At necropsy the bone marrow was found to be aplastic. There were generalized visceral petechiae and also a recent basal subarachnoid haemorrhage.

It is not known exactly when the blood picture in this case became abnormal, as the illness began when the monthly blood count was due. Previous examination had not shown any abnormality, in particular no lymphocytosis or eosinophilia. When the patient was admitted, however, the granulocytopenia was already severe. She therefore developed a fatal aplastic anaemia eight months after starting methoin therapy. The symptoms were rapid in onset, though it is possible that significant blood changes would have been recognized had more frequent examinations been carried out. There are, however, practical limits to this procedure.

Abbott and Schwab (1950) suggested that serious toxic effects were perhaps less likely to occur in patients developing a hypersensitive reaction early in the course of treatment, but such was not so in this case. It has also been maintained (though not specifically with methoin) that regular blood examinations are fruitless: that by the time changes are evident in the peripheral blood it is probably too late to attempt treatment. An alternative supposition is that blood dyscrasias are of sudden onset and therefore not subject to discovery by regular counts. Abbott and Schwab discounted this, however, and the safest course must remain that of examining the blood as regularly as possible during treatment.

Discussion

As has happened with other new preparations in recent years, methoin proves to be a valuable but sometimes toxic drug. This study confirms its usefulness in major and also in psychomotor epilepsy, and shows that dramatic results may sometimes be obtained. It is, however, a drug of definite toxicity, and its use may be fatal.

Clearly, methoin should be used only when every attempt to obtain control by other means has failed. As beneficial effects usually appear quickly, it is better not to persist with it in patients who are resistant; on the other hand, a short therapeutic trial may be made with little danger.

If methoin is used certain precautions must be routine: (1) Instruction of the patient and relatives on its possible dangers, with the warning that they must report any suspicious symptoms at once. The patient's own doctor ought also to be notified that the drug is in use. (2) It should not be used in conjunction with other drugs likely to cause similar reactions. It should not be used with troxidone, though a number of resistant cases seem to demand this very combination. (3) The patient must be seen and blood examinations carried out regularly throughout the period of treatment.

Summary

The results of methoin treatment in 24 cases of major and psychomotor epilepsy are described. A reduction of 70% or more in fits occurred in 40% of major epilepsy cases, and in 31% of patients with psychomotor epilepsy.

Details of E.E.G. changes due to the drug are given.

Toxic effects, including a fatal aplastic anaemia in one case, are described.

The value and limitations of methoin are discussed.

It is a pleasure to record the help of Dr. Denis Hill, at whose suggestion the trial was undertaken. I am also indebted to Dr. T. V. Mendelsohn for his detailed report on the fatal case,

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TUBERCULOSIS IN KENYA

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Details of the Survey

A tuberculosis survey of Kenya Colony was made between February, 1948, and February, 1949. In default of mass miniature radiography, the method of the survey was limited to tuberculin-testing, physical examination, and the examination of sputa. It was possible to get only a single specimen of sputum from each suspect, the likeliest-looking part of which was smeared on a slide, dried, and subsequently examined at the Government Laboratory in Nairobi by the Ziehl-Neelsen method. The usual methods of physical examination were employed, but care was taken to exclude all cases except those with definite and persistent signs. Unequivocal signs of cavitation were taken as a proof of tuberculosis; bronchial breath sounds and post-tussic crepitations were, unless there was evidence of recent acute infection, classified as suspicious of the disease.

Although limited in method, the survey was of considerable extent. Africans were examined at 34 different places, chosen as giving a fair sample of the more populous parts of the colony. In all, 49,812 intradermal injections of 0.1 ml. of 1 in 1,000 old tuberculin were given, and a reading was obtained 48 hours later in 42,492 instances. An indurated area more than 5 mm. in diameter, palpable 48 hours after the injection, was taken as constituting a positive reaction. A total of 2,889 physical examinations were made, and 1,298 specimens of sputum were examined for tubercle bacilli.

Control Series

Some attempt was made to rule out non-specific allergy to old tuberculin, which might be supposed to occur in the African, prone as he is to helminth and protozoal infections. Ninety-eight hospital patients were given an intradermal injection of 0.1 ml. of 5%

glycerin broth together with the Mantoux test. Fifty-four reacted to the tuberculin, and one of these, whose Mantoux test resulted in blistering, reacted to the control solution with what appeared to be a typical tuberculin reaction. Later, 89 school-children were tested with old tuberculin and at the same time with an equivalent dose of P.P.D. (Park, Davis and Co.). Forty-eight hours later 46 showed a reaction to the old tuberculin and 50 to the P.P.D. solution.

Occult leprosy has been suggested as a possible cause of false positive tuberculin reactions (d'Arcy Hart, 1932). In a recent survey of the colony Ross Innes (1948) found an average incidence of 10 lepers per 1,000 of the population. However, his figures bear no relation to the tuberculin-sensitivity rates of the same districts. A small leper colony at Msambweni, on the coast, contains 34 cases of far-advanced leprosy. These were tuberculin tested, and 29 reactors were found, or 85.3%; a comparable age group of 909 of the general population of Msambweni gave 748, or 82.4% reactors. Of the five non-reactors, two were far-advanced lepromatous cases and three had tuberculoid leprosy.

This small control series suggests that the general results obtained are not significantly affected by non-specific reactions to tuberculin.

Tuberculin Sensitivity Rates

The results of the survey are shown in Tables I and II.

TABLE I.—*Tuberculin Sensitivity Rates in 3,132 African Males and 937 African Females Resident in Mombasa and Nairobi, Kenya (May and December, 1948: Mantoux 1 in 1,000). Urban Rates*

Age Group	Males			Females			Both Sexes		
	No. Tested	Reactors		No. Tested	Reactors		Total Tested	Reactors	
		No.	%		No.	%		No.	%
Under 1	28	0	0	24	1	(4.2)	52	1	(1.9)
1-3	111	7	6.0	103	8	7.8	214	15	7.0
4-6	143	20	14.0	124	20	16.1	267	40	15.0
7-12	259	109	42.1	157	47	30.0	416	156	37.5
13-18	295	190	64.4	66	30	(45.4)	361	220	60.9
19-25	705	590	83.7	188	119	63.3	893	709	79.4
26-40	1,067	954	89.4	256	202	78.9	1,323	1,156	87.3
41-	524	502	95.8	19	18	(94.8)	543	520	95.8
All ages ..	3,132	2,372	75.7	937	445	47.5	4,069	2,817	69.2

TABLE II.—*Tuberculin Sensitivity Rates in 17,078 African Males and 16,313 African Females Resident in Native Reserves, Kenya (February, 1948, to February, 1949: Mantoux 1 in 1,000). Rural Rates*

Age Group	Males			Females			Both Sexes		
	No. Tested	Reactors		No. Tested	Reactors		Total Tested	Reactors	
		No.	%		No.	%		No.	%
Under 1	424	10	2.4	466	7	1.5	890	17	1.9
1-3	1,418	73	5.2	1,383	62	4.5	2,801	135	4.8
4-6	1,527	176	11.5	1,526	215	14.1	3,053	391	12.8
7-12	3,996	874	21.9	3,155	731	23.2	7,151	1,605	22.4
13-18	3,212	1,392	43.3	2,663	1,002	37.6	5,875	2,394	40.7
19-25	1,003	706	70.4	1,981	1,022	51.6	2,984	1,728	57.8
26-40	2,638	2,149	81.5	3,258	1,951	59.9	5,896	4,100	69.6
41-	2,860	2,345	82.1	1,881	1,163	61.8	4,741	3,508	74.2
All ages ..	17,078	7,725	45.2	16,313	6,153	37.7	33,391	13,878	41.6

There is a close similarity between these results and those recorded by Wilcocks (1938, p.44), who made a somewhat similar survey of Tanganyika fifteen years ago. His many tables cannot be reproduced here. The Kenya rates are rather higher except for the younger age groups. This is also the main difference between the Kenya findings and those of Matthews (1935), who made a survey of Zanzibar. Comparison is difficult in this case because of the more complicated technique he used.

Table III shows the low sensitivity rates found by Burrows (1935) among the southern Sudanese of the Bahr-el-Ghazal province. It is here that Lyle Cummins (1939) in 1902 found no evidence of tuberculosis "among the natives, cattle, and wild game."

MacLennan's (1935) figures for Palestinian Arabs are also considerably lower than those of the Kenya survey. He tested 11,303 urban school-children of 5-13 years of age and found 1,823, or 16.2%, reacted to 1 in 1,000 old tuberculin. Of 3,660 rural school-children, 483, or 13.2%, reacted. The figures for children of the 7-12 years age group in Kenya are 37.5% and 22.4% respectively (Tables I and II, last column).

On the other hand, a recent survey of the Gold Coast by Todd (1948) showed "no virgin areas, but high rates of tuberculinization at all ages; tests with rising strengths showed nearly 100% positive by adult age." The South African Mines Report (1932, pp. 205, 206) showed that 72% of recruits, who came from various native territories, reacted to 1 in 5,000 old tuberculin on engagement at the mines, and "repeated tests with stronger dilutions would probably give 90% reactors."

In England the Prophit Survey (1948) records that "the young adult population studied shows an incidence of tuberculin sensitization of the order of 85% for males and . . . 82% to 83% for females." In Kenya the 19-25 years group of urban residence was 83.7% for men and 63.3% for women (Table I).

The Kenya survey demonstrates the marked difference in reactor rates between males and females and between those living in town and those in country districts. Kenya, in common with other colonies, suffers from a lack of vital statistics, and there are no figures of the tuberculosis incidence in the different parts of the colony. It does seem likely, however, that the tuberculosis attack rate is higher in the towns, or at least in the two largest, Nairobi and Mombasa, than in most country districts.

TABLE III.—*Tuberculin Sensitivity Rates (Burrows, 1935) in 2,369 Sudanese Males and 1,293 Sudanese Females Resident in Bahr-el-Ghazal Province (Old Tuberculin 1 in 500)*

Age Group	Males			Females			Both Sexes		
	No. Tested	Reactors		No. Tested	Reactors		No. Tested	Reactors	
		No.	%		No.	%		No.	%
0-5	201	18	9.0	225	14	6.0	426	32	7.5
5-10	585	125	21.4	261	41	15.7	846	166	19.6
10-25	973	367	37.7	475	156	32.8	1,448	523	36.1
Over 25	610	342	56.1	332	136	41.0	942	478	50.7
All ages	2,369	852	36.0	1,293	347	26.8	3,662	1,199	32.7

Men often work for a spell in the towns and return to their wives in the Reserves when they have earned a little money. When adult males were tested in the Reserves they were asked if they had ever worked in Nairobi or Mombasa: of the 5,007 men tested, 3,029 who answered "Yes" showed an 83.1% reactor rate, while those who answered "No" gave a 72.3% reactor rate.

Disease Incidence

It was not practicable to examine more than a small proportion of tuberculin reactors. An attempt was made to examine all those with chest complaints, children who reacted and their contacts, and contacts of suspected cases of the disease, and also to collect a specimen of sputum from these people. So, besides the cases missed by physical examination—which, having regard to the type of disease the African gets and the ease with which the tubercle bacillus is recovered from his sputum, may be fewer than would be expected in European practice—it is probable that some cases of tuberculosis escaped examination altogether.

It was found necessary to divide those diagnosed as tuberculous into "proved" and "suspected" categories according to the criteria already described. The number falling into the former group for all the places surveyed averaged 4.6 per 1,000. It is felt that a closer approximation of the true state of affairs would be reached if both "proved" and "suspected" cases are combined, in which case the average incidence amounts to 11.1 per 1,000. The 1948 census gives an African population of 5,252,753. If we take the incidence of only "proved" cases found during the survey as an average for the whole colony, then there are 24,000 cases of tuberculosis in Kenya. The figure of 11.1 per 1,000 gives a total of 58,000 cases of tuberculosis, and it is felt that this is more likely to approximate to the true number.

It is interesting to find that Wilcocks estimated the incidence in Tanganyika as 11.55 per 1,000. In Southern Sudan, Burrows diagnosed tuberculosis in 173 cases, which in an estimated population of 156,300 gives an incidence of 11.1 per 1,000. In Palestine, MacLennan estimated the incidence at 5 per 1,000.

It must be made clear that the Kenya estimate is based on the number of cases actually diagnosed among the general population examined in the native Reserves. No account is taken of the few cases lying in the native hospitals of the colony. Hospital figures give no indication of the incidence of the disease. Although cases of bone, joint, and glandular tuberculosis were seen fairly commonly, there is no doubt that pulmonary tuberculosis is by far the most prevalent and most important manifestation of the disease in Kenya.

As can be seen from Tables I and II, for the purpose of tabulation the sexes are divided and each sex is classi-

fied into eight age groups. In comparing the reactor rates found in different districts, it is necessary to select one age group. Adult males are often used, but, as has been shown, their reactor rate varies with their experience of the towns. Children in the 7-12 years of age group have been chosen here. Large numbers were tested. Sexual differentiation in tuberculin sensitivity rates, at least, is not marked, and males and females may for this purpose be combined. Their peripatetic habits are free but local, and when they become infected they probably do so within their own district. Table IV compares the sensitivity rates of this age group by districts. Tests which were made at various places within the same district and tribal area have been combined. The disease incidence, computed on "proved" and "suspected" cases found in the whole sample of population tested for the corresponding districts, is also shown.

TABLE IV.—*Incidence of Tuberculosis per 1,000 ("Proved" and "Suspected" Cases) in Both Sexes and at All Ages Related to Tuberculin Sensitivity in Both Sexes at Ages 7-12 Years*

District	Both Sexes: 7-12 Years		Both Sexes: All Ages
	No. Tested	% Reactors	
Nairobi and Mombasa ..	Urban Rates 416	37.5	—
	Rural Rates		
Kapkatet (Kipsigis) ..	328	7.3	15.5
Mberi and Ciambeni ..	568	9.6	8.5
Kisii (S. Nyanza) ..	582	10.1	5.9
Embu ..	1,119	16.3	6.1
Siaya (C. Nyanza) ..	330	16.4	7.7
Maragoli (N. Nyanza) ..	188	16.6	9.5
Kajiado (Masai) ..	239	18.4	11.6
Kangundo (Machako's) ..	725	22.1	14.3
Kerugoya ..	697	22.5	8.9
Teita ..	532	29.9	8.4
Fort Hall ..	87	35.8	—
Taveta ..	105	36.2	16.0
Likoni, Kisauni, and Chamgamwe ..	367	38.4	8.0
Msamweni ..	237	39.1	9.7
Kitui ..	714	39.8	23.5
All rural areas ..	7,151	22.4	11.1

It will be seen that the highest percentage of reactors (39.8%) occurs in Kitui. The Kitui Reserve is dry and infertile (typical groundnut country), and the Wakamba living there are in a miserable state of poverty. They suffer from endemic malaria, schistosomiasis, and helminth infestation. They seem to be dirtier and to have a greater incidence of scabies than other Africans met with on the survey. The greatest number of "proved" and "suspected" cases (23.5 per 1,000) was diagnosed here.

After Kitui, the highest reactor rates were found at Msamweni, 35 miles south of Mombasa, and at Likoni, Kisauni, and Chamgamwe, all on the coast in the neighbourhood of Mombasa. In these places a comparatively low disease incidence rate of 9.7 and 8.0 per

1,000 respectively was found. Next come the colony's two principal towns, Nairobi and Mombasa. For various reasons, no estimate of disease incidence was possible here. Africans living in the towns are usually at work, and if they fall sick either go to hospital or, in the case of pulmonary tuberculosis, more often return to their Reserves. Thus, although it is likely that tuberculosis often develops in the urbanized African, it is not expected that a very high disease incidence would be found except among the indigenous population.

Wilcocks (1932) found that reactor rates and disease incidence in Tanganyika varied in direct proportion and that the former could be taken as a measure of the latter. This generalization is partially true of Kenya. Obvious discrepancies are the low sensitivity rates but relatively high disease incidence found at Kapkatet, Mberri, and Ciamburi—all remote and rather isolated up-country areas: while the coastal districts, as exemplified by Msambweni, Likoni, Kisauni, and Chamgamwe, show the reverse tendency with very high reactor rates and comparatively low disease incidence. It is suggested that the first phenomenon denotes a recent introduction of tuberculosis, while the second indicates a more prolonged exposure to the disease.

Type of Tuberculosis Seen in the African

Most observers are agreed that Africans suffer from a more acute and fatal type of tuberculosis than Europeans. Thus Vint (1937), in an analysis of 1,000 consecutive necropsies in Nairobi, found tuberculosis to be the cause of death in 132 cases. "The post-mortem findings are those of a low septicaemia, resembling acute miliary tuberculosis in European children"; while Wilcocks (1938, p. 52) finds "a preponderance of cases of acute galloping consumption. . . . These natives lie midway between the completely primitive races and our relatively resistant selves." Mine workers on the Rand were found to develop an acute form of pulmonary tuberculosis characterized by glandular involvement and miliary dissemination (South African Mines Report, 1932, p. 258). Post-mortem evidence from Mulago Hospital, Uganda, leads Davies (1947a) to suppose that "pulmonary tuberculosis as met with in Uganda is rarely of the 'adult' reinfection type but, in the vast majority of cases, is the result of primary infection leading to the development of a ghon focus, and this does not heal, but leads to dissemination of the infection with resulting death."

Gelfand (1944), writing of the Southern Rhodesian native, says he has never seen an African recover from pulmonary tuberculosis; and Davies (1947b) quotes Trowell as having an equally depressing experience in Uganda. Oswald (1946) had the opportunity of studying the disease in 416 Africans who developed pulmonary tuberculosis while serving in the Army in the Mediterranean area. Forty-seven cases which came to necropsy had a history of illness for an average of 5.4 months. He describes the usual destructive caseous pneumonic lesions, and found glandular enlargement in 96% of these cases. Caseation was present in 66%, and in 92% the mediastinal glands were involved.

The contrary opinion, that a chronic form of pulmonary tuberculosis is not rare in Africans, is less usually expressed, but Wilson (1928) believed it true of Kenya, and Burrows (1935) of the Sudan. Fennel (1932) writes of cases in the Butterworth area of South Africa: "Many were fairly chronic, lasting at least three years

and over, showing that the natives, under their own conditions, had a considerable power of resistance." Allan (1932), who made an extensive survey of the native Reserves in South Africa, found that most of the cases with pulmonary tuberculosis were "more or less chronic."

It will be noticed that pathologists, basing their opinion on necropsy reports, are agreed on the extreme acuteness and short duration of pulmonary tuberculosis in Africans. Clinicians and field workers tend to be less pessimistic, and some believe that the consumptive African may survive some years under the conditions he is used to. The difference is to be explained by the extreme degree of selection in the material available to the pathologists. Cases of pulmonary tuberculosis are not popular in overcrowded native hospitals, facilities for treating them are not usually available, and proper accommodation is lacking. The sick African is unwilling to spend long periods in hospital, especially when no active treatment is being carried out and he feels little benefit to his health. For this reason, only those pulmonary tuberculosis subjects who are too ill to be discharged or to drag themselves from hospital are likely to die there. Such cases comprise the post-mortem material of the pathologists.

During the Kenya survey a definite history was obtained from 70 of the 106 "proved" cases of pulmonary tuberculosis diagnosed. No great reliance can be placed on this, but the average duration of the disease was given as 19 months. In four cases some proof of the length of history was forthcoming, for these men had been discharged from hospitals as proved cases of pulmonary tuberculosis. Their histories were of 53, 52½, 48, and 37 months.

The very fact that infection, as demonstrated by the tuberculin test, is so widespread would seem to favour the existence of at least some chronic infectious perambulatory cases. For how could the majority of the population be infected by those acute pneumonic cases, prostrated by toxæmia and dying after a few months, which the pathologists describe?

The fact that cases of pneumonic phthisis do so badly with collapse therapy would also seem to indicate that such cases are not without exception. For Davies (1946), after 20 years' work at Kibongoto, in Tanganyika, reports an encouraging survival rate in 560 cases of pulmonary tuberculosis treated with artificial pneumothorax and ancillary measures: 42% alive after 3 years and 22% after 10.

These observations show that the fulminating type of disease, described by the pathologists as resembling progressive primary infection, is not without exception. Anyone who has had experience of tuberculosis in East Africa would agree that its usual course is much shorter and more acute than that seen in European practice.

The Kenya survey, and indeed every tuberculin-testing survey made in Africa in recent years, shows Davies's (1947a) assertion, that the African never recovers from his primary tuberculous infection, to be wrong. The majority of adult Africans have been infected and show no evidence of the disease. So far as this factor is concerned, the state of affairs is like that in Europe. However, it is probable that adult primary infection not infrequently leads to progressive tuberculosis in Africans when they are exposed to a tuberculous environment. Stott's (1950) investigation at Nakuru gaol, where tuberculosis is rife, shows that an undue mortality falls on

the small group who enter the gaol tuberculin-negative. It is likely that uninfected adult Africans, coming from their reserves to work in the towns, also are liable to develop progressive primary disease.

In view of the high tuberculin-sensitivity rates found all over the colony, it hardly seems possible to explain the extremely severe and fatal type of tuberculosis which is most commonly seen solely in terms of adult progressive primary tuberculosis, although this might in part account for the observation that tuberculosis in Africa tends to be more severe in the towns than in the reserves.

It is usually supposed that the chronic fibro-caseous type of disease commonly seen in Europe results from a partial immunity conferred by childhood infection. Many children have been found to be tuberculin-sensitive in Kenya, but only a few adults develop the chronic apical disease of Europe. It would seem that a healed primary infection may not have the same immunizing power in the African as is usually ascribed to it in the case of the European. This contention is supported by the findings of the South African Mines Report (1932, p. 97) on tuberculosis among African gold-miners on the Rand. These Africans were graded into four categories on engagement at the mines, according to their reactions to serial tuberculin tests—"positive plus," "ordinary positive," "weakly positive," and "negative" reactors. It was subsequently found that the incidence of tuberculosis was 2.9 per 1,000 for "negative" reactors, 4.5 per 1,000 for "weakly positive," 6.5 per 1,000 for "ordinary positive," and 10.3 per 1,000 for "positive plus" reactors. In South Africa Professor Lyle Cummins (1932) found that "fully 50% of healthy natives arriving at Johannesburg were capable of reacting to 1 in 1,000,000 tuberculin dilution, whereas in tests carried out by me on clinically non-tuberculous adults in Wales only 25% were found to react positively to a dilution of 1 in 100,000." In Kenya serial tuberculin-testing was not done, but of 37,460 Mantoux tests made on the general population 1,969 showed vesiculation—that is, 5.3% of the total tested and 11.8% of all reactors. In England d'Arcy Hart (1932) found less than 1% of the total tested and 2.3% of the positive results vesiculated. Wilcocks (1938, p. 31) found that in Tanganyika comparable percentages were 3.2% and 6.1%.

It does appear, therefore, that Africans are more sensitive to tuberculin than are Europeans, and it seems likely that this factor is associated with the distinctive type of disease they are liable to get.

If the same significance attaches to the tuberculin test in Africans as it does in Europeans—and there is no reason for believing otherwise—then it seems that both races are able to sustain a primary infection almost equally well. This observation is of fundamental importance, for "racial immunity" is usually thought to condition this ability. May it not be rather that racial immunity is a reflection of the power, not of sustaining the primary infection, but to form antibodies to the invader and so modify an aftercoming infection? It is not pertinent to this thesis whether the infection be endogenous or exogenous.

The African, when infected, develops a high degree of allergy to tuberculo-protein, as demonstrated by his extreme sensitivity to tuberculin, but little concomitant immunity, as exemplified by the acute infantile type of disease to which he is prone. The hyperallergic state

renders him liable to an acute and destructive type of tuberculosis on reinfection, while lack of immunity deprives him of the power of confining the lesion, as in European phthisis, but leaves a "secondary" infection unmodified except by purely allergic factors, so that it often assumes the characteristics which we are accustomed to attribute to progressive primary disease in Europeans.

Summary

A description is given of a tuberculin-testing survey in Kenya.

A small control series did not show any significant tendency for non-specific factors to interfere with the results.

The sensitivity rates were found to be high in Kenya, and are compared with those of other surveys.

A greater percentage of men than women were found to be sensitive to tuberculin. Urban residence was associated with an increased sensitivity rate.

The disease incidence is thought to be about 11 cases per 1,000. If this is correct there are 58,000 cases of tuberculosis outside the hospitals in Kenya.

A degree of correlation is demonstrated between the reactor rate and the disease incidence in the various districts surveyed. Exceptions are discussed.

Reasons are given for supposing that, although the acute fulminating type of tuberculosis, resembling a progressive primary infection, is undoubtedly very common in Kenya, it is not without exception, especially in the rural areas.

An attempt is made to correlate high tuberculin allergy with the African type of pulmonary tuberculosis.

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During the period October 15 to November 6, 1950, 570 U.S. Navy medical reserve officers arrived at Brooke Army Medical Centre, San Antonio, Texas, for assignment to active duty with the Army. These officers were interviewed by a staff officer from the office of the Surgeon General for professional classification and assignment. They were given one day's "processing" in connexion with their entrance on active duty and received a three-day intensive orientation course before leaving for their units.