In view of the fact of the periodic occurrences of East Coast fever (as long as five years and more) on certain farms and in certain areas [Spitzkop (Sabie), Tours (Letaba)], it is strongly urged that no opportunity be lost of immediately applying the slaughter out policy in every case where East Coast fever has been diagnosed.

CONCLUSIONS.

1. It would appear that in East Coast fever areas in South Africa, the appearance of Koch's bodies in organ smears in a large percentage of single deaths must be attributed to T. mutans.

2. Under certain conditions T. mutans in non-East Coast fever areas may assume the pathological picture of T. parva from which it cannot be differentiated. It is, however, believed that under natural conditions in South Africa this seldom occurs. T. parva on the other hand causes mortality direct without the operation of other agencies without which T. mutans remains a harmless parasite.

3. Acute theileriasis in South Africa (T. parva and T. mutans) under certain conditions is associated with fairly characteristic changes in the lymphoid system particularly as revealed in the spleen and lymph glands. The pathogenesis of this is at the present moment not yet understood.

4. Although the Koch's bodies of T. mutans, after splenectomy, is of the nature of a "relapse", yet at the present moment it is not possible to state whether the greater majority of cases is of such nature, or a re-infection, probably with a different strain. If it is of the nature of a relapse, then the pathogenesis of the various agencies that can bring this about is not yet understood.

5. The present East Coast fever policy, with the full co-operation of the farming community should be persisted in to eradicate the disease in South Africa, and every endeavour should be made to carry out the slaughter out policy where East Coast fever has been diagnosed.

REFERENCES.


BOVINE THEILGRIASIS IN S. AFRICA.


ACKNOWLEDGMENTS.

It gives us great pleasure in thanking Dr. P. J. du Toit, the Director of Veterinary Services, for extending facilities to us to carry out these investigations and for his advice and guidance. The assistance and observations made by various Government Veterinary Officers especially by Mr. Diesel, Senior Veterinary Officer, Natal, and Mr. Lund, Government Veterinary Officer, Pietersburg, were of great value in these studies. The assistance rendered by Messrs. V. E. de Kock, C. Hall, and J. de Jager in preparing the tables and arranging the text, by Mr. T. Meyer for the photographs, by Miss Grobler for the typing, and by Miss Moolman for the preparation of the sections, is very greatly appreciated.

Note.—The studies in connection with the pathology and haematology and the main portion of the text as well as the summary and discussion were compiled by Dr. G. de Kock, the portion on the East Coast fever policy in South Africa was contributed by Mr. C. J. van Heerden, whereas Mr. Neitz and Mr. R. du Toit assisted in connection with the examination of smears, and the observations and collection of data in connection with the various exposure and transmission experiments carried out.
## APPENDIX I.

Single Deaths with the Occurrence of Koch’s Bodies in Organ Smear.

<table>
<thead>
<tr>
<th>Farm</th>
<th>District</th>
<th>Date of Death</th>
<th>Kind of Animal</th>
<th>Number of Cattle at Date of Outbreak</th>
<th>Technical Diagnosis</th>
</tr>
</thead>
<tbody>
<tr>
<td>Tank Area No. 184</td>
<td>Inanda</td>
<td>2. 6.33</td>
<td>Cow D.</td>
<td>1</td>
<td>2,400</td>
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<tr>
<td>HYDESWOOD</td>
<td>Escourt</td>
<td>Oct., 1933</td>
<td>Ox D.</td>
<td>1</td>
<td>208</td>
</tr>
<tr>
<td>CORNHILL</td>
<td>Ladysmith</td>
<td>16.12.33</td>
<td>Calf D.</td>
<td>1</td>
<td>116</td>
</tr>
<tr>
<td>Morgenstom</td>
<td>Dundee</td>
<td>21.12.33</td>
<td>Cow S.</td>
<td>1</td>
<td>154</td>
</tr>
<tr>
<td>The Chestnut House</td>
<td>Lions River</td>
<td>26.12.33</td>
<td>Calf D.</td>
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<td>425</td>
</tr>
<tr>
<td>Abadour</td>
<td>New Hanover</td>
<td>8. 1.34</td>
<td>Adult beast D.</td>
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<td>115</td>
</tr>
<tr>
<td>Moor</td>
<td>Impendible</td>
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<td>Cow S.</td>
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<td>473</td>
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<tr>
<td>Tank Area No. 22</td>
<td>Seings</td>
<td>15. 1.34</td>
<td>Calf eight months S.</td>
<td>1</td>
<td>17 R</td>
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<tr>
<td>Tank Area No. 123</td>
<td>Ixopo</td>
<td>15. 1.34</td>
<td>Calf D.</td>
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<td>80</td>
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<tr>
<td>Tank Area No. 78</td>
<td>Ixopo</td>
<td>18. 1.34</td>
<td>Calf D.</td>
<td>1</td>
<td>2,217</td>
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<tr>
<td>Hatamba</td>
<td>Ixopo</td>
<td>18. 1.34</td>
<td>Heifer S.</td>
<td>1</td>
<td>10</td>
</tr>
<tr>
<td>Tank Area No. 177</td>
<td>Inanda</td>
<td>21. 1.34</td>
<td>Cow S.</td>
<td>1</td>
<td>125</td>
</tr>
<tr>
<td>Hydevales</td>
<td>Ixopo</td>
<td>22. 1.34</td>
<td>Calf 3-4 months S.</td>
<td>1</td>
<td>131</td>
</tr>
<tr>
<td>Lea No. 83</td>
<td>Mtunzini</td>
<td>26. 1.34</td>
<td>Calf D.</td>
<td>1</td>
<td>3,216</td>
</tr>
<tr>
<td>Tank Area No. 108</td>
<td>Harding</td>
<td>59. 1.34</td>
<td>Heifer D.</td>
<td>1</td>
<td>643</td>
</tr>
<tr>
<td>Rietfontein</td>
<td>Estcourt</td>
<td>30. 1.34</td>
<td>Adult beast D.</td>
<td>1</td>
<td>53</td>
</tr>
<tr>
<td>Bethel</td>
<td>Vryheid</td>
<td>5. 2.34</td>
<td>Calf 15 months D.</td>
<td>1</td>
<td>2,217</td>
</tr>
<tr>
<td>Tank Area No. 907</td>
<td>Reserve No. 10</td>
<td>7. 2.34</td>
<td>Ox S.</td>
<td>1</td>
<td>125</td>
</tr>
<tr>
<td>Tank Area No. 643</td>
<td>Reserve No. 17</td>
<td>12. 2.34</td>
<td>Calf 3 weeks D.</td>
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<tr>
<td>Tank Area No. 20</td>
<td>Utrecht</td>
<td>12. 2.34</td>
<td>Calf D, 2 months...</td>
<td>1</td>
<td>3,216</td>
</tr>
<tr>
<td>Tank Area No. 81</td>
<td>Montague</td>
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<td>Calf S, 10 months...</td>
<td>1</td>
<td>7</td>
</tr>
<tr>
<td>Tank Area No. 112</td>
<td>Pinetown</td>
<td>18. 2.34</td>
<td>Bull S.</td>
<td>1</td>
<td>125</td>
</tr>
<tr>
<td>Tank Area No. 612</td>
<td>Reserve No. 11</td>
<td>24. 2.34</td>
<td>Calf 3 months S.</td>
<td>1</td>
<td>53</td>
</tr>
<tr>
<td>Canelot</td>
<td>Beesville</td>
<td>28. 2.34</td>
<td>Calf 5 months D.</td>
<td>1</td>
<td>2,621</td>
</tr>
<tr>
<td>Tank Area No. 605</td>
<td>Reserve No. 11</td>
<td>1. 3.34</td>
<td>Bull D.</td>
<td>1</td>
<td>125</td>
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<tr>
<td>Canelot</td>
<td>Reserve No. 11</td>
<td></td>
<td>Bull D.</td>
<td>1</td>
<td>3,401</td>
</tr>
<tr>
<td>MELMOTH VILLAGE</td>
<td>Entonjanei</td>
<td>2. 3.34</td>
<td>Cow D.</td>
<td>1</td>
<td>749</td>
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<tr>
<td>Tank Area No. 81</td>
<td>Port Shepstone</td>
<td>3. 3.34</td>
<td>Calf 5 months D.</td>
<td>1</td>
<td>1,739</td>
</tr>
<tr>
<td>Tank Area No. 644</td>
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<td>4. 3.34</td>
<td>Calf 3 weeks D.</td>
<td>1</td>
<td>2,039</td>
</tr>
<tr>
<td>Tank Area No. 624</td>
<td>Reserve No. 7b</td>
<td>5. 3.34</td>
<td>Calf 3 months D.</td>
<td>1</td>
<td>10</td>
</tr>
<tr>
<td>Farm</td>
<td>District</td>
<td>Date of Death</td>
<td>Kind of Animal</td>
<td>Number of Death</td>
<td>Ante Mortem Symptoms</td>
</tr>
<tr>
<td>-----------------------</td>
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<td>---------------</td>
<td>----------------</td>
<td>-----------------</td>
<td>------------------------------------</td>
</tr>
<tr>
<td>Etzell</td>
<td>Richmond</td>
<td>12.3.34</td>
<td>Calf 6 months D</td>
<td>1</td>
<td>Weak—poverty</td>
</tr>
<tr>
<td>U. 85</td>
<td>Hlabisa</td>
<td>12.3.34</td>
<td>Ox D</td>
<td>1</td>
<td></td>
</tr>
<tr>
<td>Wilbarons</td>
<td>Pet Relief</td>
<td>14.3.34</td>
<td>Bull D</td>
<td>1</td>
<td>Seedy for one day</td>
</tr>
<tr>
<td>Tank Area No. 614</td>
<td>Reserve No. 4,</td>
<td>26.3.34</td>
<td>Calf 8 weeks D</td>
<td>1</td>
<td>Poor from birth—always purging, Failed to suck for a week before death</td>
</tr>
<tr>
<td></td>
<td>Lower Umbolozi</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Tank Area No. 523</td>
<td>Hlabisa</td>
<td>19.4.34</td>
<td>Cow D</td>
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<td></td>
</tr>
<tr>
<td>Tank Area No. 487</td>
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<td>14.4.34</td>
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<tr>
<td>Clifford</td>
<td>Estcourt</td>
<td>21.11.35</td>
<td>Cow D</td>
<td>1</td>
<td>Very old animal</td>
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<tr>
<td>Wombat</td>
<td>Pietermaritzburg</td>
<td>25.11.35</td>
<td>Cow D</td>
<td>1</td>
<td></td>
</tr>
<tr>
<td>Hope Valley</td>
<td>Campedown</td>
<td>28.12.35</td>
<td>Calf 4 months D</td>
<td>1</td>
<td>Apparently healthy</td>
</tr>
<tr>
<td>Wonderfontein</td>
<td>New Hanover</td>
<td>1.1.36</td>
<td>Cow S</td>
<td>1</td>
<td>Sick for 1 day, weak, staggering gait, trembling</td>
</tr>
<tr>
<td>Dickschenberg No. 163</td>
<td>Etonjanou</td>
<td>10.1.36</td>
<td>Calf 3 weeks D</td>
<td>1</td>
<td>Died suddenly</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Lilliefontein</td>
<td>Pietermaritzburg</td>
<td>16.1.36</td>
<td>Calf 5 months D</td>
<td>1</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Vryheid</td>
<td>18.1.36</td>
<td>Ox S</td>
<td>1</td>
<td></td>
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<td>Inyati Trading Store</td>
<td>Langrans No. 367</td>
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<tr>
<td>Weverdorn No. 630</td>
<td>Ngotsho</td>
<td>20.1.36</td>
<td>Cow 4 months D</td>
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<tr>
<td>Tank Area No. 17</td>
<td>Klip River Location,</td>
<td>20.1.36</td>
<td>Calf 1 month D</td>
<td>1</td>
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</tr>
<tr>
<td>Tank Area No. 548</td>
<td>Reserve No. 20, Mahlabatini</td>
<td>20.1.36</td>
<td>Calf 2 months D</td>
<td>1</td>
<td></td>
</tr>
<tr>
<td>Tank Area No. 547</td>
<td>Reserve No. 20, Mahlabatini</td>
<td>23.1.36</td>
<td>Ox 3 years S</td>
<td>1</td>
<td></td>
</tr>
<tr>
<td>Tank Area No. 178</td>
<td>New Hanover</td>
<td>29.1.36</td>
<td>Ox S</td>
<td>1</td>
<td>Broken leg</td>
</tr>
<tr>
<td></td>
<td>Vryheid</td>
<td>2.2.36</td>
<td>Calf 1 year S</td>
<td>1</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Reserve No. 21</td>
<td>6.2.36</td>
<td>1 Bull D</td>
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<td></td>
</tr>
<tr>
<td>Tank Area No. 537</td>
<td>Reserve No. 12, Nongoma</td>
<td>7.2.36</td>
<td>Bull 2 years S</td>
<td>1</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
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<tr>
<td>Tank Area No. 72</td>
<td>Umzisco</td>
<td>9.2.36</td>
<td>Cow S</td>
<td>1</td>
<td>Very poor condition</td>
</tr>
<tr>
<td>Tank Area No. 505</td>
<td>Reserve No. 16, Ingwavuma</td>
<td>14.2.36</td>
<td>Cow D</td>
<td>1</td>
<td></td>
</tr>
<tr>
<td>Tank Area No. 471</td>
<td>Pinetown</td>
<td>22.2.36</td>
<td>Calf 6 months D</td>
<td>1</td>
<td>Ill for 2 days</td>
</tr>
<tr>
<td>Lot Nos. 277-303</td>
<td>Lower Umbolozi</td>
<td>24.2.36</td>
<td>Calf 4 months D</td>
<td>1</td>
<td>Sick for 3 days, recovering—died 4 days later</td>
</tr>
<tr>
<td>Tank Area No. 662</td>
<td>Reserve No. 21, Eshowe</td>
<td>26.2.36</td>
<td>Calf 3 months D</td>
<td>1</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Koningdal No. 220</td>
<td>Bambanango</td>
<td>26.2.36</td>
<td>Heifer 10 months D</td>
<td>1</td>
<td></td>
</tr>
<tr>
<td>Dartall Butchery</td>
<td>Lower Tagela</td>
<td>29.2.36</td>
<td>Ox S</td>
<td>1</td>
<td></td>
</tr>
<tr>
<td>Tank Area No. 605</td>
<td>Reserve No. 12, Nongoma</td>
<td>4.3.36</td>
<td>Heifer calf 3–12 months D</td>
<td>1</td>
<td>Ill for 6 days, diarrhoea in-appetence</td>
</tr>
</tbody>
</table>
## APPENDIX I (continued).

<table>
<thead>
<tr>
<th></th>
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<tbody>
<tr>
<td>Lot No. 92A</td>
<td>Mtnzini</td>
<td>6.3.36</td>
<td>Calf 5 months S.</td>
<td>1</td>
<td>Calf became very poor in condition because of having been deprived of milk by native herd-boy</td>
<td>57</td>
<td>R</td>
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<tr>
<td>Pulumbane</td>
<td>Umtaa</td>
<td>7.3.36</td>
<td>Cow S.</td>
<td>1</td>
<td>Died suddenly</td>
<td>507</td>
<td>F</td>
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<tr>
<td>Doornkop</td>
<td>Newcastle</td>
<td>8.3.36</td>
<td>Calf 1 year D.</td>
<td>1</td>
<td>Calf sick for week with watery diarrhoea</td>
<td>131</td>
<td>+</td>
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<tr>
<td>Fugitives Drift</td>
<td>Helmpenkaal</td>
<td>13.3.36</td>
<td>Calf 8 months D.</td>
<td>1</td>
<td>Sick for 6 weeks, purging, loss of condition, and emaciation</td>
<td>2,817</td>
<td>+</td>
</tr>
<tr>
<td>Tank Area No. 681</td>
<td>Reserve No. 5 Lower Umfolozi</td>
<td>16.3.36</td>
<td>Calf 3½ months D.</td>
<td>1</td>
<td>Sick for 18 days, depression, drooping ears, staring coat, weakness and later diarrhoea</td>
<td>1,999</td>
<td>+</td>
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<td>Tank Area No. 641</td>
<td>Reserve No. 17 Exhove</td>
<td>21.3.36</td>
<td>Cow D.</td>
<td>1</td>
<td>Sick for 1 day—had diarrhoea</td>
<td>666</td>
<td>+ R</td>
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<tr>
<td>Boschhoek No. 489</td>
<td>Vryheid</td>
<td>29.3.36</td>
<td>Calf 5 months D.</td>
<td>1</td>
<td>Sick for 4 days</td>
<td>2,084</td>
<td>F</td>
</tr>
<tr>
<td>Tank Area No. 49</td>
<td>Mapumulo</td>
<td>4.4.36</td>
<td>Tolle 18 months D.</td>
<td>1</td>
<td>Broken horn</td>
<td>4,265</td>
<td>+</td>
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<tr>
<td>Transvaal</td>
<td>Letaba Estates Pietersburg (Letaba)</td>
<td>1.3.33</td>
<td>Ox S.</td>
<td>1</td>
<td>Heifer + 2 years D.</td>
<td>30</td>
<td>+ R</td>
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<td>Grootplaats No. 54 Pietersburg (Grootspelonken E.C.F. Area)</td>
<td>2.3.33</td>
<td>Ox D.</td>
<td>1</td>
<td>Heifer + 3 months D.</td>
<td>36</td>
<td>+ R</td>
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<td>Mohlaba's Location Pietersburg (Letaba)</td>
<td>10.3.33</td>
<td>Ox D.</td>
<td>1</td>
<td>Heifer + 6 months D.</td>
<td>28</td>
<td>+ (P. bigem)</td>
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<tr>
<td>Lille No. 380 Pietersburg (Letaba)</td>
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<td></td>
<td>1</td>
<td>Heifer + 8 months D.</td>
<td>30</td>
<td>+ (P. bigem)</td>
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<tr>
<td>Uitkyk Pietersburg (Letaba)</td>
<td>4.8.33</td>
<td>Calf + 6 months D.</td>
<td>1</td>
<td>Calf + 9 months D.</td>
<td>36</td>
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<tr>
<td>Klipbank No. 1672 (E.C.F. Area, Pietersburg) Grootspelonken</td>
<td>20.12.33</td>
<td>Calf + 3 months D.</td>
<td>1</td>
<td>Calf + 12 months D.</td>
<td>30</td>
<td>+ (P. bigem)</td>
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<tr>
<td>Poliati Butchery (Maldersplant) Pietersburg (Letaba)</td>
<td>15.5.34</td>
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<td>1</td>
<td>Heifer + 1 year D.</td>
<td>32</td>
<td>F</td>
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<td>Loudenraas Pietersburg (Letaba)</td>
<td>2.6.34</td>
<td>Calf (f)</td>
<td>1</td>
<td>Calf (f)</td>
<td>32</td>
<td>+ R</td>
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<tr>
<td>Turkeyse Pietersburg (Letaba)</td>
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<td>Calf (f)</td>
<td>1</td>
<td>Calf (f)</td>
<td>32</td>
<td>+ R</td>
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<td>Boschkopje Pietersburg (Grootspelonken Area)</td>
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<td>Calf + 3 months (f)</td>
<td>1</td>
<td>Calf + 6 months (f)</td>
<td>32</td>
<td>+ R</td>
<td></td>
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<tr>
<td>Witvijver No. 14</td>
<td>Piet Retief</td>
<td>23.1.34</td>
<td>Calf D.</td>
<td>1</td>
<td>Calf D.</td>
<td>32</td>
<td>+ R</td>
</tr>
<tr>
<td>Elandsdrift</td>
<td>Pelgrimraast</td>
<td>10.2.35</td>
<td>Ox (f)</td>
<td>1</td>
<td>Ox (f)</td>
<td>32</td>
<td>+ R</td>
</tr>
<tr>
<td>Beginel No. 181</td>
<td>Piet Retief</td>
<td>22.2.35</td>
<td>Calf (f)</td>
<td>1</td>
<td>Calf (f)</td>
<td>50</td>
<td>+ R</td>
</tr>
<tr>
<td>Makwakweni No. 37</td>
<td>Piet Retief</td>
<td>23.2.35</td>
<td>Heifer (f)</td>
<td>1</td>
<td>Heifer (f)</td>
<td>32</td>
<td>+ R</td>
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</tbody>
</table>
### APPENDIX I (continued)

<table>
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<tr>
<th>Farm</th>
<th>District</th>
<th>Date of Death</th>
<th>Kind of Animal</th>
<th>Number of Death</th>
<th>Ante Mortem Symptoms</th>
<th>Number of Cattle at Date of Outbreak</th>
<th>Technical Diagnosis</th>
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<tbody>
<tr>
<td>Manchester No. 179...</td>
<td>Nelspruit (Mlume)</td>
<td>31. 3.35</td>
<td>Calf S.</td>
<td>1</td>
<td></td>
<td>192</td>
<td>R</td>
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<tr>
<td>Rodevlakte No. 454...</td>
<td>Pietersburg (Mlume)</td>
<td>17. 5.35</td>
<td>Ox S.</td>
<td>1</td>
<td></td>
<td>129</td>
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<td><strong>NATAL</strong></td>
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<td>Umzinto Village.......</td>
<td>Umzinto</td>
<td>Dec. 1933</td>
<td>Oxen S.</td>
<td>16</td>
<td>All apparently healthy...</td>
<td>1728</td>
<td>+</td>
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<td>Tank Area No. 71......</td>
<td>Umzinto</td>
<td>17. 1.34</td>
<td>Oxen D.</td>
<td>2</td>
<td></td>
<td>268</td>
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<td>Vryheid Town..........</td>
<td>Vryheid</td>
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<td>Oxen S.</td>
<td>2</td>
<td>All apparently healthy...</td>
<td>864</td>
<td>+</td>
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<td>Empangeni Village.....</td>
<td>Lower Umlilozi</td>
<td>22. 1.34</td>
<td>Oxen S.</td>
<td>2</td>
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<td>854</td>
<td>-</td>
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<tr>
<td>Mooiplasen Witkloof</td>
<td>Ngotsho</td>
<td>25. 2.34</td>
<td>Cow D.</td>
<td>2</td>
<td></td>
<td>122</td>
<td>+</td>
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<td><strong>T</strong></td>
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<td></td>
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<td>Reserve No. 9,</td>
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<td>2</td>
<td></td>
<td>3615</td>
<td>+</td>
</tr>
<tr>
<td></td>
<td>Mtunzini</td>
<td>13. 3.34</td>
<td>Cow S.</td>
<td></td>
<td></td>
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<td></td>
<td></td>
<td>4.10.35</td>
<td>Ox S.</td>
<td></td>
<td></td>
<td>60</td>
<td>+ R</td>
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<td>5.10.35</td>
<td>Bull S.</td>
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<td></td>
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<tr>
<td>Dukumbane</td>
<td>Hlabisa</td>
<td>2-4.1.36</td>
<td>7 Cows D.</td>
<td>8</td>
<td>Poverty stricken—heavy rains</td>
<td>122</td>
<td>+</td>
</tr>
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<td>Reserve No. 16,</td>
<td>20-21.1.36</td>
<td>3 Bulls S.</td>
<td>4</td>
<td></td>
<td>3529</td>
<td>+</td>
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<tr>
<td></td>
<td>Ingwavuma</td>
<td>20. 1.36</td>
<td>Cow S.</td>
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<td>Reserve No. 12,</td>
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<td>Cow D.</td>
<td>2</td>
<td></td>
<td>1290</td>
<td>+</td>
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<tr>
<td></td>
<td>Hlabisa</td>
<td>13. 2.36</td>
<td>Cow D.</td>
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<tr>
<td></td>
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<td>30. 3.36</td>
<td>Ox D.</td>
<td></td>
<td></td>
<td>34</td>
<td>R</td>
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<td></td>
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<td></td>
<td></td>
<td>V.R.</td>
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<tr>
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<td></td>
<td></td>
<td></td>
<td>+ P. ligen.</td>
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<td>Tank Area No. 462.....</td>
<td>Ndwedwe</td>
<td>3. 3.35</td>
<td>Cow S.</td>
<td>3</td>
<td></td>
<td></td>
<td>+</td>
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<tr>
<td></td>
<td></td>
<td>7. 3.35</td>
<td>Beast D.</td>
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<td>8. 3.35</td>
<td>Beast D.</td>
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<td>Tzaneen Village</td>
<td>Pietersburg (Letaba)</td>
<td>6. 3.33</td>
<td>Oxen S.</td>
<td>3</td>
<td>In poor condition</td>
<td>12</td>
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<tr>
<td></td>
<td></td>
<td>3. 6.33</td>
<td>S</td>
<td>8</td>
<td>F</td>
<td>+</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>3. 7.33</td>
<td>D</td>
<td>6</td>
<td>F</td>
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<td></td>
<td></td>
<td>27.12.33</td>
<td>Cow 2½ years D.</td>
<td>3</td>
<td></td>
<td>1,651</td>
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<td></td>
<td></td>
<td>23. 1.34</td>
<td>Beast (?)</td>
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<td></td>
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<td></td>
<td>23. 1.34</td>
<td>Ox S.</td>
<td>1</td>
<td></td>
<td>100</td>
<td>F</td>
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<td></td>
<td>18. 1.34</td>
<td>Adult beast D.</td>
<td>1</td>
<td></td>
<td>145</td>
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<td></td>
<td>21. 2.33</td>
<td>Cow S.</td>
<td>1</td>
<td></td>
<td>135</td>
<td>F</td>
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<td></td>
<td></td>
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<td>Cow S.</td>
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<tr>
<td></td>
<td></td>
<td>7. 3.36</td>
<td>Calf 4 months D.</td>
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<td></td>
<td>3,753</td>
<td>F</td>
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<td>Calf D.</td>
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<td></td>
<td>3,228</td>
<td>F</td>
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<td>31. 1.36</td>
<td>Bull 2 years D.</td>
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<td></td>
<td>2,156</td>
<td>F</td>
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<td>6. 3.34</td>
<td>Cow D.</td>
<td>2</td>
<td></td>
<td>2,044</td>
<td>F</td>
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<tr>
<td></td>
<td></td>
<td>3. 2.36</td>
<td>Heifer S.</td>
<td>1</td>
<td></td>
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<td>26. 2.34</td>
<td>Calf 6 weeks D.</td>
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<td></td>
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<td>12. 2.33</td>
<td>Calf 4 months D.</td>
<td>1</td>
<td></td>
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<td>27. 6.34</td>
<td>Calf D.</td>
<td>1</td>
<td></td>
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<td></td>
</tr>
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<td></td>
<td></td>
<td>28. 2.33</td>
<td>Cow S.</td>
<td>1</td>
<td></td>
<td></td>
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</tr>
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<td></td>
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<td>2. 2.35</td>
<td>Bull S.</td>
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## APPENDIX I (continued)

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<th>Farm</th>
<th>District</th>
<th>Date of Death</th>
<th>Kind of Animal</th>
<th>Number of Deaths</th>
<th>Ante-mortem Symptoms</th>
<th>Post-mortem Symptoms</th>
<th>Technical Diagnosis</th>
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<tr>
<td>Lot No. K. 34</td>
<td>Lower Umfolozi</td>
<td>16. 7.34</td>
<td>2-3 Years heifer D.</td>
<td>1</td>
<td>Sick for 3-4 days</td>
<td>+</td>
<td>+</td>
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<tr>
<td>Lot No. 275</td>
<td>Lower Umfolozi</td>
<td>18. 8.34</td>
<td>(?) Cow K.</td>
<td>1</td>
<td>—</td>
<td>—</td>
<td>+</td>
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<td>T/A. 697, Nkwele: Nongoma</td>
<td>Reserve No. 12</td>
<td>10. 8.34</td>
<td>Cow K.</td>
<td>1</td>
<td>—</td>
<td>—</td>
<td>+</td>
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<tr>
<td>H. 21/73, Hluhluwe Settlement</td>
<td>Hlabisa</td>
<td>22.10.34</td>
<td>Old Bull D.</td>
<td>2</td>
<td>Both weak and emaciated for weeks—showed symptoms of Nagana</td>
<td>—</td>
<td>+</td>
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<tr>
<td>Lot No. 25</td>
<td>Mtunzini</td>
<td>19. 9.35</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>R.W.</td>
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<tr>
<td>Erindale</td>
<td>Vryheid (on Miala-batini border)</td>
<td>10.11.35</td>
<td>Cow D.</td>
<td>1</td>
<td>Cow sick before it died</td>
<td>—</td>
<td>+</td>
</tr>
<tr>
<td>Langverwacht No. 561, Jockstown</td>
<td>Ngotshe</td>
<td>24.11.35</td>
<td>Beast</td>
<td>1</td>
<td>—</td>
<td>—</td>
<td>+</td>
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<tr>
<td></td>
<td>Newcastle</td>
<td>15. 1.36</td>
<td>Old cow D.</td>
<td>1</td>
<td>—</td>
<td>—</td>
<td>F</td>
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<td>Greylock</td>
<td>Klip River</td>
<td>20. 1.36</td>
<td>Beast</td>
<td>1</td>
<td>—</td>
<td>—</td>
<td>P. tenuicaudae</td>
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<td>Lots No. K. 68, 61, 62</td>
<td>Lower Umfolozi</td>
<td>28. 1.36</td>
<td>Cow 5 years D.</td>
<td>1</td>
<td>Cow ill for 1 day and symptoms suggest Heartwater</td>
<td>—</td>
<td>+</td>
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<tr>
<td>Hermanusburg Mission</td>
<td>Umvoti</td>
<td>30. 1.36</td>
<td>Bull 18 months D.</td>
<td>1</td>
<td>—</td>
<td>—</td>
<td>+</td>
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<tr>
<td>Groenvlei</td>
<td>Lions River</td>
<td>31. 1.36</td>
<td>Adult D.</td>
<td>1</td>
<td>—</td>
<td>—</td>
<td>+</td>
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<tr>
<td>Springfield</td>
<td>Dundee</td>
<td>21. 2.36</td>
<td>Ox 21 years D.</td>
<td>1</td>
<td>—</td>
<td>—</td>
<td>+</td>
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<tr>
<td>II.</td>
<td></td>
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<tr>
<td>No. 335, Mapotwa</td>
<td>Reserve No. 19, Nkandla</td>
<td>24. 2.36</td>
<td>Beast</td>
<td>1</td>
<td>—</td>
<td>—</td>
<td>+</td>
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<tr>
<td>Puzela Plot (Taaneen)</td>
<td>Pietersburg (Letaba)</td>
<td>22. 5.34</td>
<td>Ox S.</td>
<td>1</td>
<td>—</td>
<td>—</td>
<td>Poverty in blood</td>
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<td>Alma No. 219</td>
<td>Piet Retief</td>
<td>31. 1.36</td>
<td>Calf D.</td>
<td>1</td>
<td>—</td>
<td>—</td>
<td>+</td>
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APPENDIX II.

Pathology.—Tzaneen Exposure Experiments.

In the description of the pathological and haematological findings in the various experiments carried out only those aspects will be stressed in which abnormal and specific changes were observed. A more detailed cytological study of some of the lesions observed, especially in respect of the lymphocytic proliferations in the various organs, the changes in respect of the lymphoid tissue in various parts of the body, the peculiar lesions observed in the spleen, the significance of the cytological studies observed in the smears and organs, etc., will be left for a later discussion.

The pathology of the various cases in experiment will be dealt with according to the experiments carried out.

A. Tzaneen Experiment, 1934.

C. 4929. Specimen No. 14991.

Pathological Anatomy.—Ears markedly affected as a result of tick infestation; anaemia; urine normal.

Etiology.—Sequel piroplasmosis.

Histology.—Liver: columns of liver cells slightly distorted but stain fairly well; no evidence of lymphoid hyperplasia; bile stasis around the central vein prominent; neutrophiles frequent in the intralobular capillaries. Kidney: no lymphoid hyperplasia observed. Spleen: large amount of blood in the red pulp, encroaching on the lymphoid tissue. Lymph glands: no specific changes observed in connection with the distribution of the lymphoid tissue.


C. 4830. Specimen No. 14934.

Killed at Tzaneen for post-mortem.

Pathological Anatomy.—Anaemia; haemoglobinuria; small greyish focus, about 2 mm. in diameter on the surface of the kidney; parapharyngeal lymph gland swollen and oedematous; sub-parotid lymph gland swollen and oedematous, and in one of the latter a small localised suppuration identified; external ears haemorrhages, suppuration, necrosis and partial gangrene, with portions sloughing off. Spleen: slightly swollen and pulpa somewhat granular and dry; slight serous atrophy of the adipose tissue; no evidence of icterus; apical lobe of left lung localised, hepatisation and necrosis, about 1 inch in diameter.

Etiology.—Pyroplasmosis.

Histology.—Liver: no lymphoid hyperplasia; a fair number of neutrophiles in the periphery of the lobule and in the intralobular capillaries; liver cells stain fairly well. Kidneys: no lymphoid hyperplasia. Lymph glands: in places neutrophiles not infrequent; there is evidence of a reduction of the lymphocytic cells in respect of the follicles although these are not prominently present. Spleen: in places clusters of neutrophiles, where they form small foci outside the white pulpa.

Examination of Smears.—Blood: P. bigem. not infrequent; anisocytosis. Kidney and spleen: similar changes observed except in the spleen T. mutans found very rare.
BOVINE THERLIERIASIS IN S. AFRICA.

C. 4849. Specimen No. 15023.

Pathological Anatomy.—Slight hydrothorax and slight hydropericardium; several opaque foci in the liver; haemoglobinuria.

Etiology.—Piroplasmosis.

Histology.—Liver: encapsulated abscess; no lymphoid hyperplasia; fairly extensive bile stasis around the central vein. Kidneys: no lymphoid hyperplasia. Spleen: large amount of blood in the red pulpa.

Examination of smears.—Blood: P. bigem. fairly frequent; T. mutans rare. In spleen and mesenteric, subparotid, periportal, supra-scapular, precural, mediastinal lymph glands, liver and kidney, besides above parasites mentioned no Koch’s bodies observed.

C. 4836. Specimen No. 14977.

Pathological Anatomy.—Sub-parotid lymph gland very much enlarged with necrosis; anaemia; hydrothorax and hydropericardium; pigmentation liver; urine normal colour.

Etiology.—Piroplasmosis.


Examination of smears.—Blood: P. bigem. and T. mutans rare; basophilic; polychromasia; anisocytosis; Jolly Bodies. Kidney, liver, spleen, periportal, inguinal, supra-scapular, precural, sub-parotid and mediastinal lymph glands, besides the above parasites showed no Koch’s bodies.

C. 4937. Specimen No. 1504.

Pathological Anatomy.—Slight swelling of the precural and supra-scapular lymph glands.

Etiology.—Piroplasmosis.

Histology.—Liver: increase in leucocytes in the periphery; large numbers of neutrophiles in the intralobular capillaries; no lymphoid hyperplasia. Kidney: no lymphoid hyperplasia. Lung and myocard: no specific changes. Lymph gland: in some of the secondary follicles there are remains of karyorrhexis. Spleen: a good deal of blood in the red pulpa; in places neutrophiles are frequent.

Examination of smears.—Blood: T. mutans very rare, P. bigem. rare; large number of neutrophiles present. Spleen, liver, mesenteric, supra-scapular, precural, sub-parotid, and periportal lymph glands, besides above parasites no Koch’s bodies.

N.B.—Smear from the kidney shows P. bigem. frequent.

C. 4967. Specimen No. 14960.

Pathological Anatomy.—Anaemia; urine normal; hyperaemia and oedema of the lungs.

Etiology.—Sequel piroplasmosis.

Histology.—Liver: a good deal of blood in the intra-lobular capillaries amongst which a number of leucocytes can be identified; proliferation evidenced in the periphery with the presence of a fair number of lymphocytes, not of the nature of lymphoid hyperplasia; bile stasis fairly prominent. Kidneys: no lymphoid hyperplasia. Spleen: large amount of blood in the red pulpa; lymphoid tissue prominent. Lymph gland: lymphoid tissue not prominent; blood vessels distended.

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Examination of smears.—Blood: P. bigem. rare; putrefactive bacteria; slight anisocytosis. Precrural, and supra-escapular lymph glands and spleen no evidence of Koch’s bodies.

Pathological Anatomy.—Specimen No. 14933.

Marked general anaemia; slight icterus; a moderate amount of swelling of the spleen; oedema and swelling of a number of lymphatic glands, especially the pre-escapular and sub-parotid; slight serous atrophy of the adipose tissue; slight swelling and icterus of the liver; haemoglobinemia and haemoglobinuria; pigmentation of the kidneys.

Numerous brown ticks in the ears, with slight haemorrhages.

Histology.—Liver: leucocytes prominent in the periphery; nothing of the nature of hyperplasia; bile stasis prominent besides bile pigmentation in the liver as well. Kidneys: no lymphoid hyperplasia. Spleen: a good deal of blood in the red pulp with clusters of neutrophiles in places. Myocardium: nothing unusual. Lymph glands: the lymphoid tissue fairly prominent; in some of them a fair number of neutrophiles present.

Examination of smears.—Spleen: P. bigem. not infrequent; lymphocytes with granules observed; slight anisocytosis and polychromasia; T. mutans rare. Sub-parotid lymphatic gland: many bacteria present in the smear. Parapharyngeal lymphatic gland: P. bigem. not infrequent.

Pathological Anatomy.—Specimen No. 15038.

Pathological Anatomy.—Slight post mortem changes; emaciation; cachexia; anaemia; petechiae and ecchymoses epicardium and myocardium; swelling and oedema of the lymph glands; swelling of the liver with degenerative changes; slight haemorrhages and the presence of sand in the abomasum; a slight acute catarrhal enteritis; hydropiccardium.

Histology.—Liver: slight hyperaemia and leucostasis in the periphery and in the intra-lobular capillaries; slight fatty changes around the periphery; no lymphoid hyperplasia. Kidneys: no lymphoid hyperplasia. Spleen: in places neutrophiles are prominent. Lymph glands: in places neutrophiles are prominent. Lungs: no specific changes.

Examination of smears.—Blood: P. bigem. rare; slight anisocytosis; basophilia, neutrophiles frequent. The lymphatic gland smear unsatisfactory but no Koch’s bodies identified.

Pathological Anatomy.—Specimen No. 15087.

Pathological Anatomy.—General anaemia and cachexia; oedema and swelling of the majority of the lymphatic glands; hyperaemia and swelling of the liver and kidneys; slight tumor spleen; fairly extensive oedema of the lungs; localised hepatisation with multiple necrosis and gangrene, both lungs; slight acute mucous catarrhal enteritis; few monezia in small intestines; parasitic nodules parapharyngeal and supra-mammary lymph glands.

Histology.—Liver: columns and liver cells somewhat distorted but stain fairly well; no evidence of lymphoid hyperplasia; bile stasis prominent around the central vein; neutrophiles frequent in the intra-lobular capillaries. Kidney: no lymphoid hyperplasia. Spleen: large amount of blood in the red pulp. Lymph glands: no specific changes.

Examination of smears.—Intima smears: Rickettsia ruminantium very rare, P. bigem. rare. Blood: slight anisocytosis; T. mutans very rare; no Koch’s bodies seen in spleen and pre-escapular lymph glands.
BOVINE THEILEIASIS IN S. AFRICA.

C. 5562. Specimen No. 4961.

Pathological Anatomy.—Anaemia; haemoglobinuria; swelling of the majority of lymphatic glands; slight tumor splenis.

Etiology.—Sequel piroplasmosis.

Histology.—Liver: necrosis around the central veins; bile stasis prominent on the periphery of this necrosis; no lymphoid hyperplasia. Kidneys: no lymphoid hyperplasia; haemosiderosis. Spleen: multiple necrotic foci.

Examination of smears.—Spleen: slight anisocytosis; T. mutans rare, Supra-epaculiar lymph gland: P. bigem. rare. Sub-parotid lymph gland: P. bigem. not infrequent; slight anisocytosis. Medastinal and inguinal lymph glands: P. bigem. not infrequent; slight anisocytosis. In none of these any evidence of Koch's bodies.

C. 5510. Specimen No. 14978.

Pathological Anatomy.—Hydrothorax; hydropericardium; urine in bladder normal.

Etiology.—Sequel (?).

Histology.—Liver: a good deal of blood in the intra-lobular capillaries, in which lymphocytes are prominent; proliferation of lymphocytes in the periphery but not of the nature of a hyperplasia. Kidneys: no lymphoid hyperplasia. Spleen: a good deal of blood in the red pulp; lymphoid tissue not prominent. Lymph gland: lymphoid tissue not prominent, especially in respect of the number of nodules.

Examination of smears.—Blood: T. mutans frequent. Kidney, liver, spleen, periportal, inguinal, supra-epacicular, prerural, sub-parotid and mediastinal lymph glands, besides the above no evidence of Koch's bodies.

C. 5520. Specimen No. 14248.

Pathological Anatomy.—Post mortem changes present; oedema of the subcutis; tumor splenis; general anaemia; general icterus; haemoglobinanaemia; haemoglobinuria; hyperaemia and oedema of the lungs.

Etiology.—Piroplasmosis.


Examination of smears.—Blood: P. bigem. not infrequent; T. mutans rare; slight anisocytosis. Intima smear negative. Prescapular lymph gland: no Koch's bodies.

C. 5524.

See Daily Report 15739 of 7.5.34, wherein it is stated that this animal died suddenly on the day the other animals were being trucked at Tzaneen to be returned to Pretoria. No post mortem could be made and cause of death was therefore not ascertained.

C. 5209. Specimen No. 14935.

Pathological Anatomy.—Post mortem changes present; no signs of lymphoid hyperplasia; moderate amount of swelling of some of the lymphatic glands and the spleen.

Etiology.—Piroplasmosis.

Histology.—Liver and kidneys decomposed; no evidence of lymphoid hyperplasia.
Examination of smears.—T. mutans not infrequent; slight anisocytosis; putrefactive bacteria. Spleen and sub-parotid lymph glands: T. mutans not infrequent; slight anisocytosis; putrefactive bacteria; no evidence of Koch’s bodies.

C. 5214. Specimen No. 14980.

Pathological Anatomy.—Ears badly affected; slight swelling of precrural and supra-scapular lymph glands; slight hydropericardium; urine in bladder normal.

Etiology.—Sequel piroplasmosis.

Histology.—Liver: no lymphoid hyperplasia; a number of neutrophiles in the intra-lobular capillaries; liver cells stain fairly well. Kidneys: no lymphoid hyperplasia. Spleen: clusters of neutrophiles prominent. Lymph glands: no specific changes.

Examination of smears.—Blood: anisocytosis; neutrophiles not infrequent; P. bigem. rare; T. mutans very rare; lymphocytes with clusters of granules observed. Inguinal, supra-scapular, periportal, mediastinal, sub-parotid lymph glands, kidney and liver: no evidence of Koch’s bodies.

C. 5224. Specimen No. 14999.

This animal was extremely wild and could not be properly controlled. It was therefore destroyed and its blood and tissues were utilized as controls for the above studies.

Pathological Anatomy.—Liver: in places the connective tissue in the periphery very prominent, resembling a slight cirrhosis. Leucocytes prominent in the intra-lobular capillaries. Kidneys: nothing unusual.

Etiology.—Killed. Too wild.

Examination of smears.—Blood: periportal lymphatic gland, mediastinal lymph gland and spleen: no Koch’s bodies observed.

C. 5228. Specimen No. 14993.

Pathological Anatomy.—Anaemia; multiple greyish foci in the liver.

Etiology.—Piroplasmosis.

Histology.—Liver: bile stasis prominent; slight proliferation with the presence of lymphocytes in the periphery; bile pigmentation liver cells; slight fatty changes around the periphery of the lobule; lymphocytes prominent in intra-lobular capillaries. Kidneys: no evidence of a lymphoid hyperplasia; haemosiderosis. Spleen: a large amount of blood in the red pulpa, encroaching on to the white pulpa. Lymph glands: no specific changes.

Examination of smears.—Blood: P. bigem. frequent; T. mutans rare; anisocytosis; basophilia; polychromasia. Liver, spleen, kidney, supra-scapular, precrural, parotid, mediastinal, periportal and inguinal lymphatic glands: no Koch’s bodies observed.

C. 5272. Specimen No. 14992.

Pathological Anatomy.—Multiple greyish foci liver; hepatization left lung.

Etiology.—Sequel piroplasmosis.

Histology.—Kidneys and liver: no evidence of lymphoid hyperplasia; post mortem changes present; spleen: a good deal of blood in red pulpa. Myocardium: localized fibrosis. Lungs: acute pneumonia with necrosis, probably sequel of dermatitis of external ears, as a result of bad tick infestation.

Examination of smears.—Blood: P. bigem rare; T. mutans very rare. Liver, kidney, supra-scapular, precrural, sub-parotid, mediastinal, inguinal lymph glands and spleen: no evidence of Koch’s bodies.
BOVINE THEILERIASIS IN S. AFRICA.

C. 5297. Specimen No. 14994.

Pathological Anatomy.—Hepatization right lung; anaemia.

Etiology.—Piroplasmosis.

Histology.—Liver and kidney decomposed; no evidence of hyperplasia.


C. 5373. Specimen No. 14936.

Pathological Anatomy.—Anaemia; slight serous atrophy of the adipose tissue; slight icterus; bile pigmentation urine.

Etiology.—Killed for collection of specimens.

Histology.—Liver: slight proliferation of lymphocytes in the periphery. Kidney: associated with the adventitia of the blood vessels in the medulla; there are in two places accumulations of lymphocytes; this has been seen in other animals in which no lymphoid hyperplasia could be identified in the cortex. Spleen: a good deal of blood and neutrophiles observed in the red pulpa; lymphoid tissue prominent. Lungs: nothing unusual.

Examination of smears.—Blood: slight anisocytosis. Mediastinal lymph gland and spleen: no Koch's bodies observed.

C. 5386. Specimen No. 55022.

Etiology.—Sequel piroplasmosis.

Histology.—Liver: increase of lymphocytes in the intralobular capillaries. Kidney: in the adventitia of the blood vessels there is a proliferation of lymphocytes, not of the nature of the true lymphoid hyperplasia. Spleen: large amount of blood in the red pulpa. Lymph glands: no specific changes seen.

Examination of smears.—Blood: slight anisocytosis and leucocytosis; T. mutans rare. In spleen, kidney, liver, inguinal gland, sub-parotid, supra-ocular and precrural lymph glands no Koch's bodies identified.

B. TANEEK EXPERIMENT, 1935.

C. 4872. Specimen No. 16151.

Pathological Anatomy.—Bases of the ears swollen; purulent discharge from both external ears; the skin in the region of the base swollen, whereas the subcutis in this vicinity is oedematous and sanguinous; necrosis of the fat of the sulci of the epicard; a purulent necrotic dermatitis external ears; haemorrhagic suppurative lymphadenitis; enlargement of all the lymph glands; necrotic focus lungs; tumor spleen; enlargement of the liver; multiple ulcers abomasum; localised fibrous pleuritis.

Etiology.—Killed in extremis. Sequeal T. mutans and badly affected ears result of heavy tick infestation.

Histology.—Liver: slight accumulation of lymphocytes around the vessels of the periphery; lymphocytes and neutrophiles not infrequent in the intralobular capillaries evidence of hyaline droplet degeneration in some of the liver cells. Kidneys: no lymphoid hyperplasia. Lymph glands: in places small foci of cells showing karyorrhexis; suppression of accumulations of small lymphocytes in the formation of nodules; in some of the glands the reticular cells of the secondary follicles show vacuolation. Spleen: follicles not distinct and in some of them there is the presence of a pink staining homogeneous material, besides evidence of Karyorrhexis; numerous neutrophiles observed in the red pulpa.
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Examination of smears.—Blood: \textit{T. mutans} not infrequent. Spleen: free and intra-corporeal agamonts rare. Prescapular lymph gland: a number of lymphocytes showed the presence of granules; intra-corporeal and free Koch's bodies extremely rare. Preauricular lymph gland: no Koch's bodies were identified. The periportal lymph gland: intra-corporeal and free Koch's bodies not infrequent. Mediastinal lymph gland: intra-corporeal and free Koch's bodies not infrequent.

N.B.—Note the difference in the occurrence of the number of Koch's bodies in the various organ smears.

C. 5000. Specimen 16128.

Pathological Anatomy.—Extensive swelling of the skin around the eyes and around the bases of the ears, extending into the intra-mandibular spaces; portions of the external ear have sloughed off, whereas the skin is matted with a dirty tenacious exudate; numerous fly larvae present; the subcutis in the pincinity of the head shows a large amount of clear fluid, whereas the blood vessels in the region of the ear and the masseter muscles markedly injected; the sub-parotid lymph gland markedly enlarged; on section it shows the presence of blood and purulent material; note further the extensive purulent necrotic inflammation of the external ears and the purulent haemorrhagic inflammation of the sub-parotid glands; slight anaemia; slight icterus; enlargement of the spleen and liver.

Etiology.—\textit{T. mutans} and sequel badly affected ears.

Histology.—Liver: well defined accumulations of lymphocytes in the periphery as well as in the distended intra-lobular capillaries where it causes atrophy of the liver cells and disorganization of their arrangement; liver cells appear swollen and show fatty changes; amongst the leucocytes in the capillaries there are also a good few neutrophiles present; liver cells around the central vein show the presence of bile pigmentation. Kidney: here and there between the tubules of the cortex small accumulations of cells of the lymphocytic series; some of the larger blood vessels between the medulla and the cortex show the presence of an accumulation of lymphocytes in the adventitia. Spleen: fairly well marked changes in connection with the malpighian bodies which show the presence of extensive oedema and slight necrotic changes in connection with some of the cells. Neutrophiles are frequent in the red pulp; the haemosiderosis in the red pulp is not prominent. Lymph glands: note the accumulation of neutrophiles, mainly in the cortex of the gland; the usual character of follicles and secondary follicles suppressed and here and there in their vicinity there is evidence of slight karyorrhexis, with evidence of oedema of the nature of a homogeneous pink staining material, irregularly distributed through the lymphoid tissue. Hippocampus: no specific changes.

Examination of smears.—Blood: slight anisocytosis; \textit{T. mutans} not rare; various stages of the development of Koch's bodies identified, from isolated single granules to those which show the usual Koch's bodies circumscribed in the cytoplasm of the cell together with the usual granules; free Koch's bodies also identified; it is estimated that about 30 per cent. of the lymphocytes are affected. Spleen: free agamonts as well as intra-corporeal bodies in all stages of development identified; free gamonts also seen, although much rarer. Bone marrow: similar to spleen smear. Liver: a large number of lymphocytic cells present in which Koch's bodies are frequent; free Koch's bodies also numerous. Hilie lymph gland: large numbers of free and intra-corporeal Koch's bodies identified. Parapharyngeal lymph gland: large numbers of free and intra-corporeal Koch's bodies identified. Mediastinal, prescapular, mesenteric, precrural, sub-parotid and parapharyngeal lymph glands: Koch's bodies also seen as well as in smears from the thymus.

N.B.—In the gland smears it should be noted that small lymphocytes were rare, whereas medium and large lymphocytes predominant, as well as unripe forms; Koch's bodies were also identified in lymphoblasts.
BOVINE THEILERIASIS IN S. AFRICA.

C. 5570. Specimen No. 16162.

Pathological Anatomy.—Ears have a lacerated appearance and parts have sloughed off; they are covered with a tarry substance as a result of the hand-dressing and on removal of this a purulent material is found over the surface of the ear; the right sub-parotid lymphatic gland markedly enlarged and on section half of the gland contains a dark red material while a sanguinous fluid escapes from cut surface; swelling and oedema of the other lymph glands; slight general anaemia; evidence of cachexia; enlargement of the liver, kidney and spleen; slight icterus; multiple necrotic foci, both lungs, with localised fibrinous pneumonia; slight epicarditis and a slight acute mucous catarrh of the nasal cavities; multiple fairly extensive ulcers abomasum.

Etymology.—Sequel T. mutans and badly affected ears as a result of extensive tick infestation.

Histology.—Liver: fatty changes around the central vein; no evidence of lymphocytic hyperplasia, but there is evidence of slight increase of connective tissue in the periphery; leucocytes frequent in the intralobular capillaries. Kidney: well defined localised lymphocytic hyperplasia cortex of the kidney, about $\frac{1}{4}$ cm. in diameter; comparable to an East Coast fever nodule. Lungs: hyperaemia and oedema with necrosis in one part of the section. Spleen: lymph follicles in places show the presence of rather extensive homogeneous eosin stained material, distributed like a network through the lymphoid tissue; there is a good deal of blood in the red pulpa in which neutrophiles are not infrequent. Lymph gland: loss of the usual formation and distribution of follicles; homogeneous, eosin stained material as described in the spleen also encountered, especially in places where there is still some indication of follicle formation. Hippocampus: no evidence of inclusion bodies, nor any changes to be observed in respect of the blood vessels.

C. 5629. Specimen No. 16170.

Pathological Anatomy.—Ears swollen and show fairly extensive necrosis extending towards the middle ear; abscess formation underneath the ears with necrosis extending on to the sub-parotid as well as the salivary glands; the skin around the eyes markedly swollen; the parapharyngeal lymph glands also show a purulent material which exudes on section; marked cachexia and emaciation; interstitial oedema lungs; abscess diaphragmatic lobe of lungs; marked tumour spleen; multiple greyish foci cortex of kidney; icterus of liver; multiple localised ulcers abomasum.

Etymology.—Killed in extremis. Sequel T. mutans and badly affected ears as a result of mass tick infestation.

Histology.—Liver: fatty changes over a small extent around the central vein; nuclei of liver cells stain well; number of neutrophiles present in the intralobular capillaries; no evidence of lymphoid hyperplasia. Kidney: no evidence of lymphoid hyperplasia. Spleen: large amount of blood in the red pulpa; the periphery of the malpighian bodies is very irregular in outline and a few show the presence of a pink staining homogeneous material in the form of a network through the lymphoid tissue; haemosiderosis occurs only to a slight extent; large number of neutrophiles, which in places are in form of dense clusters. Lung: extensive fibrosis with necrosis and serous catarrhal pneumonia.

Examination of smears.—Spleen: T. mutans rare; note large number of neutrophiles and many unripe lymphocytes; no Koch's bodies identified; pre-scapular lymph gland: free agamonts frequent; smear is somewhat disorganised. Pre-ocular lymph gland: a few free agamonts and intracorpuscular forms rare and somewhat indistinct. In the parapharyngeal lymph gland, liver and kidney smears no Koch's bodies seen.

N.B.—In this case note the irregular distribution of Koch's bodies in the organ smears.

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Pathological Anatomy.—The bases of both ears markedly swollen and the external ears have a lacerated appearance as a result of the sloughing off of portions; the surface is covered with a tarry substance and on removal of this there is a yellowish, purulent material present, which covers a granulating surface underneath; the sub-parotid lymph gland is the size of an orange and in the centre is a cavity which contains a purulent material; other lymph glands markedly swollen; marked emaciation and general anaemia; haemoglobinuria and haemoglobinaemia; slight general icterus; enlargement of the spleen, liver and kidneys; pneumatic foci both lungs; multiple ulcers abomasum, with oedema of the folds of the mucosa.

Etiology.—Piroplasmosis, complicated with sequel of extensive tick infestation.

Histology.—Liver: fairly extensive necrosis around the central vein; in places affecting almost two-thirds of the lobes; in the necrotic parts there is an increase in the number of neutrophiles; in view of this necrosis the columns of liver cells have become markedly disorganised around the central vein; many of the liver cells show fairly extensive fatty changes; no evidence of lymphoid hyperplasia. Kidney: evidence of pigmentation in the lumen of the tubuli and in the epithelial cells; here and there increase of lymphocytic cells around the blood vessels in the cortex; in one place, however, a large area of lymphocytic cells identified, with marked atrophy of the kidney structures. Lymph gland: loss of formation of follicles with the presence of oedema in patches; around some of these patches the nuclei show necrobiotic changes. Myocardium: very slight fatty changes in a few places. Central nervous system: various portions examined but no changes identified, especially of the small blood vessels. Spleen: the follicles here and there show the presence of this homogeneous pink staining material; large numbers of neutrophiles present in the red pulp; also evidence of necrobiotic changes here and there in the follicles of the spleen; P. bigem fairly frequent in the red cells of the spleen. Parotid salivary gland: a chronic supplicative inflammation was identified.

Examination of smears.—Spleen: unsatisfactory, but shows the presence of a large number of P. bigem, T. mutans rare. In the pre-scapular, precrural, bronchial, iliac, parapharyngeal, periporta1, and mediastinal lymph glands, large numbers of free and intra-corporcular agamons identified; the gamons are less frequently present; in these lymph glands the small lymphocytes are much rarer than medium, large and unripe forms. In the spleen, bone marrow, liver and kidney smears Koch's bodies were identified in very few numbers.

Note therefore in this case the peculiar distribution of the Koch's bodies in organ smears.

Pathological Anatomy.—Ears swollen at the bases; lacerated on account of sloughing off of parts; the surface is covered with a tarry substance and on removal of this a purulent material can be scraped off; the sub-parotid lymph glands on section show the presence of a sanguinous turbid fluid; the subcutis in the region of the ears show the presence of a turbid fluid mixed with a good deal of blood; other lymph glands swollen and oedematous; ascites and necrotic foci both lungs; enlargement of the spleen; multiple ulcers abomasum.

Etiology.—Killed in extremis. Sequel T. mutans and badly affected ears as a result of tick infestation.

Histology.—Liver: a number of lymphocytic cells in the intra-lobular capillaries besides a few neutrophiles; slight fatty changes in some of the liver cells, but these on the whole stain fairly well; lymphoid hyperplasia identified in the periphery. Kidney: multiple lymphoid hyperplastic foci in the cortex of the kidney, together with one large focus resembling those observed in East Coast fever. Lungs: serous catarrhal pneumonia with localised necrosis. Hippocampus: no changes observed, especially in respect
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of haemorrhages or infiltrations. Spleen: some of the follicles disorganised due to the presence of a pink staining, homogeneous material; a good deal of blood here and there in the pulp, together with a fair number of neutrophiles; haemosiderosis suppressed. Lymph glands: loss of follicles evident; here and there traces of a slight pink staining homogeneous material, as well as necrotic changes of some of the cellular elements; in places neutrophiles are prominent.

Examination of smears.—Blood: lymphocytes frequent and in a number of these agamonts in all stages can be identified, from a few granules to such bodies well circumscribed in the cytoplasm of the cell; free agamonts not infrequent; slight anisocytosis; slight polychromasia; T. mutans rare. Liver: vacuoles identified in the liver cells; these are circular and well circumscribed; intra-corpuscular and especially free agamonts identified but not frequent. Spleen: Koch’s bodies all stages identified. Kidney: Koch’s bodies all stages identified. Periportal lymph gland: agamonts in all stages frequent; gammonots also identified; the majority of lymphocytic cells are mainly of the middle and large type; small lymphocytes extremely rare. Mediastinal lymph glands: similar to previous one. Bone marrow: Koch’s bodies not infrequent.

C. 5663. Specimen No. 16171.

Pathological Anatomy.—Bases of the ears slightly swollen; sub-parotid lymph glands markedly enlarged and in the substance of the left sub-parotid is a circumscribed nodule, caseous in nature but not tuberculous in character; in the right sub-parotid lymph gland abscess formation with necrosis; the other lymph glands are swollen and oedematous; few abscesses in the lungs; slight tumor spleus; enlargement of the liver with icterus; slight acute catarial enteritis; hydropericardium.

Etiology.—T. mutans and the sepsul of badly affected ears resulting from tick infestation.

Histology.—Liver: columns of liver cells, especially around the central veins, slightly disorganised; some of the liver cells show slight hyaline droplet degeneration; bile stasis identified in the vicinity of the periphery of the lobule; no evidence of lymphoid hyperplasia. Myocardium: no specific changes. Lymph glands: a reduction in the number of lymphoid follicles. Spleen: the changes in the lymphoid follicles are fairly extensive as a result of the presence of homogeneous pink staining material in the lymphoid tissue.

Examination of smears.—Spleen: no Koch’s bodies identified even after prolonged search; T. mutans present. Liver: no Koch’s bodies identified even after prolonged search; T. mutans present. Precrural lymph gland: not well stained but no Koch’s bodies identified. Sub-parotid lymph gland: not well stained but no Koch’s bodies identified. Periportal lymph gland: intra-corpuscular gammonots not frequent; free agamonts more frequent; the majority of these bodies are small with only a few granules present. Mediastinal lymph gland: one free agamont identified after prolonged search.

Comment.—Unfortunately the first batch of smears were understained and the re-stained smears were not satisfactory but in spite of this the distribution, especially in respect of the frequency of Koch’s bodies, are very significant.

C. 5665. Specimen No. 16132.

Pathological Anatomy.—Ears swollen at the bases and the greater portions of the external ears have sloughed off; the subcutis in the vicinity of the ears show the presence of a purulent sanguinous fluid; the right sub-parotid lymph gland markedly swollen and the skin shows the presence of a sauginous necrotic material; the left sub-parotid is markedly swollen and on section shows the presence of a dark greyish fetid material; other lymph glands are swollen and oedematous; slight swelling of the spleen and liver; multiple greyish foci in the kidneys; impaction omasum; haemonchosis abomasum; atelectasis, emphysema, with multiple haemorrhages both lungs; multiple ulcers abomasum.

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Etiology.—Killed in extremis. Sequel T. mutans and badly affected ears as a result of tick infestation.

Histology.—Liver: no lymphoid hyperplasia; slight fatty changes around the central vein. Kidney: localised necrosis of the tubuli in the cortex, with infiltration of cells amongst which a number of neutrophiles can be identified; neutrophiles also present in the intra-lobular capillaries. Note: the greyish foci are the result of embolism associated with the badly affected ears. Skin: the deeper layers show an extensive phlegnosis with necrosis. Spleen: slight evidence of the presence of a homogeneous, pink staining material in a few of the follicles. Lungs: the presence of haemorrhages besides a slight hyperaemia can be identified. Abomasum: a circumscribed necrosis extending up to the muscularis mucosae with desquamation of the necrotic material. Lymph glands: reduction in the number of well defined follicles in some the presence of a pink staining, homogeneous material can be identified.

Comment.—This is undoubtedly a sequel to tick infestation in which lymphoid hyperplasia had not yet established itself in the liver and kidneys, although early lesions were already present in the spleen and lymph glands.

Examination of smears.—Spleen: free agamonts not rare; gamonts definitely identified although very rare; no T. mutans observed; slight anisocytosis; bronchial lymph gland: Koch's bodies rare; bone marrow: Koch's bodies very rare. Preepipulmonary, periportal and mediastinal lymph glands: Koch's bodies not infrequent.

C. 5676. Specimen No. 36120.

Pathological Anatomy.—Ears markedly swollen, especially at the bases, the swelling being about 6 inches in diameter; oedematus in character; the intra-mandibular spaces: go. The external ears are covered with a crust and on removal of this a suppurating surface can be identified; in the deeper layers of the ear purulent and necrotic material identified. Sub-parotid lymph gland markedly swollen and in the substance haemorrhages identified. The rest of the lymphatic glands slightly swollen and oedematous. Slight swelling of the liver and kidneys.

Etiology.—Killed for the collection of specimens.

Histology.—Liver: intra-lobular and inter-lobular blood vessels show the presence of a number of polymorphs; the majority of these are definitely of the lymphocytic series; slight fatty changes of the liver cells around the central veins; in the periphery there is a slight increase of lymphocytic cells associated with the blood vessels. Kidney: in a few places in the cortex there is an increase of lymphocytic cells, but these seem to be associated with an increase of the connective tissue elements as well, more of the nature therefore of an interstitial nephritis than a lymphocytic hyperplasia. Spleen: some of the follicles show the presence of a pink staining material and associated with it some of the cells show necrotic changes. Lymph glands: a definite loss of follicles. Myocardium and hippocampus: no changes identified.

Examination of smears.—Spleen: free agamonts in all stages frequent; a few free gamonts identified. Mediastinal lymph gland: free agamonts in all stages; a few free gamonts identified. Periportal lymph gland: Koch's bodies less frequent; note the large number of unripe lymphocytic cells. Iliac sub-parotid, and precrural lymph glands are more or less similar to the mediastinal lymph gland, whereas the mesenteric is again similar to the periportal in respect of the frequency of Koch's bodies.

Comment.—In the iliac lymph gland Koch's bodies were identified with only two granules present (these were not rare; compare similar bodies described by Cowdrey.
Pathological Anatomy.—Left ear: base swollen; less in the right ear; the surface is covered with a tarry material and the skin shows the presence of a purulent material. The left parapharyngeal lymph gland markedly enlarged and on section a purulent sanguineous fluid escapes; in the cortex there is a dark red necrotic area about 2 cm. in diameter. Rest of the lymph glands swollen and oedematous; slight anaemia; slight cachexia; few alveolar areas in the lungs; slight swelling of the spleen and the liver; slight haemosiderosis; multiple ulcers abomasum.

Etiology.—Killed in extremis. Sequel T. mutans and badly affected ears.

Histology.—Liver: capillaries and veins show a fair number of neutrophiles; in a few places in the periphery small accumulations of cells of the lymphocytic series around the blood vessels. Kidney: accumulations of cells of the lymphocytic series in the adventitia of a few of the vessels in the medulla; on such foci seen in the cortex. Hippocampus: no changes identified, in connection with the blood vessels. Spleen: majority of the follicles show the presence of a pink staining homogeneous material in between the lymphoid tissue. Lymph glands: lymph follicles affected; much fewer in number and those present show reduced peripheral accumulations of lymphocytes; in some these are somewhat irregular in outline; some of the remaining follicles show the presence of a pink staining, homogeneous material; in the vessels running transversely to the capsule large numbers of neutrophiles can be identified. Myocardium: no specific changes. Lung: atelectasis and in places bronchiolitis show the presence of acute bronchiolitis.

Examination of smears.—Spleen: no Koch's bodies. Liver: slight anisocytosis; parapharyngeal lymph gland: a number of lymphocytes with the presence of granules; one free agamont identified. Sub-parotid lymph gland: a number of lymphocytes with the presence of granules; one free agamont identified. Mediastinal lymph gland: a number of lymphocytes with the presence of granules; one free agamont identified. Marrow: no Koch's bodies identified. Kidney: no Koch's bodies. Periportal lymph gland: Koch's bodies rare.

C. 5742. Specimen No. 16122.

Pathological Anatomy.—Ears much swollen, especially at the bases; the swelling is oedematous in character; the surface of the ears show the presence of an exudate which is encrusted on a soft suppurating surface; portions of the ear have sloughed off; the parapharyngeal lymph glands markedly enlarged and in the substance there are several large haemorrhages; sub-parotid lymph gland enlarged and on section show the presence of a fetid sanguineous fluid; the substance of the gland is partly necrotic.

Etiology.—Killed in extremis. Sequel T. mutans and badly affected ears.

Histology.—Liver: a number of lymphocytes in the intra-lobular capillaries. Proliferations of cells of the lymphocytic series in the periphery of the lobule; slight fatty changes in some of the liver cells.

Comment.—A case in which there is slight lymphoid hyperplasia in the liver. Kidneys: in a few places around blood vessels in the cortex there are accumulations of cells of the lymphocytic series. Lymph glands: loss of follicles; in places neutrophiles present, sometimes in clusters. Spleen: lymphoid follicles not prominent; large numbers of neutrophiles in the red pulpa.

Examination of smears.—Spleen: numerous free agamonts; no T. mutans seen. Prescapular and mediastinal lymph glands: free agamonts in all stages identified; intra-corpuscular forms also not infrequent.

C. 5763. Specimen No. 16147.

Pathological Anatomy.—Ears markedy swollen and the external ears on the inside show the presence of a fetid, necrotic material; the subcutis in the region of the ear and face is oedematous and reddish; sub-parotid lymph...
glands markedly swollen and on section show the presence of a gangrenous necrotic substance; rest of the lymph glands are swollen and oedematous; no changes in the lungs; swelling of the liver.

Etiology.—Killed in extremis. Sequel T. mutans and badly affected ears.

Histology.—Liver: fairly well marked lymphocytic accumulations in the periphery of the lobule; fatty changes of the liver cells around the central vein. Kidneys: round the blood vessels in the medulla slight accumulation of cells of the lymphocytic series; although these may be regarded as of the nature of a lymphoid hyperplasia it is only present to a slight extent. Myocardium: no specific changes. Lymph glands: blood vessels running transversely to the capsule of the spleen show numerous neutrophilic; loss of the formation of follicles, especially along the marginal zone; note the presence of a homogeneous, pink staining material in places of the follicles, associated with necrobiotic changes of some of the cells. Spleen: fairly extensive changes in the lymphoid follicles of the nature of accumulations of a homogeneous, pink staining material in between the follicles in places reaching fairly large dimensions; there is a good deal of blood in the pulpa. Hippocampus: no changes but note in the red cells of the capillaries numerous P. bigem can be identified. Lung: atelectasis in a few places associated with an acute bronchitis.

Examination of smears.—Agamonts free and intra-corporcular identified in the spleen periportal, mediastinal, iliac, prescapular, mesenteric and parapophraryngeal lymph glands; in the liver smears rare; in the kidney smears none identified; in the spleen slight anisocytosis, slight polychromasia; T. mutans rare; P. bigem. rare; in the majority of lymph gland smears medium and large lymphocytes predominate, in fact, small lymphocytes rare; note also the peculiar distribution of P. bigem.

C. 6287. Specimen No. 16121.

Pathological Anatomy.—Slight necrosis of the tips and the internal surface of the ear; slight haemorrhous; slight swelling and eedema of some of the lymph glands.

Etiology.—Killed, clinically healthy, for the collection of specimens.

Histology.—Liver: slight accumulation of lymphocytes here and there in the periphery of the lobule; lymphocytes in the intra-lobular capillaries also prominent. Kidney: no evidence of lymphoid hyperplasia. Lymph glands: some show numerous neutrophilic present in the blood vessels along the marginal zone. Lung: no specific changes.

Examination of smears.—Koch's bodies frequent in the spleen, kidney, bone marrow, and the majority of lymph glands; in the liver smears rare; no T. mutans identified.

Comment.—Only very slight lymphoid hyperplasia in the liver seen, although Koch's bodies are frequent in all the organs examined. In spite of the frequency of Schizonts gametocytes not identified.

C. Tzane Experiments, 1936.

C. 5653. Specimen No. 17731.

Pathological Anatomy.—No changes in connection with the ears; anemia; haemoglobinuria; slight icterus; subendocardial and subendocardial haemorrhage; degenerative changes liver and kidneys.

Etiology.—Piroplasmosis.

Histology.—Liver: fairly extensive necrosis around the central veins; no lymphoid hyperplasia. Kidney: no lymphoid hyperplasia; extensive haemosiderosis, especially in the cortex.

Examination of smears.—Spleen and blood: P. bigem. frequent; no T. mutans seen. Mediastinal, parapophraryngeal, prescapular, periportal lymphatic glands, kidney and liver; no Koch's bodies identified.
BOVINE THEILERIASIS IN S. AFRICA.

C. 5442. Specimen No. 17689.

Pathological Anatomy.—Slight general anaemia; cachexia; very extensive oedema of the lungs; marked hydrothorax; extravasations left endocard; slight acute mucous catarhal enteritis; hyperaemia and a few petechiae abomasum; hyperaemia, swelling and slight degeneration liver; slight swelling of the spleen; oedema of the majority of lymphatic glands.

Etiology.—Heartwater.

Histology.—Liver: slight leucostasis in the periphery of the lobule; very slight evidence of cirrhosis. Kidney: no evidence of hyperplasia.

Examination of smears.—Intima smear: Rickettsia identified. Blood: clusters of granules of all classes and numbers not infrequently seen in the lymphocytes. Spleen, sub-parotid, mediastinal, parapharyngeal, periportal and precrural lymph glands, as well as liver and kidney show no Koch's bodies.

C. 5561. Specimen No. 17736.

Pathological Anatomy.—Slight anaemia; slight emaciation; marked oedema of the lungs; localised bronchial pneumonia left lung; slight acute mucous enteritis; lymphoid tissue prominent in the majority of lymphatic glands and spleen; slight swelling of the spleen; slight pigmentation of the liver; extravasations epicard and both endocard.

Etiology.—Sequel piroplasmosis.

Histology.—Liver: an irregular cirrhosis; fairly extensive haemosiderosis of the R.E. cells; a large amount of blood in the intra-lobular capillaries are prominent. Kidney: haemosiderosis; no evidence of lymphoid hyperplasia. Lung: acute broncho pneumoniia. Spleen: lymphoid tissue prominent; extensive haemosiderosis. Lymph glands: lymphoid tissue haemosiderosis, in places associated with the R.E. cells.


Comment.—A case in which one Koch's body was seen in spite of the fact that several lymph glands were carefully examined. Furthermore, the number of gametocytes present is of interest in relation to the almost complete absence of Koch's bodies.

APPENDIX III.

UMZINTO (NATAL) EXPOSURE EXPERIMENT, 1934.

A brief summary of the most important symptoms, pathological changes, etc., observed in the cattle exposed at Umzinto:

I. C. No. 6; branded 77 off hip; inner paddock; red tillie about 24 years; died on 31.3.34; badly tick infested and both preseapular glands distinctly swollen on 28.3.34. On 29.3.34 the swelling on the head had extended down the neck to the dewlap. On 30.3.34 the animal was lame in both fore and walked with difficulty. On 31.3.34 it was found dead.


Etiology.—Probably septicemia as result of gross tick and maggot infestation.

Histology.—No lymphoid hyperplasia in liver and kidneys.
G. DE KOCK, C. J. VAN HEERDEN, R. DU TOIT, AND W. O. NEITZ.

Smear Examination.—(a) During life: negative. (b) After death: negative.

II. C. No. 1, branded No. 579; inner paddock; black tollie about 18 months old; died on 9.4.34. It showed symptoms similar to the first animal. This animal was poor in condition when it arrived from Allerton. Although the treatment of the ears alleviated the position very considerably, the animal became more and more unthrifty. It exhibited no pronounced symptoms beyond general debility and listlessness.


Etiology.—Septicemia from tick and maggot infestation and malnutrition. This animal had hardly any teeth although only eighteen months of age.

Histology.—No lymphoid hyperplasia in liver and kidneys. Spleen: lymphoid tissue prominent; no Koch's bodies identified in the spleen, blood, prescapular and periportal lymph glands.

III. C. No. 8, branded 77 and tag No. 565; outer paddock; red and white tollie about 21 years old; died on 13.4.34. It showed no special symptoms during life except general unthriftiness, loss of condition, dullness, both suprascapular glands swollen. No maggots were present in its ears, which showed only slight inflammation from tick infestation.

Post Mortem. Specimen No. 14941. Carcase emaciated and anaemic; prescapular glands intensely swollen and haemorrhagic; mandibular, parapharyngeal and precrural lymph glands slightly swollen and haemorrhagic; mucous membrane of abomasum pale and swollen; liver slightly enlarged and friable; bile thick and dark; kidneys slightly swollen and hyperaemic; tumor spleen.

Etiology.—Sequel piroplasmosis.

Histology.—Liver: no definite lymphoid hyperplasia; good few neutrophiles in the intra-lobular capillaries. Kidney: no lymphoid hyperplasia. Spleen: extensive haemosiderosis in the red pulp; haemorrhagic areas. Examimation of smears.—Blood, kidney, spleen, liver, mediastinal, precrural, periportal, mandibular lymph glands: no Koch's bodies identified but in the precrural P. bigem identified. These smears are somewhat disorganised.

This animal was immunised against redwater.

IV. C. No. 11, branded 67, tag No. 557; outer paddock; black and white tollie about 15 months; died on 13.4.34 and showed symptoms and post mortem changes similar to animal No. 3, except that in addition red urine in the bladder was present.

Etiology.—Piroplasmosis.

Histology. Specimen No. 54940.—Liver: no lymphoid hyperplasia; slight cirrhosis; lymphocytes prominent in the intra-lobular capillaries. Kidney: no lymphoid hyperplasia. Spleen, lungs and glands: no specific changes.

Examination of smears.—Blood: T. mutans fairly frequent; slight anisocytosis. Spleen, bronchial, mandibular, prescapular, mediastinal lymph glands: no Koch's bodies identified.

V. C. No. 4, branded 588; inner paddock; black and white tollie about 15 months old; died on 16.4.34. It showed no outstanding symptoms except general unthriftiness, dullness, weakness, loss of appetite and condition and just prior to death slight anaemia. The prescapular glands on both sides were swollen. No maggots in the ears.
BOVINE THEILERIASIS IN S. AFRICA.

Post Mortem. Specimen No. 14939. Carcase emaciated, prescapular glands intensely swollen and haemorrhagic (due to making of smears). Mandibular, parapharyngeal and precrural lymph glands slightly swollen and haemorrhagic. Mucous membrane of abomasum pale and swollen—a few wire worms present; liver slightly enlarged and friable; bile thick and reddish in colour; kidneys slightly swollen and hyperaemic; urinary bladder filled with red urine; tumor splenius; slight hydrothorax; ascites and oedema of the lungs.

Etiology.—Piroplasmosis.


Examination of smears.—Bronchial, mandibular, precrural, mediastinal, periportal, retro-pharyngeal, prescapular lymph glands, spleen, liver and kidney: no Koch's bodies identified, although the smears were somewhat disorganised.

VI. C. No. 2, branded 592; inner paddock; black toliie about 18 months old. It showed symptoms and post mortem changes similar to Animal No. 5. It died on 16.4.34.

Etiology.—Piroplasmosis.

Histology.—No lymphoid hyperplasia in kidney and liver.

Specimen No. 14937.

Examination of smears.—Blood: P. bigem., not infrequent; T. mutans rare. Spleen, parapharyngeal and prescapular lymph glands: no Koch's bodies identified.

VII. C. No. 3, branded 583; inner paddock; a black and white toliie about 15 months old. It showed post mortem changes (Specimen No. 14938) similar to those exhibited by animals 5 and 6, except that a gland smear taken on 6.4.34 showed Koch's bodies frequent but no small piroplasms. The gland smears taken on 3.4.34 and 9.4.34 showed Koch's bodies very rare. Attempts were made to increase the resistance of this animal by giving it bran and mealie meal night and morning but it unfortunately died of redwater on 16.4.34.

Blood from this animal was taken for subinoculation into other animals at Allerton. Blood smears taken at the time the blood was tapped revealed no sign of P. bigemum, yet in one of the subinoculated cattle at Allerton (which was not immunised against redwater), redwater was produced. The reaction was controlled with trypan blue and the animal recovered.

After death of C. 3 spleen and gland specimens were sent to Allerton and the pulp thereof used to submoculate cattle at Allerton. No reaction has, however, been observed in these inoculated cattle.

Pathological Anatomy.—Ears slightly swollen and inflamed; cachexia; slight icterus and anaemia; slight swelling of the lymph glands; slight swelling of the liver and kidneys; haemoglobinuria; tumor splenius; slight hydrothorax; ascites and oedema of the lungs.

Etiology.—Piroplasmosis.

Histology.—Liver: no lymphoid hyperplasia. Kidneys: no lymphoid hyperplasia. Lung: acute broncho-pneumonia. Spleen: large amount of blood in the red pulp, amongst which there are a large number of neutrophiles. Lympgh glands: loss of the regular distribution of the follicles; some of the lymph glands contain a large amount of blood.

Examination of smears.—Spleen: P. bigem., not infrequent; T. mutans rare; no Koch's bodies identified.

VIII. C. No. 9, branded 88; outer paddock; red toliie about 2½ years old. It showed the same general symptoms as the others, except that on 17.4.34 it showed red urine. It was at once injected with trypan blue.
Post Mortem. Specimen No. 14956. Whole carcase and organs discoloured with trypan blue; prescapular lymph glands swollen and haemorrhagic; liver slightly enlarged and friable; bile thick and red in colour; kidneys slightly swollen and hyperaemic; urinary bladder filled with chocolate coloured urine; tumour spleen; slight hydrothorax, ascites and oedema of the lungs.

Etiology.—Piroplasmosis.

Histology.—Liver, spleen, lungs and kidneys decomposed, but no evidence of lymphoid hyperplasia identified in liver and kidneys.

Examination of smears.—No Koch's bodies identified in the blood, spleen, liver, perportal, bronchial, mediastinal and precrural lymph glands.

IX. C. No. 5 and branded 66; inner paddock; red and white tollie about 2½ years old; died on 23.4.34. This animal showed much the same symptoms as the others, only it appeared particularly emaciated and dejected just before death.

Post Mortem. Specimen No. 14982. Whole carcase discoloured by trypan blue. Prescapular lymph glands very swollen and haemorrhagic. Mandibular and precrural glands slightly swollen and haemorrhagic. Liver slightly enlarged and friable; bile thick and dark in colour; kidneys slightly swollen and hyperaemic; spleen slightly swollen; slight hydrothorax, ascites and oedema of the lungs; anaemia and emaciation.

Etiology.—Sequel tick infestation.

Histology.—Liver: slight cirrhosis; no lymphoid hyperplasia. Kidney: no lymphoid hyperplasia.

Examination of smears.—Blood: T. mutans not rare; slight anisocytosis; spleen, precrural, bronchial, periportal lymph glands: no Koch's bodies seen.

X. C. No. 7, branded 66; outer paddock; white tollie about 2½ years old; died on 23.4.34. It showed symptoms and post mortem changes (Specimen No. 14982) similar to C. 9 and in addition a very large and inflamed gall bladder.

Etiology.—Anaplasmosis.

Pathological Anatomy.—Both ears slightly swollen and inflamed; liver slightly swollen; kidney slightly swollen; slight hydrothorax, ascites and oedema of the lungs; slight emaciation and general anaemia.

Histology.—Liver: central necrosis; cirrhosis; no lymphoid hyperplasia. Kidney: no lymphoid hyperplasia. Blood, spleen, mandibular, mediastinal and prescapular lymph glands: no Koch's bodies or parasites identified.

XI. C. 12, branded 68 and tagged 59; outer paddock; black and white tollie. It showed symptoms and post mortem changes (Specimen No. 14997) similar to C. 9 and C. 10. It showed redwater (clinical) on 17.4.34 and was injected with trypan blue. It apparently recovered from this disease, but developed anaplasmosis, became weaker and weaker, showed diarrhoea. It continued to eat sparingly until it died.

Pathological Anatomy.—Slightly decomposed; ears slightly swollen; anaemia; emaciation; trypan blue discoloration; cachexia.

Etiology.—Anaplasmosis. [T. mutans identified.]

XII. C. N. 10, branded 99; outer paddock; red tollie about 2½ years; slaughtered 29.5.34. In kidneys white foci identified besides trypan blue discoloration; anaemia; slight emaciation; slight hydrothorax and ascites; haemonchosis (Specimen 15105). The white foci in the kidneys of the nature of a lymphocytic nephritis.
### APPENDIX IV—TICK TRANSMISSION EXPERIMENTS.

**Table 1. Feeding of Tsetse Tick on Cattle at Onderstepoort during 1935 and 1936.**

<table>
<thead>
<tr>
<th>No. of Expt.</th>
<th>No. of Animal</th>
<th>History</th>
<th>Tick Feeding: <em>R. appendiculatus</em> (adult)</th>
<th>Result</th>
<th>Sub-inoculation</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
<td>Batch No.</td>
<td>Origin</td>
<td>Infested.</td>
</tr>
<tr>
<td>S. 5612</td>
<td>C. 4676</td>
<td>Splenectomized. 9-8-32. Carrier of <em>T. mutans</em>, <em>P. biocusae</em>, <em>A. marginale</em></td>
<td>1356Xa1</td>
<td>Transvaal</td>
<td>11.3.35</td>
</tr>
<tr>
<td>S. 5654</td>
<td>C. 6627</td>
<td>Carrier of <em>T. mutans</em>. At time of feeding Transvaal ticks, bovine, was infected with <em>R. decolorum</em>, <em>A. holomos</em>, <em>E. equiendalvis</em> which had been picked up at Onderstepoort.</td>
<td>1356Xa2</td>
<td>Transvaal</td>
<td>25.3.35</td>
</tr>
<tr>
<td>S. 5661</td>
<td>C. 5023</td>
<td>From Vryburg</td>
<td>1372Xa3</td>
<td>Transvaal</td>
<td>24.4.35</td>
</tr>
<tr>
<td>S. 5888</td>
<td>C. 6403</td>
<td><em>T. mutans</em> susceptible</td>
<td>1382Xa1</td>
<td>Transvaal</td>
<td>4.2.36</td>
</tr>
<tr>
<td>S. 5888</td>
<td>C. 6291</td>
<td><em>T. mutans</em> carrier</td>
<td>1382Xa2</td>
<td>Transvaal</td>
<td>4.3.36</td>
</tr>
<tr>
<td>S. 5888</td>
<td>C. 4019</td>
<td><em>T. mutans</em> carrier and had been exposed to <em>T. annulata</em></td>
<td>1382Xa1</td>
<td>Transvaal</td>
<td>4.3.36</td>
</tr>
</tbody>
</table>