

## SPINAL TUBERCULOSIS IN NIGERIA

Arris and Gale Lecture delivered at the Royal College of Surgeons of England

on

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by

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I AM CONSCIOUS of the honour of being permitted to speak to you to-day, an honour which you have bestowed not so much perhaps on me as indeed on the university and teaching hospital at which I have worked for the past 10 years, the University College, Ibadan, and the University College Hospital, Ibadan.

I am informed that Percivall Pott was one of the earliest lecturers. This is indeed an encouraging omen for my discourse. I hope also that there will be enough references to anatomy, or at least distorted anatomy, to satisfy the spirits of Edward Arris and John Gale.

Ibadan, with its three-quarter million African inhabitants, lies in the tropical forest belt of the Coast of Guinea, in the Western Region of Nigeria. Tuberculosis is one of the most important diseases in that area. It is estimated that by the age of 20 years, 80 per cent. of the population have been infected as shown by sensitivity tests (W.H.O., 1958). As to the general picture of tuberculosis, lung disease is often seen in the late stages, "destroyed" lung being common. A great worry are the drug-resistant lesions. These are produced in two ways: firstly, there are the victims who have been treated by professionally or morally unqualified persons; secondly, there are the absconders who abandon treatment once they feel better.

The pattern of glandular tuberculosis differs radically from that seen in the old days in areas of widespread bovine infection. Instead of the often localized tonsillar gland-complex, cervical involvement is diffuse and not amenable to excision, presumably rising into the neck from the mediastinum. Tabes mesenterica, the calcified glandular complex in the ileo-caecal mesentery, is never seen in adults. We presume that, as fresh milk is not consumed in Ibadan, bovine infection does not exist.

On the other hand, generalized tuberculous adenopathy is common and can lead to a particular form of lymphoedema of the extremities or the breast.

Tuberculous peritonitis is common, and occasionally tuberculoma of the liver is difficult to differentiate from malignant hepatoma.

There is a curious near-immunity of the renal tract to tuberculosis whereas the genital tract is not so protected. Epididymitis and tuberculosis of the cervix, tube and ovary are by no means rare. This is an

interesting reflection on the general validity of the concept of so-called "genito-urinary" tuberculosis. In our part of the world there certainly is no such entity.

Similarly tuberculous dactylitis—spina ventosa—is not seen, and if one wishes to instruct students on the subject one has to fall back on the osteomyelitis of the digits caused by smallpox of which we had a dreadful epidemic a few years ago.

Tuberculous pericarditis is common and can often not easily be distinguished from certain forms of endomyocardial fibrosis. In fact, tuberculosis enters almost all differential diagnosis and often constitutes a reasonable guess.

Among the extra-pulmonary forms, tuberculosis of the spine is the most frequent. A full survey of the condition as seen and treated in 207 patients is about to appear elsewhere and to-day I will therefore discuss only some aspects of the problem.

As we have seen lately, on an average, two new patients each week, it is clear that, with our hospital of 500 beds and no other accommodation whatsoever, the problem of treatment is quite beyond the scope of conventional methods, and a simple, cheap and efficient form of therapy had to be devised, no matter how much it might offend against established conceptions, teaching and practice (Konstam, 1958). In this aim we were aided by two factors: firstly, the beginnings of experiments with ambulatory treatment of the pulmonary form around 1952 at Ibadan, and, secondly, by the particular pattern of the disease process in the peoples of the West Coast of Africa of which we shall hear more presently.

Often it seems to me as if surgeons generally did not appreciate the full power of the new antituberculous drugs. In the latest edition of a popular British textbook of surgery the drugs are listed among "general systemic measures", the specific means, of course, being immobilization and surgical procedures. In the U.S.A. 725 million dollars are spent yearly on tuberculosis, and more than half of this sum is used for hospitalization. At Ibadan we had to trust the drugs far more than is necessary in most places where bed space and operating time are plentiful. Because we had no choice we have learned that constitutional (whatever that may mean) and institutional treatment fade away in importance compared with the drugs provided one can get it across to the patients that they must take them as often and as long as they are told.

## THE METHOD

The method, then, for the patient who can walk consists in attendances once a month, X-ray control at 3-monthly intervals, and the taking of P.A.S. and I.N.H. in cachet form for at least one year. Streptomycin is

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not used in the patients without severe C.N.S. involvement. Distances to the hospital are too great and our hospital organization could not cope with any mass injection regime. Fortunately it is not necessary either.

Plaster-of-paris jackets or "Minerva" collars are only used when pain or muscle spasm makes it desirable. We give them to the patient who holds his chin up with his hands or has his hands on his thighs when walking. Then they are much appreciated. Otherwise, they are not used as they become soft from tropical rain or stained and soggy from palm oil. Also they are hot and harbour vermin, and, again, they are not necessary.

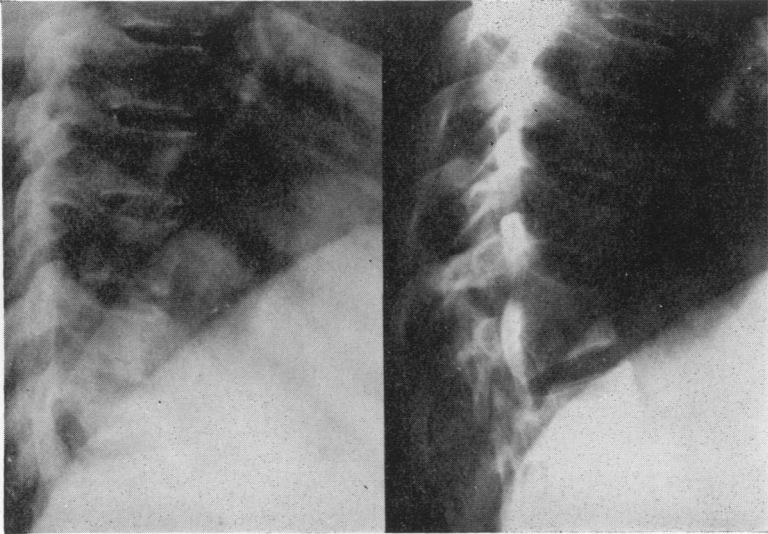


Fig. 1. X-rays showing a good result from the treatment of spinal tuberculosis with drugs.

This "medical" treatment of a condition hitherto classed as "surgical tuberculosis" has yielded very gratifying results. Perispinal abscesses disappear, necrotic and sequestered areas recover, consolidation occurs almost without exception and, most important, paraplegia does not, as a rule, develop once the course of drugs has got under way and remains uninterrupted. Figure 1 shows a typical good result. Figure 2 shows the effect of the weight of the spine above the lesion exerting itself in the manner of a "compression arthrodesis" (Charnley, 1953).

It is obvious that increasing angulation is sometimes the price that has to be paid. The degree of angulation that will result is difficult to predict. In order to assess, in measurable terms, what happens we determine the angle formed by two lines drawn through the upper and lower borders of the vertebrae in question. Figure 3 shows angulation before treatment and after consolidation. I think that ideally an attempt should be made

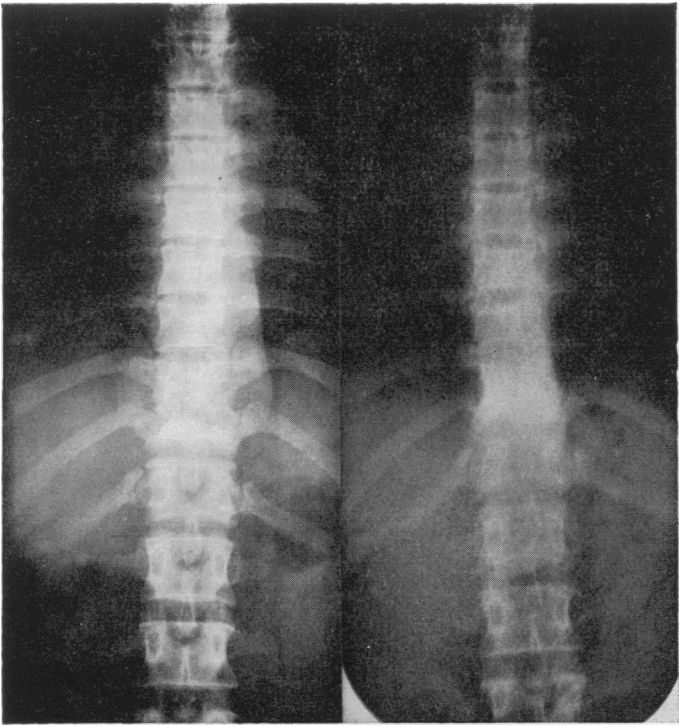


Fig. 2. X-rays demonstrating the spine simulating a "compression arthrodesis".

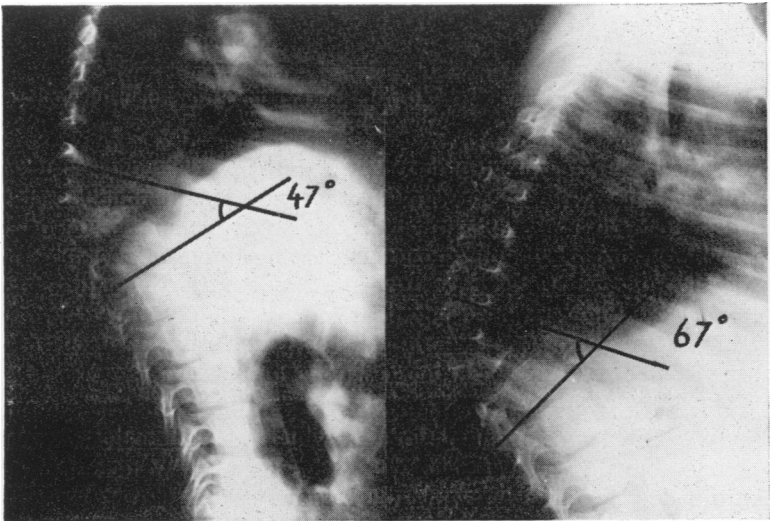


Fig. 3. X-rays showing angulation.

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in children to prevent severe angulation by more orthodox methods on account of cardio-respiratory complications which may ensue in later life, but at present I am prepared to face increasing angulation. The aim embodied in the name "orthopaedics", meaning "straightening children", is an anatomical and not a functional one and can, up to a point, be disregarded provided there is no risk of C.N.S. damage. Of this risk we have now no fear. Furthermore, the angulated spine is capable of considerable compensatory changes. Figure 4 shows how, in a patient with old disease and sinuses, a thoracic lordosis has developed and how vertebrae can elongate in compensation. This elongation can occur in the lumbar as well as in the dorsal segment.

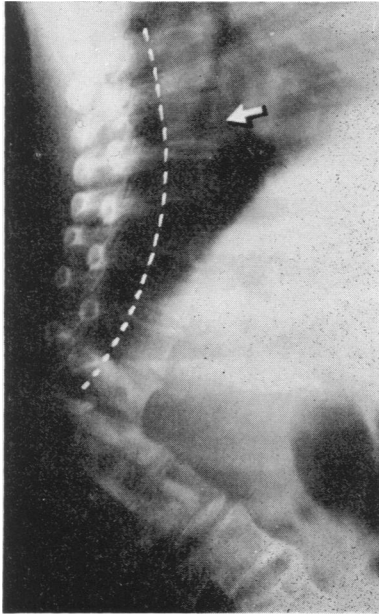


Fig. 4. Elongation of vertebrae in thoracic lordosis.

### THE ABSCESS

Let us now consider some aspects of the abscess problem. It is inherent in our method of therapy that abscesses which accompany all active lesions are disregarded if they cause no symptoms. Of their role in the production of paraplegia we shall speak presently. Otherwise they are aspirated or incised when they reach the surface or cause symptoms such as hip flexion or stridor. The rest are left to absorb under drug treatment. A large retropharyngeal abscess with destruction of the upper cervical spine is shown by Figure 5. Under treatment a solid bony mass has formed and the pharynx is back in its place. No surgery was employed and the patient remained fully ambulant. What we call a thoracic-inlet

abscess results from disease in the lower cervical or upper dorsal spine (Fig. 6). The pleura is stripped away laterally by the abscess and returns to its former normal position as the abscess absorbs.

The well-known spindle-shaped abscess forms in disease of the mid-dorsal segment. It is called "bal de Rugby" by our colleagues in Dakar. The highest level of the spindle corresponds to the level of the disease. A horizontal line drawn from the spindle summit on to the spine points to the maximal disease focus.

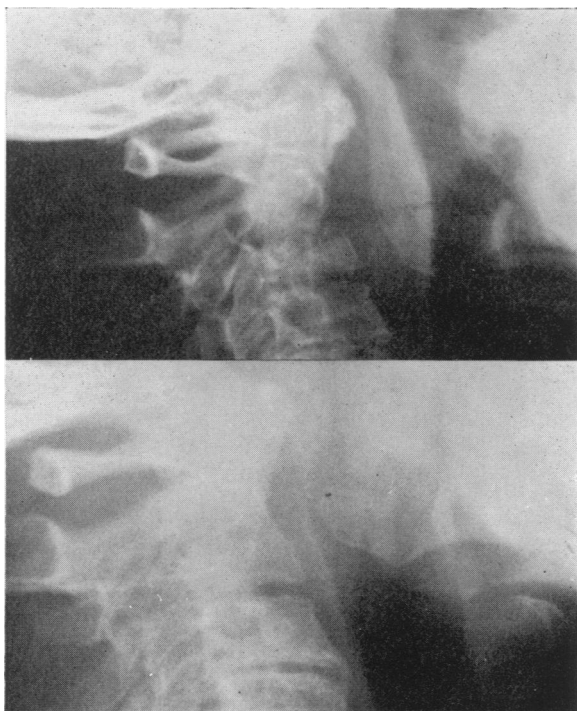


Fig. 5. Retropharyngeal abscess with destruction of the upper cervical spine.

The "petering abscess" characterizes low dorsal and upper lumbar disease. Here the abscess has an ill-defined lower border where the diaphragm overlies the site where the pus leaves or enters the chest. This is supposed to occur through the medial lumbo-costal arch. In the post-mortem room I have, however, been unsuccessful in making fluid injected into the psoas sheath ascend upwards beyond the level of L 1, admittedly in a cadaver with no spinal disease present.

Below this level we have, of course, the psoas abscess. This localization can give rise to diagnostic difficulties. In several instances patients were referred to us with a diagnosis of tuberculous hip on account of hip

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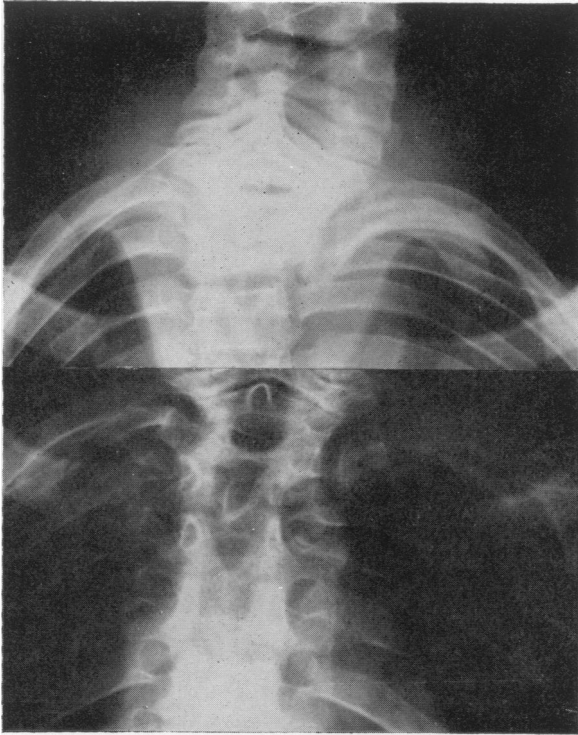


Fig. 6. Thoracic-inlet abscess.

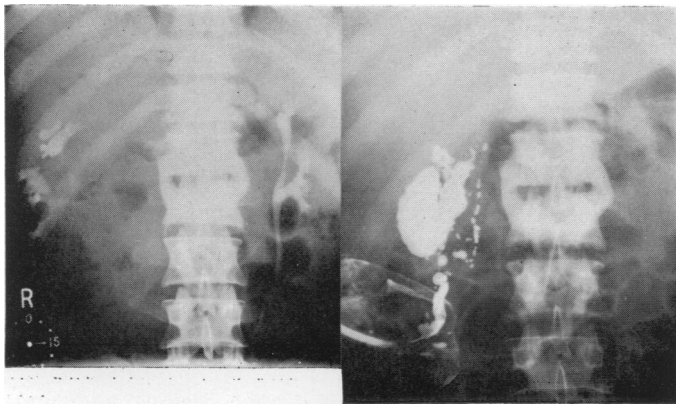


Fig. 7. Intravenous pyelography showing the right kidney displaced due to a large abscess.

flexion and calcification around the joint. In such cases there is no joint disease but calcification of the psoas tendon down to the lesser trochanter. The joint is subluxated from effusion and flexor spasm, but the origin of the trouble lies in severe spinal disease with psoas abscess.

Another diagnostic pitfall is shown in Figure 7. This was a young man who had a large stony-hard swelling in the right loin. Intravenous pyelography showed the right kidney well pushed to the right. There was a "petering abscess" and a tell-tale bony bridge between L 1 and L 2 of the kind to be discussed later. An enormous abscess was drained through a loin incision and the patient recovered quickly.

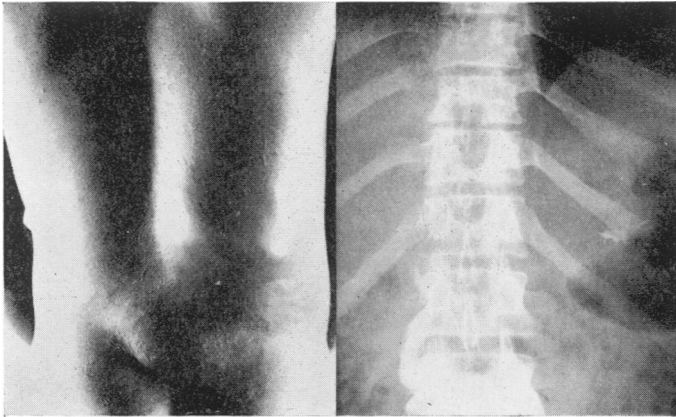


Fig. 8. "Cold" abscess in the hollow of the sacrum.

### THE "COLD ABSCESS" PROBLEM

Let us for a brief moment consider the meaning of the term "cold abscess". In the early days of the development of our hospital we predominantly saw late manifestations of the disease and people came to us as a last resort. Now, with the clinic well established, we see early cases with more frequency and the pattern of what we see is changing, a development which pleases us greatly.

Thus, a number of patients have submitted themselves lately with early and rather fulminating symptoms such as high fever, multiple lesions and often minimal bony changes but with abscesses of considerable size and tension.

The patients with older lesions are, of course, always with us. They show signs of exuberant ligamentous and bony repair and often have abscesses which have travelled long distances along and across tissue planes and which have eventually reached the surface in the usual places but sometimes in unusual ones such as the posterior wall of the rectum.



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Figure 8 shows such a chronic abscess, situated at the hollow of the sacrum. As is the rule, an unqualified local colleague, formerly called a native healer and now known as traditional healer, had been in action first, leaving the scarification marks clearly seen on the photograph. These people take a lot of pains in localizing the disease, which in this case he attacked, not where the pus was pointing, but higher up on the right, where our X-ray showed a completed healing process! These scarification marks can be of great help in the absence of visible rubor or lymphangitis in the pigmented skin. They correspond to the hot-water-bottle erythema of our gall-stone ladies, and are of about the same use.

Although it is customary to speak of a tuberculous abscess as "cold" it seemed that only these latter deserve that name but that there is nothing "cold" about the acute ones. It is permissible to think of bone tuberculosis in terms of tuberculosis of the lung, namely as of exudative and proliferative types or stages. Consequently, in collaboration with Professor Patrick Collard of the Department of Microbiology and his assistant, Dr. Okubadejo, and with the help of my house surgeon, Dr. Nkere Uwem, the humoral defence mechanisms were studied in 19 adult patients in whom pus could be obtained either by aspiration or by operation.

The fixed antibody (allergy) was determined by the Mantoux test and the circulating antibody by the haemolytic modification of the Middlebrook-Dubos test (Sherris, 1953). Further, specimens of the pus were sent to the laboratory for a differential count of the cells and a total and differential white count was done on the patients' blood.

There were four groups:

- Group 1*      High allergy combined with moderate circulating antibody titre (seven patients).
- Group 2*      Low allergy combined with moderate to high antibody titre (eight patients).
- Group 3*      High allergy with negligible circulating antibody titre (two patients).
- Group 4*      Low allergy with negligible circulating antibody titre (two patients).

Judged by the clinical picture, six of the seven patients in Group 1 were acutely ill, the mean evening temperature over the first three days under observation being 101.4° F. The eight patients of Group 2 were not acutely ill and only in one was the temperature 100° F. The X-ray changes in this group showed repair taking place in various stages or completed, or there was a chronic sinus or abscesses distant from the lesion.

It then looks as if the disease pattern falls into the following stages:

No infection	<	Allergy - Circulating antibody -
Primary infection	<	A + CA +
Recovery	<	A + CA -
Re-infection acute	<	A + + CA +
Re-infection chronic	<	A - CA + - + +

It is noteworthy that in the course of this study we have had two young girls, both of whom developed flaccid paralysis with loss of sphincter control accompanied by hyperpyrexia within 24 hours of the Mantoux test being carried out. In one who had not been paraplegic before, limb movements returned within 36 hours of the catastrophe and she is now well and walking. In the other, a previously spastic paraplegia turned into a flaccid one which later returned to the spastic state. It is probable that an allergic reaction had produced this dramatic picture.

In view of these findings it is arguable whether the acute phase of the disease could not be influenced by adding drugs of the steroid or anti-histamine group to the antituberculous drugs.

Finally, as far as the cell counts were concerned, none of the patients showed marked leucocytosis in the blood and all had normal differential counts. The notable feature of the differential count on the pus was that in both the acute and the chronic group there was a high polymorphonuclear content, a mean of 75 per cent. in the acute and of 71 per cent. in the chronic group. A high lymphocyte count had been anticipated.

## BRIDGES

We have said in the beginning of this lecture that the disease pattern in West Africa is a factor which makes our ambulant drug regime feasible. Unlike the situation in Central Asia, where rarefying and destructive forms are the rule (Serafino *et al.*, 1960), there is, in our part of West Africa, as also farther north at Dakar, a strong tendency of the diseased adult spine to consolidate and, as it were, carry out an anterior fusion on itself. This phenomenon of bridging is often associated with low-grade activity and such patients often produce large quantities of pus and are first seen on account of a sinus or a pointing abscess. It would appear that often it takes about as much time for pus to appear on the surface as it takes for the bridge to form. Figure 9 shows such a patient. Tuberculosis was

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proved. Extensive bridging is seen “comme le stalactite vient à la rencontre de la stalagmite”, as they say at Dakar. Also seen in this illustration is the fact that bridges form at levels beyond the lesion in the vertebra.

The mechanism of this bridge formation is obscure as is the exact plane in which the new bone forms. Figure 10 was taken while exploring a huge lumbar abscess in an acute case with an additional high dorsal lesion causing paraplegia. The bridge shape of the abscess cavity is evident and a bony bridge might well be laid down later as if poured into this form.

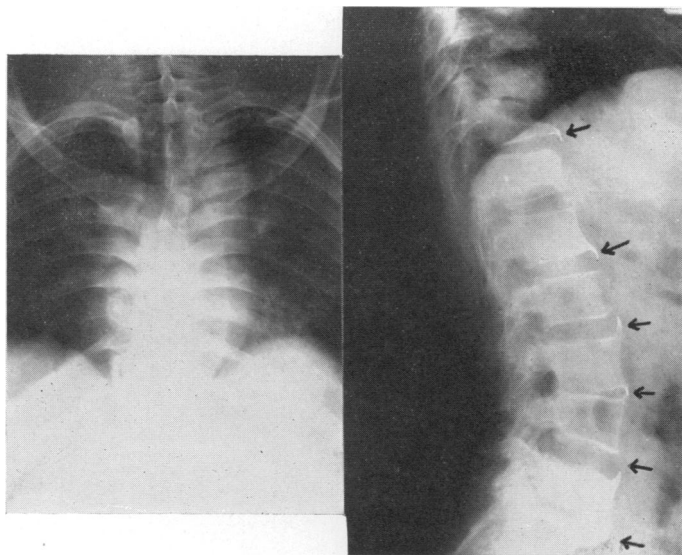


Fig. 9. X-rays showing the formation of bridging.

By courtesy of Professor Alistair Smith of our Department of Anatomy, I have been able to study the spine shown in Figure 11a. It shows low lumbar and sacral tuberculosis and bridging in the dorso-lumbar segment. Some of the bony spurs are not actually united yet but still articulate with each other, possibly due to interposition of part of a disc.

Various manifestations of the “physiological ageing process” are also seen at Ibadan as elsewhere. Figure 11b shows another specimen from the collection of Professor Smith. This is an example of what is called “unilateral thoracic spondylosis” (Shapiro *et al.*, 1960), the condition being usually, as in this case, accompanied by hypertrophic changes in other parts of the spine. The smooth bridging in the dorsal spine has been ascribed to the left position of the aorta, but it has also been ascribed to right-handedness (Guentz, 1937). It is worth mentioning that in our case

a zone of distinct calcification runs, however, right down the right side well beyond the zone where the aorta could exert a preventive role.

The formation of bridges in osteoarthritic lesions has given rise to much other speculation. Schorr *et al.* (1956) suggest that lime salts liberated by rarefaction of vertebral bone may be deposited in vertebral ligaments whose tension or mobility are diminished. Oppenheimer (1942) also holds that the different forms of calcification and ossification of vertebral ligaments have two factors in common, disuse of ligament and rarefaction of vertebral bone. These views may have some application in tuberculosis also. The fundamental difference, however, is the presence of the abscess which appears to play an essential part in the development of the bridges seen in West African spinal tuberculosis. As abscesses shrink, bridges may



Fig. 10. X-ray showing lumbar abscess with a bridge-shaped cavity.

form (Fig. 11c). It would appear that they are laid down on the outside of the ligaments and within a shrinking abscess cavity. For this reason also it might be wiser not to interfere operatively with a perispinal abscess unless one is forced to by paraplegia.

### PARAPLEGIA

Finally let us, for a moment, consider the problem of paraplegia, as we meet it at Ibadan. Our classification places the patient into our scheme of ambulant treatment and is strictly a practical one. It is as follows:

- Stage 1* Spastic but able to walk somehow.
- Stage 2* Unable to walk but voluntary movements present.
- Stage 3* No voluntary movements: paraplegia-in-extension.
- Stage 4* Paraplegia-in-flexion or flaccid paraplegia.

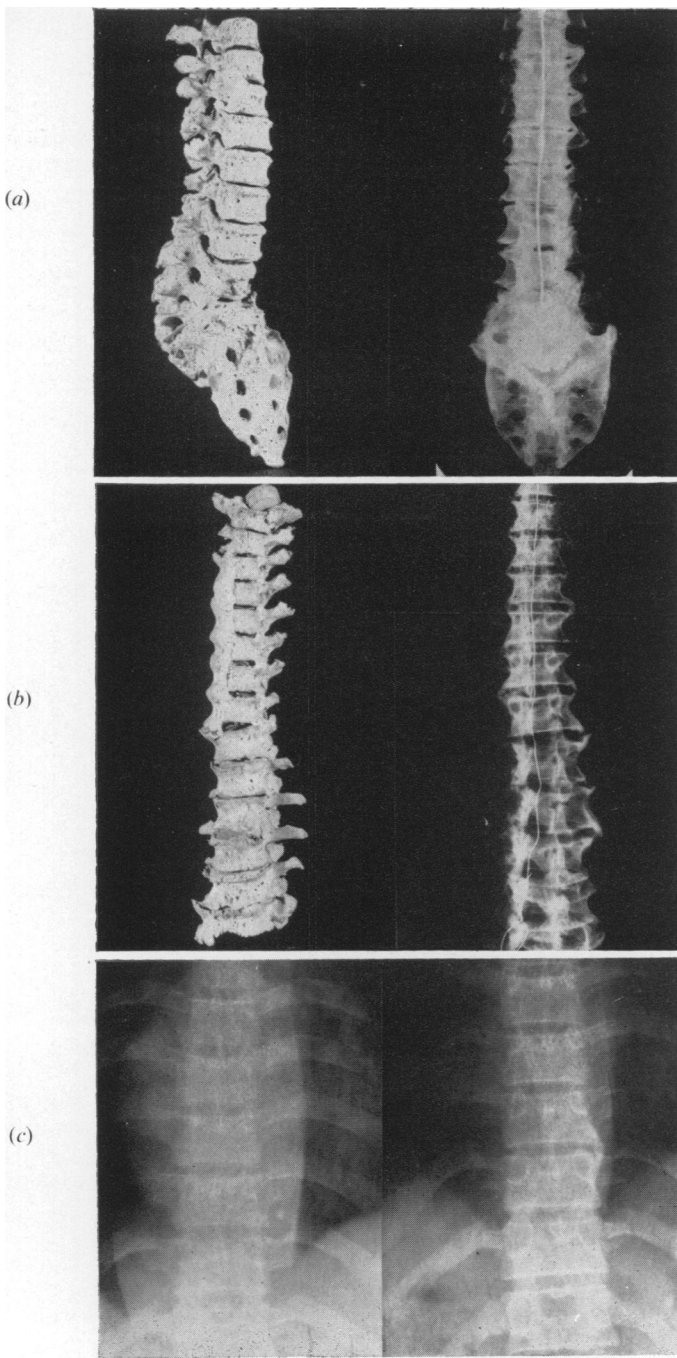


Fig. 11. (a) Tuberculosis of the lumbar and sacral spine with bridging in the dorso-lumbar segment. (b) Unilateral thoracic spondylosis with hypertrophic changes in other parts of the spine. (c) X-rays showing formation of a bridge in a shrinking abscess.

Patients with Stage 1 paraplegia are not (and cannot) be admitted to hospital—there are too many of them. From Stage 2 onwards we try to admit them with the main aim of making them ambulant again so that they can join the outpatient clinic. We make the perispinal abscess the main objective of our attack. We have already said that angulation *per se* is not as a rule the causative agent in the early case. Minimal radiological bone changes are often associated with severe paraplegia with abscesses under tension. In our experience there is ample evidence that the pressure of the abscess, liquid or inspissated, and not sequestra, debris or other bony

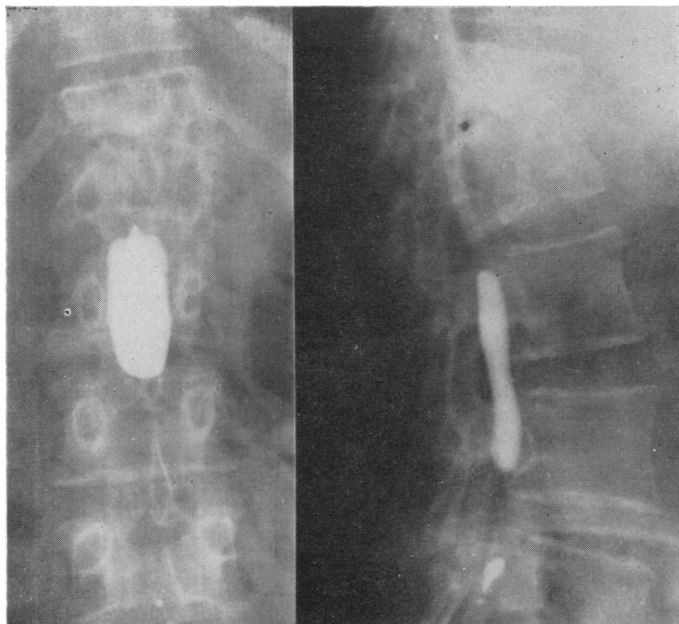


Fig. 12. Myelogram showing a block below the actual bony lesion.

ruins are responsible. In short, as the abscess disappears so does the cord recover. To illustrate the kind of pressure we are discussing consider Figure 12, in which a myelogram shows a block below the lesion; there was a Stage 3 paraplegia which passed after three weeks of drugs without surgery. On later films, when the paraplegia was cured by drugs, the contrast medium had passed beyond the level of the diseased vertebra (Fig. 13).

In the few patients with “paraplegia of late onset” the “internal gibbus” might well play a part, but even then paraplegia can be due to reactivation of disease or pressure from inspissated pus. In these patients the damage to the cord is often irreversible and the results of antero-lateral decompression (Griffiths *et al.*, 1956) in my hands in these patients

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have been poor. The situation I fear most is the "ghost abscess", a shadow without removable substance, a puzzling phenomenon we sometimes see. I suspect that in these patients there is subluxation and they do as badly as cases of spinal trauma.

Any explanation of the true detailed mechanisms of tuberculous paraplegia as we now see it remains, fortunately, conjecture. In our series of 207 patients there have been three deaths, all patients with a Stage 4 paraplegia. With antituberculous drugs and without major surgical procedures the early-onset cases survive and no post-mortem studies are

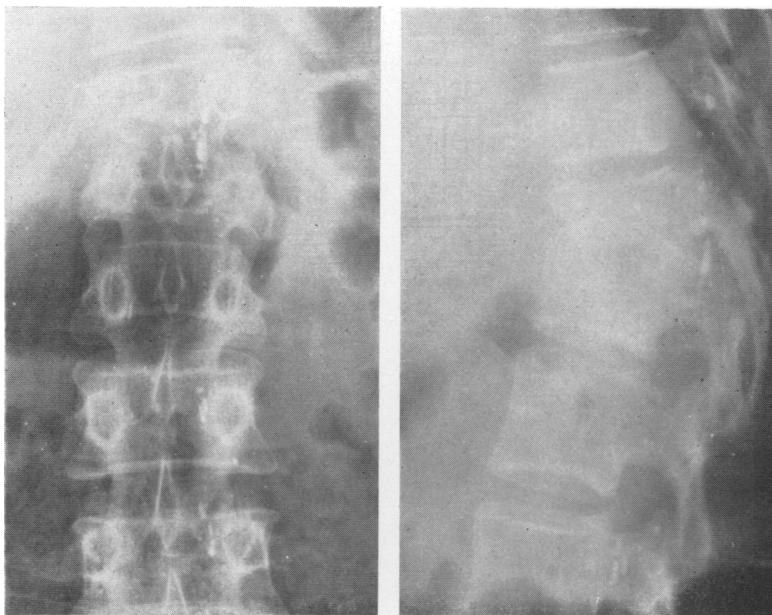


Fig. 13. Contrast medium now well *above* level of bony lesion.

available. We have to rely on accounts given before the introduction of the drugs for a description of post-mortem appearances. In Weedon Butler's series (1935) the mortality of early active disease was 30 per cent. It was nil in our series. Weedon Butler, from his autopsy material, ascribed the paraplegia in all instances to "much toxic and vascular reaction in the cord", and he considered compression by granulation tissue or pus only an additional factor. The greatest credit must be given to his comprehensive and admirable work for which he was in those early days, along with Professor Seddon, awarded the Robert Jones Medal and Prize. The fact that we cannot compare his findings with our material is a tribute to the power of the drugs and to those who created them: the lethal stages

of cord pathology described by Weedon Butler are not reached to-day because the cords recover.

From the foregoing it is obvious what our policy as regards early-onset paraplegia must be. Scarce bed accommodation, operating time and the enormous demands from the sick from near and far demand the simplest and shortest procedures. This we have found in the operation of costectomy. The posterior segment of one or two ribs corresponding to the radiological summit of the abscess which is also the summit of the gibbus, where such a one has developed, is resected, the pleura is pushed laterally, the elastic perispinal abscess is felt and aspirated, the needle is left in place and a knife blade is passed alongside it opening the cavity widely. The contents are evacuated and the wound is closed. The removal of a transverse process is unnecessary. Such patients are given streptomycin in addition to P.A.S. and I.N.H. while in hospital. The physiotherapists go to them at once, the aim being to make the paralysed patient fit to attend the ambulatory outpatient clinic as soon as his legs can carry him somehow. There simple drugs are administered for at least a year.

Mr. Chairman, Ladies and Gentlemen, I have given you to-day an account of the problems of spinal tuberculosis as we encounter it. The methods I have outlined rather dogmatically are not necessary in Europe and America and they may well not work in Asia, although in South India there is now evidence that human tuberculosis is caused by bacilli of lesser virulence than those in Britain (*Lancet*, 1960). Our experience is in accord with the findings of the great Madras experiment, where patients with lung disease were treated with P.A.S. and I.N.H. and where it was found that this is as effective in malnourished Indian patients treated in their poor, overcrowded homes as it was in similar patients treated in good hospitals (*Lancet*, 1961). Similarly, radical surgical attack as advocated in many centres to-day is not feasible with us in Nigeria. It is just possible that we may have made a virtue out of necessity here. By force of social circumstance we had to be, like Sir Reginald Watson Jones, "Surgeons destined to the practice of Medicine" and have been forced to become, to quote John Hunter, "civilized men who get by stratagem what an armed savage would get by force". We are not denying that such force can lead to excellent results; but we have found that, under the conditions prevailing in tropical Africa, it is not often required.

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**HONOURS CONFERRED ON FELLOWS AND MEMBERS**

IN THE RECENT New Year Honours List, the following Fellows and Members were graciously honoured by Her Majesty The Queen:

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**RESEARCH DEFENCE SOCIETY**

WE HAVE BEEN asked to bring to the notice of the readers of the *Annals* the following extracts from a letter received by the President of the College from Sir John Boyd, Chairman of Council of the Research Defence Society.

" You are no doubt aware that it is proposed to set up a Committee of Enquiry into the working of the 1876 Cruelty to Animals Act. This is of much interest to the Research Defence Society . . . and we have decided to set up a small *ad hoc* committee to protect the interest of research workers. We propose, first of all, to draft our own amendments to the Act, which undoubtedly has its faults, and, secondly, to prepare evidence to submit to the Committee when we are asked to do so.

" Although the College makes a generous donation to the fund of this Society each year, there are many Fellows who do experimental work and who are not members. Our case would be very much strengthened if we had a larger membership than we have at present, and I wonder if you could bring to the notice of Fellows who carry out animal experiments that it would be in their interests to join the Society. The subscription is nominal—10s. a year."

The address of the Research Defence Society is 11 Chandos Street, Cavendish Square, London, W.1.