If we accept Borrel's minimum figure of one month for the duration of the first—latent or glandular—stage as applying here (we have no evidence available on this point, but we may state that Professor Lyle Cummins has told us that he has information that the latent period in monkeys is also about one month) and also his minimum of a fortnight for the second stage, which we are prepared to do from local experience, then we have either to admit that if these cases acquired their infection after arrival they all died within the minimal period (this is allowing for a fortnight in hospital in addition to a month's service, and some of the cases we know did not even have that time), or else they are cases arriving already infected and passed with latent lesions. It is not credible that they all fall into the former category; some, at least, must be in the latter.

The same argument would apply to some of the cases of tuberculosis dying after two or three months of service as minimal periods cannot apply to all cases, but it cannot be pushed to the same extent in these.

It must be taken as established, therefore, that some of the tuberculosis occurring early in mining service is due to the lighting-up of infections already present on arrival but undetected at examination, or, in other words, it is of endogenous origin.

In the case of tuberculosis developing later, infection acquired on the mines, *i.e.*, exogenously, may reasonably be suspected of playing a more important rôle, either as a primary infection or as a super-infection.

Just how important a rôle exogenous infection plays is difficult to estimate. Herewith follows a consideration of this point on purely pathological grounds. Other arguments bearing on the question are considered in Chapter VI., p. 277.

The ordinary chronic phthisis of Europe is commonly held to be the result of exogenous re-infection or re-infections. One of the main arguments in favour of this view, as set out in standard text-books on tuberculosis or, more recently, as by Aschoff[61] and here summarized, is as follows :—

The *primary affect*, if not healing completely, goes on to a stage of generalization from which the individual may die or the metastasizing process may come to an end, but the local infections, the so-called organ phthisis, goes on. Thus develop such forms as chronic bone, joint or urogenital tuberculosis. *Chronic pulmonary phthisis* is rarely seen in such cases, the mutual exclusion of the simultaneous occurrence of chronic pulmonary phthisis and chronic phthisis of other organs being well recognized. It must be inferred, therefore, that the lungs are not liable to be affected by endogenous spread, and that subjects with organ phthisis are too well immunized for exogenous infections to take root. In persons in whom the primary affect has healed without any generalization, on the other hand, such high immunization is not present and in such individuals subsequent exogenous infections may be able to set up a chronic pulmonary phthisis which will then be present without the presence of organ phthisis.

How does the phthisis of our long-service Natives fit in with this conception ?

The pathological records show that in approximately 50 per cent. of the chronic cases the pulmonary phthisis is unassociated with any organ phthisis. Therefore, if the same argument holds good here, it is to be inferred that these cases have resulted from exogenous infection or infections.

Furthermore, in many of the other 50 per cent. of cases—those in which organ phthisis was present—the involvement of the other organs was obviously much more recent than that of the lungs, so that these also might be regarded as starting exogenously, although there had been a later endogenous spread.

There is, however, a possible fallacy in the whole argument as applied to these cases, associated with the presence of silicotic lesions in the lungs, a factor which does not complicate the ordinary European case.

In those cases where the tuberculo-silicotic lesion is a single one it has been observed that the situation is commonly sub-apical, *i.e.*, in the locality regarded by Aschoff (*loc. cit.*) as characteristic of secondary exogenous infections. The character of these solitary foci is, moreover, usually suggestive of being initially tuberculous and merely secondarily taking on a tuberculo-silicotic facies through the accumulation of silica and pigment in the tuberculous tissue.

The more common class of case, however, with multiple tuberculo-silicotic lesions, while also explicable on the theory either of the simultaneous inhalation of silica and tubercle bacilli or of the earlier development of silicotic lesions and the subsequent trapping of inhaled tubercle bacilli in them, is capable of another explanation.

It might well be argued that although tubercle bacilli of endogenous origin are rarely responsible for the initiation of chronic phthisis in the otherwise healthy lungs, in the case of lungs with silicotic foci such bacilli might well be trapped and be able to gain a footing in these areas of already damaged tissue.

This argument would imply two things :—(*a*) The presence of an endogenous source, and (*b*) that bacilli from this source get loose and travel around either in the blood-vessels or lymphatics.

The first implication will readily be granted. The high rate of tuberculization of the Natives has been proven, and it has been shown by Puhl (*cit.* by Aschoff) amongst others that tubercle bacilli may remain alive and virulent for many years in lesions which are calcified or even ossified and apparently quite dormant.

On the second point we have, unfortunately, no definite evidence to offer, but it may be pointed out that numerous recent workers have shown that a tubercle bacillaemia without any necessary spread of actual disease is no uncommon thing, and the possibility of lymphatic spread is mentioned because our pathological investigations have shown that lymphatic dissemination in tuberculo-silicosis may be a widespread affair.

On purely pathological grounds, therefore, it seems probable with regard to these cases of chronic pulmonary phthisis that—

(*a*) A few of the more acute cases are almost certainly of endogenous origin from the erosion of blood-vessels by slowly progressive foci with a resultant miliary spread.

(*b*) A few cases—those with solitary lesions—are almost certainly of exogenous origin.

(*c*) The majority of cases may be explained either on an endogenous or an exogenous basis.

(*d*) *Radiographic Pathology of the Lungs.*—At the conclusion of the examination of the 600 cases which form the basis of the foregoing Pathological Report, a special examination of a further series of 87 cases was undertaken in which an X-ray photograph of the lungs was taken after removal from the body. The lungs and tracheo-bronchial glands were then carefully examined from the point of view of any information given by the radiograph.

The technique employed was to remove the thoracic contents intact along with the trachea and larynx. The lungs were then inflated through the trachea with a bicycle-pump and Kaiserling's No. 1 fixing fluid run through them by means of a canula introduced into the pulmonary artery. The trachea and pulmonary artery were tied off, the main mass of the heart cut away and the photograph was then taken. We are indebted to Dr. Irvine, chairman of the Miners' Phthisis Medical Bureau for permission to have the photographs done at the Bureau.

The object in making this series of examinations was to see how the findings would fit in with the views of the pathology and pathogenesis of tuberculosis as championed by such writers as Aschoff[61] and Opie.[65]

Their view, is briefly, that ordinary pulmonary tuberculosis is an exogenous or endogenous recidive of an infection experienced in childhood and that in adults not so immunized in childhood a tuberculous infection first encountered in adult life will be of the infantile type.

According to Aschoff, the primary infection may be in any part of the lung, but the upper lobes, especially the right, are the site of predilection. It is almost always immediately under the pleura and is usually single. It is sharply delimited, rapidly caseates and is always accompanied by a somewhat larger caseous focus in the related lymph node near the root of the lung.

In the great majority of cases this primary affect heals completely. The healing takes place about both foci by the development of a specific granulation tissue which becomes transformed into a hyalinized fibrous tissue scar surrounded by ordinary non-specific fibrous tissue. In the encapsuled caseous masses calcification occurs, and they are gradually absorbed by invading connective tissue which builds typical bone. The marrow of this bone is often richly pigmented by carbon-laden cells.

The reinfection, in contrast to the primary infection, may be multiple. It occurs most often at the apex of the lung, but is more often within the substance of the lung than subpleural, so that when it heals it brings about contraction of the surface of the lung in the process of cicatrization. The productive phthisical focus resulting from reinfection also tends to caseate and calcify but only very rarely is bone formed. Especially characteristic of reinfection is the freedom from involvement of the related lymph node.

In a certain proportion of cases the primary affect does not heal but carries over to the stage of generalization or metastasizing phthisis. In such cases the process manifests itself not so much in the lungs as in the lymph nodes. Rapid caseation may take place in the whole of of the tracheo-bronchial glands, not merely in those corresponding to the primary lung focus. These may invade bronchi and bring about a rapidly-caseating aspiration pneumonia, or there may be a break into the blood-stream and metastases in various organs of the body.

Opie (*loc. cit.*) from his work in children, gives a very similar description of the occurrence and characters of primary infections and reinfections, except that (so far as we can judge from those writings of his which we have been able to consult) he does not appear to lay stress upon the development of bone in primary affects.

Ordinary chronic pulmonary phthisis as seen in most civilized communities of the present day, presupposes a previous immunization through a primary infection in childhood or youth. Phthisis in adults not so immunized in youth tends to run a virulent course like that seen in childhood in the stage of generalization. Such absence of primary immunization is the explanation of the virulent character of the tuberculosis of primitive races, not any racial feature *per se*.

Let us see how our findings fit in with these views.

The 87 *Cases*.—The cases were quite unselected, being 87 consecutive deaths at the W.N.L.A. Hospital, irrespective of the cause of death.

Group A. Showing no Tuberculosis (18 cases).—These 18 cases showed no evidence of any tuberculous infection either by ordinary examination or radiographically.

12 were " new recruits " with ages in the early twenties, with no record of any mining service and, judging by the amount of pigment in the lungs, they were all actually newcomers to the Rand. In only 1 of the 12 was there a record of a tuberculin test and that was negative.

3 cases whose ages were 30, 30 and 35 respectively were also apparently truly newcomers.

1 case *aet.* 20 had 2 months' service.

1 case *aet.* 24 was marked " time-expired," *i.e.*, he had probably served one contract of 9 months' duration.

1 case *aet.* 40 had a record of 4 years and 8 months' service.

Group B. Tuberculosis, but not as the Cause of Death (12 cases).—This is an interesting group in view of the dictum of Opie, "I am inclined to believe that more knowledge of the pathology of tuberculosis can be obtained by careful examination of the tuberculous lesions of those who die from causes other than tuberculosis than from fatal instances of the disease."

Of the 12 cases, 6 had silicotic lesions associated with the tuberculosis, 6 had no accompanying silicosis.

Sub-group. Uncomplicated by Silicosis (6 cases).—Of these 6 cases, 4 were young new recruits (1 with a tuberculin reaction recorded as positive).

1 was a Pondo *aet.* 32 with 1 year of service, and 1 was a Msutu *aet.* 40 with 15 years' service.

5 of the 6 cases showed single subpleural calcified foci and 3 of the 5 also had calcified corresponding lymph nodes; in the other 2 no corresponding glandular lesion was found.

1 of the 6 had several subpleural calcified, pigmented, scarred areas, but no corresponding glandular foci.

None of the 6 showed any tuberculous lesions other than those mentioned.

3 of the 6 might be described, therefore, as showing typical full primary affects (except for the absence of bone); 2 had primary affects subpleurally only, and 1 had multiple subpleural primary affects but nothing in the lymph nodes.

It may be noted here that although Aschoff states that the primary subpleural affect is always accompanied by a corresponding glandular focus, he quotes statistics of Puhl which show that in about one-third of cases a primary affect was found in lung alone or in lymph node alone but not in both situations.

Sub-group. Complicated by Silicosis (6 cases).—The 6 cases with associated silicosis all occurred in older subjects, with records of service varying between 2½ and 13 years.

2 cases showed tuberculo-silicotic lesions in the tracheo-bronchial glands only. 1 of these showed marked calcification in numerous glands on both sides but no apparent lung-lesion of any kind. The other showed caseation but no calcification in several glands and near one apex was a subpleural calcified area with the character of a primary affect.

1 case showed calcified tuberculo-silicotic tracheo-bronchial glands and scanty simple silicotic nodules in the lungs but nothing suggestive of any tuberculous lesion in the lungs.