TABLE 34.

Percentage involvement of various organs in 338 cases of Acute Tuberculosis.

Glands—
- Cervical .......... 16
- Clavicular .......... 21
- Tracheo-bronchial .......... 88
- Diaphragmatic .......... 31
- Pancreatic .......... 48
- Lower Retro-peritoneal .......... 30
- Portal .......... 46
- Mesenteric .......... 34
- Axillary .......... 1
- Inguinal .......... 0.3

Lungs—
- Caseation .......... 25
- Caseation with Excavation .......... 81
- Milia only .......... 26

Pleura—
- Definite Tuberculous Pleurisy .......... 32

Heart and Pericardium—
- Definite Tuberculous Pericarditis or Myocarditis .......... 17
- Adhesions, possibly Tuberculous .......... 4
- Spleen .......... 70
- Liver .......... 61
- Kidneys .......... 31
- Suprarenals .......... 10
- Peritoneum .......... 31
- Intestine .......... 21
- Brain or Meninges .......... 2.5
- Bone or Joint .......... 4
- Genitalia .......... 1.5

For comparison we have made in tabular form (Table 35) an analysis of the records of these 200 London cases and of 200 Witwatersrand mine Native labourers, taking 100 W.N.L.A. cases and 100 mine medical officers' cases. All cases with silicosis were excluded, but otherwise there was no selection, the cases being taken consecutively as they occurred. The comparison is therefore between uncomplicated tuberculosis in the two series of cases.

Comment may be made on the following points brought out in Table 35:—

Glands.—Cervical glands are rarely involved in the Native, as compared with the European, but there is a strikingly more common involvement in the Natives of the "other thoracic" (retrosternal and diaphragmatic) glands, also of the "other abdominal" (portal and various groups of retro-peritoneal) glands.

Lungs and Pleura.—Lung lesions are more extensive as well as more constantly present in the European as compared with the Native. Scarring, fibrosis, pleural adhesions, caseation and cavitation are all obviously more striking features in the European. Definite caseous deposits in the pleural sacs, however, are more common in the Native.
<table>
<thead>
<tr>
<th>Degree of Involvement</th>
<th>Glands</th>
<th></th>
<th>Lungs</th>
<th></th>
<th>Pleur.e</th>
<th></th>
<th>Heart and Pericardium</th>
<th></th>
<th>Glands</th>
<th></th>
<th>Abdominal Organs</th>
</tr>
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<tbody>
<tr>
<td></td>
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</tr>
<tr>
<td></td>
<td>Cervical</td>
<td>Tracheo-Bronchial</td>
<td>Other Thoracic</td>
<td>Affected</td>
<td>Caseous</td>
<td>Cavities</td>
<td>Sacs</td>
<td>Fibroid</td>
<td>Milky</td>
<td>Effusion</td>
<td>Adhesions</td>
</tr>
<tr>
<td>London Patients.</td>
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<tr>
<td>+++</td>
<td>11</td>
<td>46</td>
<td>—</td>
<td>168</td>
<td>60</td>
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<td>42</td>
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<tr>
<td>++</td>
<td>19</td>
<td>66</td>
<td>—</td>
<td>20</td>
<td>44</td>
<td>56</td>
<td>10</td>
<td>17</td>
<td>15</td>
<td>16</td>
<td>90</td>
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<tr>
<td>+</td>
<td>49</td>
<td>56</td>
<td>1</td>
<td>9</td>
<td>43</td>
<td>30</td>
<td>20</td>
<td>27</td>
<td>7</td>
<td>14</td>
<td>54</td>
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<td>23</td>
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<td>1</td>
<td>—</td>
<td>2</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>2</td>
<td>3</td>
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<tr>
<td>Total 200</td>
<td>106</td>
<td>191</td>
<td>1</td>
<td>198</td>
<td>147</td>
<td>157</td>
<td>32</td>
<td>54</td>
<td>45</td>
<td>38</td>
<td>189</td>
</tr>
<tr>
<td>Witwatersrand Native Mine Workers.</td>
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<td>+++</td>
<td>1</td>
<td>20</td>
<td>1</td>
<td>24</td>
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<td>13</td>
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<tr>
<td>+</td>
<td>19</td>
<td>91</td>
<td>81</td>
<td>74</td>
<td>47</td>
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<td>10</td>
<td>2</td>
<td>48</td>
<td>18</td>
<td>51</td>
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<td>—</td>
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<tr>
<td>Total 200</td>
<td>27</td>
<td>192</td>
<td>94</td>
<td>175</td>
<td>105</td>
<td>63</td>
<td>11</td>
<td>3</td>
<td>79</td>
<td>29</td>
<td>67</td>
</tr>
</tbody>
</table>
**Heart and Pericardium.**—Although the two figures given for the two series are almost identical, there is actually a very marked difference in character. Of the 35 European cases, only 1 was a case of actual tuberculous pericarditis, the other 34 being merely cases of some excess of fluid or of slight adhesions. In the Native series, 31 were definitely cases of tuberculous pericarditis or myocarditis, and 6 cases with adhesions of doubtful origin.

**Abdominal Organs.**—These show perhaps the most striking difference between the incidence in the two series. Involvement of the spleen and liver is very much more common in the Native, and in the spleen especially the degree of involvement is much greater. Tuberculous peritonitis and involvement of "other abdominal organs" such as suprarenals and genitalia is also more common in the Native.

Certain other items not brought out in the Table might also be referred to:

*Laryngeal Involvement* is mentioned in 43 of the 200 Europeans. It was only observed in 2 Native cases, but it was not systematically examined for in all of them, so that it might possibly have been actually more common, although it is improbable with the short life of so many of the Natives that the figure would have been as high as for the Europeans.

*Tuberculous Meningitis or Cerebral Tuberculomata* were present in 16 European cases, only in 2 Native cases. Here it must be noted, however, that head examinations were only made in those Natives who had shown definite cerebral symptoms during life.

*Amyloid Disease* was met with in 13 of the European cases; it was not present in any of the Native cases.

**Comparison with Tuberculosis amongst Cape Coloured (Eurafrican) People.**—Through the courtesy of Professor Ryrie, Department of Pathology, University of Cape Town, we have received post-mortem records made at the City Hospital, Cape Town, of 25 cases of tuberculosis in Coloured persons (Eurafricans). The series is small, but it is of interest as giving some idea of the character of tuberculosis in a section of the South African population in which the disease is rather rife.

An analysis of these records on the same lines as that shown in Table 35 shows that the type *en masse* is much nearer that of the Witwatersrand Native cases than that of the London European cases. The figures for scarring and fibrosis of the lungs are, however, of the London type rather than of the Witwatersrand type.

This discrepancy is readily explicable by a consideration of the age grouping of the Cape Town series. Of the 25 cases, 15 were under the age of 15, and only 10 over that age. All the cases with "chronic" lesions in the lungs occurred in the group over 15 years of age.
Amongst the Cape Coloured adults, therefore, the suggestion is that their tuberculosis is more of the European adult type than of the African Native type. The numbers are insufficient to warrant any more definite conclusion.

*Tuberculosis approximating to the "Natural" Type.*—Consideration of the above comparison with European cases and of the figures in Table 34 brings out the fact that the "acute" tuberculosis of the Witwatersrand Native mine labourers approximates to the "primary," "unmodified" or "natural" tuberculosis which, amongst Europeans, is seen almost only in young children. It is not, however, entirely the natural tuberculosis of completely virgin soil, but is modified to a certain extent.

This is brought out by comparison of our tuberculosis with that of other localities or populations where natural tuberculosis prevails. For this purpose, we have made comparisons with the general record of Bushnell\(^1\) and the particular one of Borrel\(^2\).

Numerous illustrations have been met with of the forms cited by Bushnell as characteristic of "primary" tuberculosis. He states that the most characteristic feature is general infiltration of the lymph glands, not in a chronic form as in the imperfectly immunized, but manifesting itself often as great packets of enormously enlarged caseating and suppurating glands. Our figures show the frequency of involvement of many groups of glands and very often indeed they were in "great packets" (see Pl. I, Fig. 3). Caseation was very common, but actual suppuration, although not exactly rare, could not be said to be very frequent. More common than suppuration was some hardening of the glands, microscopic examination showing a mixture of caseation, some fibrosis and considerable proliferation of endothelial cells—an approach to the "large-celled hyperplasia" of older writers. Associated with this there was occasionally a remarkable degree of hyaline degeneration of the fibrous tissue in the glands, but apparently not the condition described by Aschoff\(^6\) as characteristic of healing primary infections. This degree of fibrosis and endothelial proliferation probably points to the tuberculosis occurring, not in a perfectly natural condition, but in a subject slightly although very imperfectly immunized. A terminal miliary spread was commonly seen when there was extensive caseation in one or more of the gland groups.

Tuberculous lobar pneumonia, or gelatinous pneumonia, is another feature mentioned by Bushnell as characteristic of natural tuberculosis. It was rather rare in our series, only two really characteristic cases being seen, although we might have added another six or seven cases with a confluent broncho-pneumonia involving the whole or nearly the whole of one lobe (see Pl. I, Fig. 4). We have the impression from previous experience that the massive tuberculous pneumonias are usually proportionately more numerous, and that it just happened by chance that we have not encountered more of them in this series, but we have not definite figures to prove this.
"Another characteristic," Bushnell says, "is the primary involvement of serous membranes—the disease is a tuberculous serositis."

The figures given for the pleural, pericardial and peritoneal involvements illustrate the occurrence of this type frequently in our series. Further, "very characteristic of primary tuberculosis are caseous tubercle of the myocardium and the pericardium." He quotes Zone Clark's figures from a series of 452 autopsies amongst West Indian negroes at Panama, with 15 cases (3.3 per cent.) of large caseous nodules in the myocardium, and 62 cases (14 per cent.) of tuberculous pericarditis. Our figures for the whole 538 cases dying from tuberculosis are 5 cases of tuberculous nodular myocarditis (1 per cent.) and 80 cases of tuberculous pericarditis (15 per cent.), i.e., a slightly higher figure for pericarditis, but a lower figure for cardiac tuberculomata.

In the same series of cases, he gives the involvements of the spleen, liver and kidneys (expressed as percentages) as 58, 52 and 42 respectively. Our corresponding figures are 61, 52 and 28, i.e., almost identical for the spleen and liver, but distinctly lower for the kidneys. For illustrations of spleen and liver involvements, see Plates II–IV.

Borrel states that tuberculosis as he saw it amongst Senegalese troops in France during the war had an evolution which might be divided into two stages—(1), an initial latent period with no symptoms and which might last one, two or three months, in which there was glandular involvement only, the general condition of the patient being unaffected. Gradually, however, there is the supervention of Stage (2), in which general symptoms appear—fever, loss of weight and signs of the spread of the disease beyond the limits of the original glandular focus. Caseous pneumonia (lobar or lobular), serositis and miliary tuberculosis (either primary or following on involvement of the lungs or serosa) are the features of the second stage. This stage is usually short and death takes place in from two weeks to two months. Only rarely does one see chronic local pulmonary tuberculosis with cavity formation.

We are unable to say what proportion of our cases had an evolution similar to this, not having been in touch with the cases during life, but probably a considerable number of the cases figuring in Tables IX and XI of Appendix 7, as dying after less than one year's service, would have shown at all events a similar rapidity of development when Stage (2) supervened. The majority of these cases occurred in the mine medical officers' series of cases, and we do not know the actual duration of illness, but we have records of some cases which have died within a month of their arrival on the Rand, and of others who have been working up to within a few days of their death. In some instances, therefore, the course of the final stage is very rapid.

The figures given on p. 113 for the period of survival of cases dying in the mine hospitals are in keeping with the view that Stage (2) is here also often a very short one.
Borrel states further that the initial lesions are almost always in some of the glands draining the upper air passages (90 per cent. of cases); relatively rarely in the cervical glands; in 70 per cent. of cases in the "clavicular" glands (see p. 399), and in 80 per cent. in the tracheobronchial glands. These glands form the starting-point of the disease; they break down and further spread takes place either by the lymph or blood-stream. The glands follow on an erosive lesion or tuberculous chancre in the tonsils, pharynx, larynx or at the level of the primary bronchi. Direct initial lesions in the lungs are rare, tuberculosis starting near the apex not being found in more than 5 per cent. of cases. Pulmonary involvement in most cases is secondary to caseation in the tracheo-bronchial glands. A hilus gland may break down and discharge directly into a bronchus with the development of a massive caseous pneumonia in the middle or lower lobe. This is the commonest occurrence and was found in 30 per cent. of cases. At other times, the caseation may be lobular and more widely distributed over one lung or both lungs. Death may occur rapidly with no cavity formation, or without any further spread of the disease, but often there is a parallel blood-stream spread and in 20 per cent. of cases there was found miliary tubercle along with caseous lesions. In 25 per cent. of the cases death occurred from a purely miliary spread from the original caseous glandular focus.

In two respects our series of cases shows a very striking difference from the tuberculosis of the Senegalese as outlined above by Borrel.

In the first place, there is the comparative rarity of primary lesions in the "clavicular" glands in our cases. This is commented upon in greater detail in Appendix 7. The position in this respect of the South African Natives obviously more nearly approaches that found by Borrel among the Malagasy, and he states that the tuberculosis of the Malagasy is more like that of Europeans than is that of the Senegalese.

The second difference is that of the seat of the primary lesion. Borrel found it in the Senegalese to be in the lungs in not more than 5 per cent. of cases. In our series we put the figure much higher, probably in over 30 per cent. of cases. Whilst we have many cases similar to the majority of those of Borrel, with massive initial caseous lesions in the glands, more especially in the tracheo-bronchial, with secondary spread to the lungs or with a generalized miliary spread, we have also many where there were extensive lung lesions, especially in the upper lobes, with well-developed cavity formation and only comparatively slight and recent infection of the tracheo-bronchial glands. Also, we had quite a considerable number in which there were caseous lesions in both the lungs and the glands of about equal severity and apparently equal age. In this last group we did not feel that one could say with any certainty which was the primary focus, hence our inability to state more definitely the percentage of cases with a primary lung lesion. We believe we are justified in saying it is not less than 30 per cent., but it may be well over that figure. Some further light is thrown on the question of the primary site of infection by radiographic examination of lungs (see pp. 174–179).
The number of cases showing pulmonary cavities must be taken as indicating that many of our cases ran a somewhat longer course than did the majority of Borrel's. This may be regarded as another indication that they were occurring in slightly-immunized individuals.

Cavities in themselves are not, however, evidence of very long survival. That is indicated by the formation of fibrous tissue round them. Only 6 of our cases (314, 322, 407 and 446 in Group B, and 189 and 439 in Group C) showed this to an extent comparable with ordinary European phthisis. It is, of course, highly probable that had we had access to repatriated cases, a higher percentage of this type would have been encountered.

That healing or arrest of primary pulmonary lesions does occur is evidenced by the finding amongst the 62 cases of death from conditions other than tuberculosis of 5 cases showing such features (Cases 229, 234, 369, 447 and M128 in Group A).

Borrel also distinguishes a group of cases where the lesions, secondary to an initial glandular focus, are predominatingly in the serous sacs. This, of course, we can parallel abundantly.

Then he records a few cases showing an initial infection by the abdominal route with no lesions in the tracheo-bronchial glands, lungs or thorax, but abundant evidence in the abdomen—ascites, mesenteric glands, omental cake, matting of the intestines, etc. He excludes from this group certain cases of "type abdominale d'origine thoracique" developing mainly by blood-stream spread from a tracheo-bronchial focus.

We have also separated a series of 97 cases (Group D) in which the lesions were mainly extra-thoracic. Only in 6 of these, however, did we feel quite satisfied that the original invasion was by the abdominal route; a few were doubtful as regards the source of spread. In 75 we were of opinion that although the main brunt of the disease fell outside the thorax, the spread was definitely from tracheo-bronchial glands.

A few cases were very suggestive of the converse condition, i.e., of invasion by the abdominal route but with extension of infection to, and main development in, the thorax. Cases of this type are Nos. 7 and 59 in Group B and Nos. 55, 117, 196 and 272 in Group C.

Borrel states that he found bone tubercle relatively rare as a primary lesion, bone involvement being almost always merely a complication of a primarily glandular lesion. In our 12 cases showing bone or joint lesions (15 if we include the 3 cases in Group B) only 2 (Nos. 9 and 388 in Group D) appeared not to be obviously primarily glandular.

To sum up, it may be said that the acute, progressive tuberculosis as seen mainly in the younger Natives early in their mining career, although partly "natural" tuberculosis, is largely somewhat modified and has reached a stage intermediate between the completely "natural" tuberculosis of virgin soil and the "modified" tuberculosis of the European adult.
Before passing on to a consideration of tuberculosis associated with silicosis, we have a few remarks to make about tuberculosis of the pancreas and of the spleen.

Pancreas.—According to MacCallum the pancreas is especially resistant to tuberculous infection, and references to tuberculous lesions other than miliary tubercle are hardly to be found. Some cases described are probably really affections of embedded lymph glands.

We have observed 5 cases (Nos. 277, 354, 360 and 361 in Group C, and 427 in Group D) with definite caseation in pancreatic tissue. Nos. 360 and 361 were merely slight involvements by direct extension from neighbouring caseous glands. No. 377 showed a rather more diffuse caseation, but was probably also a direct spread from an adjacent gland. In No. 354 the whole tail of the pancreas for a distance of 5–6cm. from the tip was caseating, with some haemorrhages. Adjacent pancreatic-lienal glands were caseous. The condition may have originated by direct spread from these but, even if so, it had extended well beyond their immediate neighbourhood. No. 427 was the most interesting. In this instance, there was very little involvement of adjacent lymph glands, but almost the whole pancreas was caseating. There were numerous ulcers high up in the jejunum and recent involvement of the corresponding mesenteric glands suggesting that there had been an abundant outpouring of tubercle bacilli from the pancreatic duct. Elsewhere there were a few caseous nodules in the spleen and numerous small caseous areas in the liver.

Primary Tuberculosis of the Spleen.—According to MacCallum (l.c. p. 240), tuberculosis of the spleen, notwithstanding that there have been descriptions in the literature of a primary affection, may be regarded as always secondary to tuberculous lesions of other organs. With this dictum we are, of course, in general agreement, but would nevertheless like to record in the next paragraph two possible exceptions to it.

Amongst the mine medical officers’ cases three were recorded with lesions limited to the spleen, but as we do not know in these cases how meticulous a search was made for other lesions we will leave them aside. Amongst our own cases we have recorded two (Nos. 256 and 401 in Group A) in which there was apparently a tuberculosis limited to splenic tissue. In neither case had the disease gone on to gross caseation, but the diagnosis was confirmed by microscopic examination. Careful examination failed to reveal any glandular or other lesion in either case, but we now realize from our own experience with X-ray examination of lungs, that we might have overlooked some minute pulmonary focus. We can only claim definitely for these two cases therefore that the first gross lesions were in the spleen.

(b) Chronic Tuberculosis (associated with Silicosis.)—We have brought together in Group B 200 cases in which the tuberculosis which was the cause of death was associated with more or less silicosis.

The age and mining service distribution of these is strikingly different from that of the cases uncomplicated by silicosis. More than half of the cases fall into the age-group 40–49 and fully two-thirds into the age-period 30–49. As regards the period of mining service, the average works out at over 6 years.
Table 36 shows the percentage of cases in which the various organs and structures tabulated were the seat of tuberculous lesions.

TABLE 36.
Percentage involvement of various organs in 200 cases of Chronic Tuberculosis.

<table>
<thead>
<tr>
<th>Glands</th>
<th>Percentage</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cervical</td>
<td>12</td>
</tr>
<tr>
<td>Clavicular</td>
<td>38</td>
</tr>
<tr>
<td>Tracheo-bronchial</td>
<td>95</td>
</tr>
<tr>
<td>Diaphragmatic</td>
<td>52</td>
</tr>
<tr>
<td>Pancreatic</td>
<td>54</td>
</tr>
<tr>
<td>Lower Retroperitoneal</td>
<td>31</td>
</tr>
<tr>
<td>Portal</td>
<td>47</td>
</tr>
<tr>
<td>Mesenteric</td>
<td>36</td>
</tr>
<tr>
<td>Axillary</td>
<td>0</td>
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<tr>
<td>Inguinal</td>
<td>0-6</td>
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<table>
<thead>
<tr>
<th>Pleurae</th>
<th>Percentage</th>
</tr>
</thead>
<tbody>
<tr>
<td>Definite Tuberculous Pleurisy</td>
<td>18-5</td>
</tr>
<tr>
<td>Marked Adhesions, probably Tuberculous</td>
<td>32</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Heart and Pericardium</th>
<th>Percentage</th>
</tr>
</thead>
<tbody>
<tr>
<td>Definite Tuberculous Peritonitis</td>
<td>10-5</td>
</tr>
<tr>
<td>Adhesions, possibly Tuberculous</td>
<td>10-5</td>
</tr>
<tr>
<td>Myocardial Tuberculomata</td>
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<table>
<thead>
<tr>
<th>Spleen</th>
<th>Percentage</th>
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<table>
<thead>
<tr>
<th>Liver</th>
<th>Percentage</th>
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<td>37</td>
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<table>
<thead>
<tr>
<th>Kidneys</th>
<th>Percentage</th>
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<td></td>
<td>21-5</td>
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<table>
<thead>
<tr>
<th>Suprarenals</th>
<th>Percentage</th>
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<td></td>
<td>11-5</td>
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<table>
<thead>
<tr>
<th>Peritoneum</th>
<th>Percentage</th>
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<table>
<thead>
<tr>
<th>Intestine</th>
<th>Percentage</th>
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<tr>
<td></td>
<td>26</td>
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</table>

<table>
<thead>
<tr>
<th>Brain or Meninges</th>
<th>Percentage</th>
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<tbody>
<tr>
<td></td>
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<table>
<thead>
<tr>
<th>Bone or Joint</th>
<th>Percentage</th>
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<tr>
<td></td>
<td>1-5</td>
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<table>
<thead>
<tr>
<th>Genitalia</th>
<th>Percentage</th>
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<td></td>
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</table>

Comparison of this table with Table 34, which shows the corresponding involvements in the cases of tuberculosis uncomplicated by silicosis brings out the fact that the greatest difference lies in the much less abundant affection of the spleen, liver and peritoneum in the tuberculo-silicotic cases, although the figures are still well ahead of those for the London cases.

In this Report we are not specially concerned with the pathology of silicosis per se, merely with the modifications which it imposes upon tuberculosis when the two conditions are associated. In Appendix 7 are described the naked-eye character of silicotic and of tuberculo-silicotic lesions as they are met with in the Witwatersrand Native mine labourers. For illustrations of pulmonary tuberculo-silicotic lesions, see Plates V–VIII.

Note.—“Tuberculo-silicosis” is a pathological term originally introduced by Watt, Irvine, Pratt-Johnson and Steuart (“Silicosis on the Witwatersrand,” Appendix No. 6 to the General Report of the Miners’ Phthisis Prevention Committee, Pretoria, 1916, p. 96), as descriptive of the areas of fibroid consolidation arising as the end result of a chronic tuberculous invasion of the silicotic lung.

In this Report and in Appendix 7 the term is used in a somewhat wider sense, including under it any association of tuberculosis and silicosis, whether as separate lesions or as a combined condition, although it is also used as descriptive of a definitely combined condition when it is employed in antithesis to simple silicosis. No implication is made, however, as regards priority of one or other of the two factors, silicosis and tuberculosis.
Influence of Intermittency of Employment on Silicosis.—The development of silicosis is dependent upon the permanent arrest of silica in the lung; it is not the amount inhaled that matters so much as the amount arrested. One factor leading to the arrest of silica-dust is that, in contrast with coal-dust, it does not provoke expectoration, but the extent of arrest varies from subject to subject, and the following observation appears to have some bearing on factors influencing dust-retention.

In re-engaged mine Natives dying shortly after their return from their kraals, we have been impressed by the comparatively moderate degree of pigmentation often presented, although they may previously have worked several contracts.

If contracts be short, say, twelve months or less, and be followed by an interval of three months or more before underground work is resumed, then it would appear as if the lungs possessed considerable power of ridding themselves of the silica which has been inhaled.

In the absence of a clinical silicosis, much of the lung pigmentation is due to soot deposited on silica particles and aggregates of particles. Such soot on or with silica may be retained for years after exposure to it has ceased, but if the silica has not had time to do much damage and become permanently arrested, then both soot and silica may largely come away during a period of non-exposure to underground conditions.

In a death from pneumonia of a newly re-engaged mine Native large numbers of silica-laden cells are to be found in the alveoli, so it appears as if inhaled silica retains for some time, so to speak, considerable ability to move about in the lung and, consequently, to get out of the lung.

This ability is limited, however, and it is because it is limited that silica is a phthisis-producing dust. Many silica-laden cells appear to be retained in fluid but stagnant lymph; once this lymph becomes organized the dust-laden cells are finally trapped, but till then they have a chance of escape.

Intermittent employment favours escape and this observation may well be related to the fact that the incidence of silicosis is lower in the Native than in the European with equal total length of service, although the Native is, in most cases, more exposed to dust. In the case of Natives continuously employed, they appear to have about the same silicosis-rate as do European miners and to develop clinically recognizable silicosis in less time.

Incidentally, it may be noted here that the tubercle bacillus is about the same size as a phthisis-producing dust, and shares the ability of such a dust to become arrested in the lung without being destroyed. If the tubercle bacillus shares the ability of silica-dust to move about within the lung in response to an exudative inflammation, we may have some light thrown on the tendency of tuberculosis to flare up following on an attack of influenza—the bacilli passing from a region of less allergy to a region of more allergy.
Actually, we have very little evidence of such flare-ups occurring in the population we have dealt with in the Report. A special entry was provided for, on their own request, in the Report Form issued to the mine medical officers (see Appendix 7), in the belief that some useful information might be accumulated with regard to the lighting-up of tuberculosis after attacks of other infections. Evidence reported suggesting that this has occurred is of the most meagre, and in some of the cases where it has been supposed to have occurred it is much more probable that the so-called influenza or pneumonia preceding the tuberculosis was really the early stages of the tuberculous process itself.

Silicotic Lesions not limited to the Lungs.—In the 200 cases in which tuberculosis and silicosis were associated, there were 7 in which the lungs showed no tuberculosis. In 2 of these there was no silicosis either (the lungs showing merely an excess of pigment but without macroscopic fibrosis, the silicosis being in the tracheo-bronchial glands); in the other 5 there were silicotic nodules in the lungs.

Stress is laid in Appendix 7 on the fact that the lesions of silicosis and tuberculo-silicosis are not limited to the lungs but may be found also in various groups of lymphatic glands.

That they should be found commonly in the tracheo-bronchial glands is not surprising and, actually, they are more consistently to be found there than in the lungs, and the earliest development is usually to be seen in these glands. Out of 178 cases, 167 showed tuberculo-silicotic lesions, 8 silicotic lesions without obvious tuberculosis, 2 tuberculosi without silicosis, and only 1 neither silicosis nor tuberculosis.

What has come as rather a surprise to us, however, is the frequency with which tuberculo-silicotic lesions extend to other groups of glands, both upwards into the neck and downwards in the thorax and into the abdomen. So frequent, indeed, is the extension to the portal glands and to the glands which we have grouped together under the headings "Diaphragmatic" and "Pancreatic" that we have come to look upon the lungs, tracheo-bronchial, diaphragmatic, pancreatic and portal glands as "the tuberculo-silicotic zone," Tuberculo-silicosis in these various groups of glands has probably been observed before, but we have not seen any published account of it. For an illustration, see Fig 12, Plate IX.

To explain this curious distribution on anatomical grounds is not quite so simple.

A study of lymphatic connexions as described by Bartels makes it quite clear that there can be no direct carriage of pigment, etc., from the lungs to abdominal glands. Bartels states precisely: "There are no direct connexions known between the pleural, lung or bronchial glands with the abdominal lymph system. The outlet paths of the latter go into the thoracic duct or into the posterior mediastinal glands. Only the lymph vessels of the diaphragm provide a direct connexion
between the lymphatics of the chest and those of the abdomen through their anastomoses with the lymphatics of the pleural and peritoneal coverings thereof."

The lymphatics of the diaphragm itself, it may be said, drain into the lowest group of the retro-sternal glands and into the anterior and posterior mediastinal glands, three groups which Sappey classes together as the "diaphragmatic" glands.

Bartels says further, however: "It is questionable how far the results obtained by the injection of healthy cadavers can be applied to the living body and especially how far altered conditions such as stream reversal from pressure variations, or retrograde transport, may play a part. These are not subjects for anatomical decision."

In addition to the description of the lymphatic system of all the area concerned given by Bartels and in other anatomical text-books, an excellent account has been given by Scott and Beattie.

Three possible explanations of the distribution of lesions over what we have termed the "tuberculo-silicotic zone" occur to us.

1. That the anatomical arrangement of lymphatics is different in the Bantu and the European, and that in the former there is some direct connexion between the lung drainage and the upper abdomen. This explanation is, on the face of it, improbable, and moreover it appears to be quite ruled out of court by the fact that the same lesions are to be found in the White miners here. We can state this from our own limited observations, but we have had these observations confirmed by Dr. A. Sutherland Strachan, of the Pathological Department, South African Institute for Medical Research, who has had a much larger experience of post-mortem examinations on European miners here than we have had.

2. By two somewhat roundabout routes:

"Dust-cells" carrying all or any of the three factors—carbon, silica and tubercle bacilli—which go to the development of tuberculo-silicosis ordinarily travel from the lungs to the tracheo-bronchial glands either by the deep plexus of lymphatics running along with the bronchi or by the superficial plexus running in the subpleural tissue. Having reached the tracheo-bronchial or paratracheal glands they might then travel—

(a) To clavicular or upper retro-sternal glands, thence to lower retro-sternal glands, and from there through the lymphatics of the diaphragm and its peritoneal covering via the falciform ligament to the glands in the portal fissure;

(b) Via the lymphatics of the oesophagus. The lymphatics of the middle third of the oesophagus drain into the tracheo-bronchial and posterior mediastinal glands, those of the lower third into the superior gastric glands. Doubtless there is an anastomosis between those of the middle and lower thirds, so that a route is open between the tracheo-bronchial and the superior gastric glands whence extension to the neighbouring upper retro-peritoneal or "pancreatic" glands is easy.
These are certainly possible routes and travel by them would satisfactorily explain the development of tuberculo-silicotic lesions in the sites where they are commonly found. The occasional occurrences in even more distant groups, such as the cervical and the lower retroperitoneal, are readily understandable through their connexions with the upper retro-sternal and upper retro peritoneal groups respectively.

The only objection which we see to acceptance of these routes as the path taken is that it is not easy to see why obstruction in the tracheo-bronchial glands should set pigment, etc. (which must perforce pass through these obstructed glands), travelling by either or both of these roundabout routes, more especially as the carriage over parts of them would needs be either by stream reversal or by carriage against the stream.

(3) By direct trans-pleural carriage:

Given some degree of fibrosis of the tracheo-bronchial glands and of the various small lymph nodes and around the lymphatics between the visceral pleura and these glands, with consequent partial blockage of the normal flow from the visceral pleura towards the roots of the lungs, it is conceivable that the "dust-cells" might migrate through the visceral pleura into the pleural cavity, then through the parietal pleura and their further course would be determined by the drainage of the parietal pleura.

Passage across the pleural space would be made easier if pleural adhesions were present. Scott and Beattie (l.c.) state that "lymphatic vessels are formed in pathological adhesions in serous cavities. These lymphatic spaces are derived probably from a pre-existing lymphatic plexus by an invasion of endothelial-lined vessels. Injections carried out on adhesions show that the vessels are formed soon after the invasion of fibroblasts into the primary inflammatory adhesion." A greater or lesser degree of formation of pleural adhesions is very common in tuberculo-silicosis of any standing; indeed, it might be said to be exceptional not to have some pleural adhesions present.

When we come to consider the drainage of the parietal pleura, we find that it will satisfactorily account for the more distant developments of tuberculo-silicosis.

The anterior upper areas of the thoracic wall drain via the internal mammary (or sternal) and infraclavicular glands, a route which would account for the common finding of tuberculo-silicosis in these glands. The rarer further extension of the process upwards into the lower deep cervical glands can be accounted for by passage through or past the "clavicular" glands.

 Inferiorly, the sternal trunk vessels connect with the vessels of the falciform ligament and the convex surface of the liver. This would explain the spread to the glands in the portal fissure, with involvement of the gland behind the xiphisternum which we have so commonly found affected. The small glands lying on the central tendon of the diaphragm and usually surrounding the inferior vena cava receive some
of the drainage from the lower parts of the thoracic wall and diaphragm, and correspond with the gland or glands which we have commonly found in this situation affected with tuberculo-silicosis. There is also drainage from the lower portions of the thoracic wall to glands lying in the region of the origin of the celiac axis on the lateral aspects of the aorta. These correspond with the glands which we have termed the "pancreatic" or "upper retro-peritoneal." Further extensions to glands adjacent to these are readily understood.

Two objections may be raised to acceptance of this theory of a transpleural route:

(a) That some trace of it should be visible in the shape of pigmented spots on the parietal pleura similar to those seen so commonly on the visceral pleura. We have never observed any such. We have sometimes observed the development of flat tuberculo-silicotic plaques on the diaphragm subpleurally (see Fig. 12). These are always associated with well-developed tuberculo-silicotic lesions in the "diaphragmatic" glands above the diaphragm and in the "pancreatic" and portal glands below it. They are indicative of a blockage in the lymphatic passages through the diaphragm rather than necessarily of a transpleural carriage.

(b) Seeing that some of the drainage of the more posterior portions of the parietal pleura goes through the small vertebral glands lying near the heads of the ribs, tuberculo-silicotic lesions in these might sometimes be expected, but actually we have never found any.

On the other hand, it may be that transpleural carriage does not occur readily except when adhesions are present, and in that case these objections would largely fall away. Scott and Beattie (l.c.) in discussing the stages of dissemination of tuberculous lesions of pulmonary origin figure as Stages III and IV a transpleural spread via lymphatics in adhesions with involvement of sternal glands and glands just above and below the diaphragm. They evidently regard this as the normal method of spread prior to generalization by blood-stream invasion through the paratracheal glands and thoracic duct. They also state, in reference to tuberculosis in monkeys, that the central lobe of the liver differs from the other lobes in that it has wide connexions with the lymphatics outside the liver, and it is significant that it is only in the region of the central lobe that tuberculous deposits secondary to tuberculosis within the thoracic cavity are to be found.

Which of these routes from the lungs to the abdomen is actually followed, or whether both do not come into action, we have not decided, but it appears to us that the distinctive features of tuberculo-silicosis as seen on the Rand have made apparent to the naked eye a mode of spread of tuberculous from the lungs to the abdomen by lymphatic channels which has not hitherto been fully appreciated.

Tuberculo-silicosis a "Chronic" Form of Tuberculosis.—Attention is drawn in Appendix 7 to the resemblance between tuberculo-silicosis as seen here and the ordinary fibro-caseous tubercle of European adults.
in two respects. Firstly, in the fact that there is much fibrous tissue formation, and, secondly, in the comparative frequency with which the tuberculosis is limited to the lungs or, at all events, to the tuberculo-silicotic zone. In 70 out of the 200 cases, the tuberculosis was completely limited to this zone, and in another 49 cases the tuberculous developments outside this zone were very slight. Finally, we might point out that with the exception of a very few cases of fibro-caseous tubercle referred to in the "acute" section, tuberculo-silicosis furnishes the only cases of "chronic" tuberculosis which we have seen amongst these mine-labourers. Although silicosis may be regarded usually as a factor predisposing to tuberculosis, its possible beneficent action should not be overlooked. In a race with little resistance to tubercle, it would seem to play a considerable part in converting an acute, progressive and often rapidly fatal disease into a chronic, long-lasting one.

**Age and Limitation of the Disease in the Mine Native.**—In European adults there is commonly acquired the ability to prevent tuberculosis from becoming a generalized condition. This ability is strikingly lacking in the Native except when the tuberculosis is associated with silicosis.

To illustrate this point we have gone over the records of 681 post-mortems (the present series of 600 cases plus some others) where death was due to tuberculosis. In this series of 681 cases there were 264 in which the age was given as 40 or over. Of these 264 cases, silicosis was associated with the tuberculosis in 176, whilst in 88 there was no silicosis.

In the 88 cases without silicosis the disease was a more or less generalized condition in 80; only in 8 was it limited to the lungs or to the lungs and tracheo-bronchial glands.

In the 176 cases associated with silicosis, generalization had only occurred in 76; in 100 it was limited to the bounds of the tuberculo-silicotic zone.

Localization or limitation of the disease in the Native may be said, therefore, to be a factor, not of age, but of silicosis.

In the elderly Native, apart from the influence of silicosis, tuberculosis tends to become generalized practically as much as in the younger Native.

**Oesophageal Fistulae.**—A point of some interest is the frequency with which oesophageal fistulae had formed in relation to breaking down tuberculo-silicotic tracheo-bronchial glands (see Fig. 13). No fewer than 9 actual cases of fistulae were found (Nos. 61, 80, 86, 302, 346, 371 and 392 in Group B and Nos. 406 and 444 in Group A), whilst in 3 other cases (Nos. 383 and 430 in Group B and No. 349 in Group A) stages leading up to the formation of a fistula were found.

Oesophageal diverticula are not uncommon in relation to adjacent fibro-caseous glands and have been described as a stage in the formation of fistulae. In this series of cases we did not encounter any traction diverticula, but we have since seen one whose tip was attached to a tuberculo-silicotic tracheo-bronchial gland.
(c) Evidence bearing on Etiology.—Having made a general survey of the pathological anatomy of tuberculosis amongst Native miners, we now come to two specific questions upon which it was hoped that this investigation would throw some light.

(i) Point of Entry of the Tubercle Bacillus.—The first of these is the point of entry of the tubercle bacillus, and we think the answer to this is that in the great majority of cases infection takes place by the respiratory route.

The infrequency of involvement of the cervical glands would appear to indicate that the point of passage past the primary defences was but rarely in the tonsils, pharynx or larynx.

The commonest route is probably by passage of the bacilli from the lungs to the tracheo-bronchial glands with the development of initial lesions there. Running this fairly close, however, is the development of initial lesions in the lung substance, especially near the apices, with secondary spread to the tracheo-bronchial glands. Later studies of X-ray examinations of lungs (see pp. 174–179) show that this opinion may have to be modified, however.

Infection by the abdominal route is much less common. Among the 62 cases dying from causes other than tuberculosis (Group A) there were 3 (Nos. 66, 88 and 306) showing initial lesions in mesenteric glands, and 1 (No. 148) showing what was probably a completely healed initial lesion in that situation.

6 cases in Group D, 2 in Group B, and 4 in Group C have already been referred to on p. 161.

We have, therefore, a total of 16 cases out of the 600 in which invasion appeared to have been definitely by the abdominal route, but, as noted under Group D in Appendix 6, there were 8 others which might possibly be added. This gives a total, at the outside, of 24 cases (4 per cent.) of primary abdominal tuberculosis.

In 3 of the undoubtedly abdominal cases the initial lesions were in glands of the mesentery of the uppermost loop of the jejunum, and in several other cases we have observed that these particular glands, although sharing in the process of caseation with other mesenteric glands, were the most severely affected. We are not aware of attention having been drawn before to this particular group.

From a clinical standpoint abdominal tuberculosis is commonly looked upon here as being of frequent occurrence in the Native. This may be correct if one merely takes into consideration where the main lesions are situated, but it cannot be regarded as correct if one takes into consideration the source of the spread. The tuberculosis of the Rand Native mine labourer must be regarded as essentially a respiratory disease, only rarely an abdominal one.

(ii) Is Tuberculosis Acquired on the Mines or merely Lit Up?—If a canvass of opinion had been taken a few years ago on this question there can be little doubt but that the answers would have been overwhelmingly
in favour of the view that the tuberculosis of the Native mine labourers was acquired on the mines ab initio. The recent demonstration of the fact that 70 per cent. or more of the Natives arrive with a positive tuberculin reaction, however, has made it possible to hold the view that many of them arrive already infected, and that the disease develops either through super-infection or through the lighting-up of old foci through stress of work or other circumstances.

What has pathological anatomy to say on this point?

Well, the fact that amongst "new arrivals" with no evidence of any previous mining service dying from conditions other than tuberculosis within a few days of their arrival, there were found a certain number of cases with definite tuberculous lesions, is clear proof that some Natives do arrive already infected.

A consideration of the proportion of these cases is of some interest, and for this purpose we will limit ourselves to the figures for one year (1927) as it is easier by so doing to make a comparison with the average total complement. In that year among 73 new arrivals there were 6 cases of deaths with tuberculous lesions which unquestionably ante-dated their arrival. 6 is not a large number, but it constituted one-twelfth of all the deaths among new arrivals, and we are never likely to get any larger figure to base any argument on, as such findings are dependent upon deaths from fortuitous causes.

The actual turnover of boys on the mines in the course of a year is generally taken as being about twice the mean complement, so we will not be far out if we put the turnover for the year at 360,000. If the proportion of cases showing tuberculous lesions among those dying from other causes within a few days of their arrival holds good throughout the whole force, this would give us a figure of 30,000 cases per annum arriving with latent tuberculous lesions.

At first sight such a figure may seem absurd, but if a positive tuberculin reaction be accepted as evidence of some previous tuberculization, then a 70–80 per cent. rate of positives gives us a figure, not of 30,000 but of 270,000. Also, we ought to have found lesions not in 6 cases out of the 73, but in 55! As it is, we may regard the 6 as representing that proportion with tuberculization ante-dating their mining service and severe enough to be recognized readily at post-mortem examination, although not readily detectable during life. Also, it may be added, that we have little doubt from our later experience with X-ray examination of lungs (see p. 174) that if such examinations had been made of all these 73 new arrivals we would have found numerous small pulmonary lesions that escaped ordinary pathological investigation.

There is another line of consideration which leads us to believe that latent tuberculosis in new arrivals is by no means a negligible factor in the production of active tuberculosis. That is the number of cases in which death occurs after a period of service of only one month or less. In all, 28 such cases are recorded, 24 in Group C, and 4 in Group D.
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