cell. Local dilatations of axis cylinders, with loss of neurofibrils at points remote from the cell, have been observed also.

Discussion
This condition, which clinically is so similar to swayback, is remarkable in that the pathological changes are so different from the demyelinating lesions of that disease. In addition to demyelination, Innes & Shearer (1940) described degenerative changes in the neurons of the red nucleus, but these were not present in the mild or chronic cases in which they were unable to demonstrate demyelination. In the present series the nerve cell changes are the prominent feature.

A neighbouring farm undergoing a similar programme of pasture improvement has this year also lost a few lambs from a condition clinically similar to that described; but the opportunity of making a pathological examination has not yet arisen. In 1954 Watt (1956) noted an outbreak of disease in young lambs clinically identical with swayback but in which demyelination was absent. The condition occurred on an arable farm carrying 90 Blackface ewes crossed with Border Leicester rams, and in that year, and subsequently, about half a dozen lambs have been
affected annually. In Watt's case, however, all the lambs were affected from birth. Re-examination of some of the material from this case has revealed changes similar to those described.

It is emphasised that at the time of writing the histological study is very incomplete and the description of the changes presumptive. Later, it is hoped to publish a full account of the pathological aspects of this condition, together with results of biochemical investigations and soil and herbage analyses. The purpose of this communication is to draw attention to the existence of the condition so that further material may not be overlooked.

**Summary**

A condition in lambs clinically resembling sway-back but lacking the characteristic lesions of that disease is described. A preliminary investigation into the nature of the lesions has been conducted and it is suggested that this condition may not be uncommon.

**Acknowledgment.**—I am grateful to Dr. I. Zlotnik for suggesting the Holmes technique, and for his guidance in the interpretation of the results.

**References**


RECENT ADVANCES IN SWAYBACK

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The title “Recent Advances in Swayback” is perhaps misleading, implying work carried to a stage when definite conclusions may be drawn. This paper is an interim report of work in progress and any conclusions must be considered more in the nature of suggestions.

The investigation has entailed co-operation between clinician, biochemist, pathologist, and soil chemist and so I propose to give a general statement on swayback, followed by an account of our own findings with emphasis on the clinical and pathological features.

Though swayback has been known for well over a century no description appeared until 1917 when Gaiger mentioned it in an account of “Renguera” a similar disease occurring in Peru. Since 1932 an extensive bibliography has developed and swayback or very closely related conditions are recognized in many parts of the world (Stewart, 1932; Bull et al., 1938; Bennetts, 1932; Bennetts and Beck, 1942; McDonald, 1942; Tabusso, 1942; Innes and Shearer, 1940; Palsson and Grimsson, 1953; Schulz et al., 1951; Dandemaev and Abramova, 1956; Spais, 1956; Gracey and Todd, 1958). A comprehensive review has been recently published by Innes and Saunders (1957).

Clinical features.—Swayback is a condition affecting the lambs of apparently healthy ewes of all breeds, at birth or during the first few weeks of life. It is an afebrile, ataxic disease which may be progressive and fatal in all but the most mildly affected cases, and these may accommodate themselves to show almost complete recovery and may in turn give birth to healthy offspring. Ewes may give birth to healthy or affected lambs in successive years.

The most severely affected are stillborn, or weak at birth, may be unable to rise and suckle, and die within twelve to twenty-four hours. If bottle-fed they may survive for long periods. Less severely affected lambs will suckle naturally, but they walk with a staggering, spastic type of gait, often collapsing. The bleat is normal but they may be blind. The ataxia is most noticeable in the hind-limbs but sometimes also affects the fore-limbs, and the degree of inco-ordination increases greatly with exercise or excitement. Death in these cases is often the result of secondary infections. Single lambs, or one or more siblings of a multiple birth, may be affected and to different degrees.

Pathology.—Innes and Shearer (1940) suggested that the condition was an antenatal, bilaterally symmetrical demyelination of the cerebrum with consequent destruction of the axis cylinders and later cavitation, together with secondary degeneration of the motor pathways. They compared it with Schilder’s disease in man. The few pathological studies carried out since 1940 agree in general with these findings and such differences as there are will be discussed later.

Aetiology.—Early attempts to incriminate an infectious agent met with no success, and it was not until the mid-1930s that Bennetts and Beck (1942) in Australia proved that the disease was associated with copper deficiency and could be prevented by administering copper supplements to pregnant ewes. His results were confirmed in this country by Dunlop et al. (1939). An important aetiological difference, however, between the disease in Australia and Britain is that in Australia the condition occurs on pastures low in copper, i.e. below
5 p.p.m.¹ D.M.² and frequently below 3 p.p.m., whereas in this country it occurs irrespective of copper content of the pasture and is prevalent in Derbyshire on pastures containing 7–15 p.p.m. copper or more (Allcroft, 1952). Thus it appears that some conditioning factor may be involved which reduces the availability of copper to the animal (Green, 1951), or causes its increased elimination.

In cattle such a conditioned copper deficiency syndrome occurs on the teart pastures of Somerset, in which copper storage by the liver is reduced due to high dietary molybdenum (Ferguson et al., 1943; Dick and Bull, 1945; Cunningham, 1949). There are, however, no records of swayback occurring on teart pastures, and an experimental attempt to produce the condition by feeding ammonium molybdate and sodium sulphate to pregnant sheep failed, though a degree of hypocuprosis associated with wool dystrophy and hypochromatia, was obtained (Wynne and McClymont, 1956). Further, there are local areas in Westmorland where copper deficiency occurs in sheep, associated with wool dystrophy and wasting, in which swayback has not been known to occur (Heath, 1958). These observations suggest that another factor operating in the presence of a low copper status may be necessary for swayback to develop. Antenatal plumbism has also been considered as a factor in the causation of swayback (Bennettts, 1935) but experimental support is lacking.

Thus although copper supplements provide a useful practical answer to the swayback problem little is known regarding the causal factors. The very variable annual incidence which reaches serious proportions on some farms in some years still makes the disease of considerable importance to the individual farmer, although on a national scale it is of small consequence. Its importance in the field of comparative medicine need not be stressed. In this connection the very well-known report of Campbell et al. (1947) on seven swayback research workers, four of whom developed disorders of the nervous system clinically indistinguishable from disseminated sclerosis.

The work in Edinburgh.—In the spring of 1956 a hill flock was encountered in which over 20% of the lamb crop had been lost as the result of an ataxic condition. An account of the clinical features and pathology has already been published (Barlow, 1956). At the time of examination all the remaining lambs were over a month old; the history and clinical signs suggested swayback, but in no case was it possible to demonstrate cerebral demyelination. The predominant lesion was a myaline necrosis of multipolar nerve cells scattered throughout the nuclei of the brain-stem and ventral horns of the cord; though numerically insufficient it seemed to account for the clinical signs. The livers of the few lambs examined biochemically were found to have low copper values.

Accordingly it was resolved in collaboration with Dr. E. J. Butler of Moredun Institute and Dr. D. Purves of the East of Scotland College of Agriculture to study this farm throughout the season 1956–57. The two adjacent farms, one of which had a similar history of ataxia, the other being alleged free, were also included for detailed observation. A controlled dosing experiment was recorded out on one of these farms. In addition, during the 1957 lambing season another score of affected farms within the College area were visited, as detailed a history as possible taken, suspected cases examined clinically and material obtained for biochemical and pathological examination, and pasture for trace element analysis. Much of the information obtained has yet to be correlated and its significance assessed but it is hoped to publish a detailed report in due course. The following is a brief account of our findings so far.

More than 75% of the outbreaks occurred on marginal or hill farms lying chiefly between the 500–1,500-feet contours, the sheep having a run-off to variable amounts of ploughed land. With few exceptions the disease occurred on land which had been heavily and repeatedly limed. Most of the farms had a previous history of sporadic cases or small outbreaks but in 1957 there was an overall increase in incidence, losses varying from 2-25% of the lamb crop. The condition was seen in Blackfaces, Border Leicesters, Cheviots, and Suffolk, and their various crosses, but most commonly in Border Leicester × Blackface (Greyface) lambs.

The most severe cases were stillborn or very weak at birth and failed to rise and suckle the ewe and unless bottle-fed died very soon. Others could get to their feet and suck naturally but showed the typical ataxia which became more pronounced with exercise or excitement. Fresh mild cases were noticed until the lambs were 8–10 weeks old.

In no case was there clinical evidence of hypocuprosis or other disease in the ewes.

Soil and pasture from suspect areas were collected by a method designed to give representative samples (Calder and Voss, 1957), and analysed spectrographically for a wide range of trace elements. With respect to copper, the herbage values obtained from 46 fields varied from 1.8–25.5 p.p.m. dry matter. The average was 5.9 p.p.m. and the standard deviation was roughly within the normal range for pasture in England and Wales reported by Eden (1944).

¹Parts per million. ²Dry Matter.
Biochemical findings.—Blood samples were taken from ewes giving birth to affected lambs on all the farms visited, and the copper levels determined. In addition the livers of all lambs taken for pathological examination were analysed, and in some cases the blood copper values of these lambs were also determined.

It was found that the mothers of swayback lambs had unusually low blood copper values, i.e. 60 µg./100 ml. and in some cases down to <1·0 µg./100 ml. Affected lambs were also found to have unusually low blood and liver copper. A close correlation between the degree of hypocuprosis at the time of sampling and the severity of the lesions at post-mortem was not apparent, though these data have yet to be analysed statistically.

Dosing experiment.—The flock examined in 1956 was divided into two groups of about 100 ewes and 50 ewes respectively. The larger group was dosed with 1·0 gram of CuSO₄, per os three times, at monthly intervals, during pregnancy, the last dose being given about ten days before lambing was due to commence. The other group was kept as a control. Blood copper levels of individual animals in both groups were determined at monthly intervals. Further blood samples were taken from the adjacent farm which had no history of swayback.

Whilst very great differences in blood copper levels between individual sheep on both farms were found, the majority were below 60 µg./100 ml., i.e. within the range where swayback is likely to occur (Allcroft, 1952). The administration of copper sulphate produced no significant alteration in the blood copper levels when sampled one month after dosing. Subsequently it was found that the dosed group showed a 3% incidence of swayback whilst in the control group it was about 23%. Some ewes giving birth to normal lambs had, in fact, significantly lower blood copper values than those with affected lambs. The adjacent farm which was similar in respect to geographical and climatic conditions, but where no liming or other attempts to improve the pasture had been made, had no cases of swayback, though the blood copper values of the ewes were within the swayback range. These findings bear out other authors' conclusions of a "one-way correlation" between low copper status and swayback (Allcroft, 1952).

Pathology.—With only a few exceptions the lambs for pathological examination were freshly killed by a standard method, the central nervous system removed to fixative as soon as possible and the subsequent treatment of tissues was by standardised techniques throughout. A total of 47 lambs representing all ages from birth to 10 months old and all degrees of clinical involvement was examined.

The macroscopic cerebral lesions of cavitation or gelatinization described by Innes and Shearer (1940) were only observed in 38% of cases, as compared with 70% in their series of 167 cases. Unless hand-fed no lamb showing these lesions was more than 2 weeks old. On the other hand, equivalent clinical cases without gross cerebral lesions were found throughout the series. Some cases showed slight dilatation of the lateral ventricles and reduction in size of the diencephalon in the region of the anterior cerebral commissure.

Lesions were not observed in other organs apart from unilateral gluteal atrophy in one older case (9 months) and periglomerular fibrosis with tuft atrophy and hypertrophy of the lining epithelium of Bowman's capsule in the kidneys of a few cases aged 4-5 weeks. Similar renal lesions have been observed in other conditions of lambs (Butler et al., 1957; Hartley, 1953).

Histology of the Central Nervous System

Cerebrum.—Even in the motor area cortical changes were few and consisted of dilatation of the perivascular spaces and occasional fine stippling of the cytoplasm of nerve cells with shrinkage and pericellular cedema.

The white matter of the hemispheres showed lesions varying from gross fluid-filled cavities to tiny foci of gelatinous appearance at the tips of the gyri or in the centrum ovale. The cavities were bounded by a feltwork of fibrous glia having the appearance of a compression capsule whilst the gelatinous areas consisted of a loose glial network containing few axis cylinders or myelin sheaths. Intense glial proliferation has not been seen, no inflammatory reaction observed, and in all but 2 recent cases fat phagocytes have been remarkably few in number; findings which are in close agreement with those of Innes and Shearer (1940). In 2 cases, however, masses of glister cells have been present around the lesions extending into the adjacent cortex along the vessels. Using Spielmeyer and Marchi techniques, normal myelin appeared to extend up to the edge of the lesions.

In 62% of cases, however, no lesions of cerebral myelin could be detected, which compares with the findings of McDonald (1942).

Cerebellum.—No lesions were observed apart from a few Marchi-positive fibres in the corpus medullare of the youngest animals.

Brain-stem and cord.—In 97.5% of all cases under 12 weeks old chromatolytic changes extending to a hyaline-like necrosis were found in varying numbers of the large neurons of the stem and ventral horns of the cord, and using Holmes' (1947) technique the neuro-
fibrillary apparatus would appear to be damaged in a larger proportion. The number and situation of affected cells varied from case to case. Cell changes have been mentioned by Stewart (1932), Innes and Shearer (1940), McDonald (1942), Dandemaev and Abramova (1956).

In the younger cases Marchi-positive fibres have been seen running through the brain-stem to become localized in the dorsolateral and subcomarginal funiculi of the cord. Smaller concentrations were also seen in the septomarginal funiculi. In no case has sclerosis of the cord (McDonald, 1942) been observed.

Sciatic nerves.—A few Marchi-positive fibres have occasionally been seen in the sciatic nerves.

Normal animals.—A small series of normal animals has been examined by the same standardised methods. In those under 10 days old Marchi-positive material has been found in the same sites as in swayback lambs. In addition similar quantities of sudanophilic material were present between nerve fibres and in the perivascular spaces.

Examination of material prepared by a bulk fixation technique has indicated that some of the Marchi-positive material may be, in fact, stretch artifact (Smith, 1951, 1956).

A preliminary histological examination of foetal nervous tissue has revealed a picture somewhat similar to that seen in some cases of swayback. The cortical neurons show fine stippling of the cytoplasm and lie in "retraction spaces" and areas of active medullation have the appearance of the gelatinous lesions of the disease, similar quantities of sudanophilic material being present. Further work, however, is required to determine how far this similarity exists.

DISCUSSION

Our clinical, chemical and biochemical observations bear out those of previous workers to a large extent and identify the condition we are studying with that previously described.

In our experimental group of farms the one on which no cases of swayback occurred was adjacent to the one showing a 23% incidence in the untreated group. The geographical and climatic conditions were similar on both farms, and comparable copper values were found in both pasture and sheep. On the farm with the disease, however, a more intensive flock and pasture management was practised which included heavy repeated liming over a period of years. It would appear that some factor associated with this type of management may be involved in the causation of swayback.

The definitive cerebral lesions were observed in only 38% of our cases as compared with 70% in Innes' series. It was not possible to predict clinically the presence or absence of these lesions in animals less than 2 weeks old and thus one is inclined to doubt that they constitute the significant lesions of the disease.

The presence of necrotic neurons in 97.5% of our cases, on the face of it, appears to be much more significant. In many instances, however, these are not numerous and even taking into account the less severe chromatolytic changes in other nerve cells, the total number of cells involved frequently does not appear to be sufficient to account for the clinical condition of the animal. It is tempting to postulate the presence of an extensive functional lesion of neurons not visible by the methods so far employed. In this connexion mitochondria and Golgi apparatus may be worthy of attention.

In spite of the varying degree of cerebral involvement, the extent of the so-called "secondary tract degenerations" appeared remarkably constant and did not seem to be related to the numbers of abnormal multipolar nerve cells observed in the brain-stem. It thus seemed likely that some of this Marchi-positive material was artifact. If so, the constancy of distribution at all levels of the cord suggested that it might be the result of stretching of unfixed fibres.

The study of the central nervous system of normal lambs of equivalent ages by the same standard techniques showed a similar distribution of Marchi-positive fibres. Bulk fixation methods designed to avoid stretching of unfixed fibres may reduce this to some extent.

This modified treatment of tissues has still to be applied to swayback material, but in the light of these findings it seems likely that some difficulty may be experienced in interpreting the results.

The relatively small amounts of intra- and extra-cellular lipoid present around the cerebral lesion in the great majority of cases have been considered as evidence of a demyelinating process. That similar amounts are present in animals showing no lesions, in normal young animals and also in fetuses where medullation is occurring lends support to the theory that the cerebral lesion is due to myelin aplasia rather than demyelination. This theory was advanced by Innes and discarded as untenable on the grounds that in swayback the "... brain as a whole is a well-developed full-time organ in respect of size, conformity and structure ..." and because "... it would not explain the porous areas, the gross cavitation, nor the myelin destruction demonstrated by positive staining reactions (Marchi or Scharlach R methods) ... ."

Further support for an aplasia or dysplasia theory may become apparent from a closer study of the foetal cerebrum prior to the completion of medullation, in relation to the swayback brain showing the gelatinous lesion. Superficially they appear to show some similarity.
CONCLUSIONS

(1) Whilst the clinical picture of swayback is fairly characteristic, variations in the pathological manifestations have been observed.

(2) Though diagnostic when present, the inconstancy of the cerebral lesion, and its absence in some of the more severe cases, suggests that the fundamental lesion may be elsewhere.

(3) The interpretation of Marchi preparations of the spinal cord taken by routine methods should be treated with reserve.

(4) The presence of necrotic neurons is a useful aid to diagnosis. The suggestion of more widespread cellular changes indicates the need for further work.

(5) Similarities between normal fetal and swayback brains have been observed which would support the theory of myelin aplasia or dysplasia, if substantiated.

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