Introduction

While tuberculosis remains a major killer particularly in developing countries, in the last 2 decades in developed countries it had been thought that tuberculosis of spine was a curable medical disease without sequelae. About 95% of tuberculosis patients are in the developing world; China has 1.4 million new cases every year. Tuberculous spondylitis, although less common, is the most dangerous form of skeletal tuberculosis, and the World Health Organization estimates that there are 1.81 million deaths from tuberculosis in Asia each year. The US center for disease control (CDC) predicted that the number of new diagnoses of active tuberculosis worldwide will increase from 7.5 to 11.8 million per year. The incidence of the disease will rise from 143 to 173 per 100,000 and deaths due to tuberculosis will climb from 2.5 to 3.5 millions or more per year.

HIV/AIDS

In 2006 there were 42 million HIV-infected, and yearly deaths from AIDS of 25 millions. HIV disables and destroys the thymic lymphocytes and tissue macrophages that are the body's main defense against tuberculosis making those who are HIV-positive extremely susceptible to the disease. The risk of acquiring an opportunistic infection is proportional to the extent and duration of immunosuppression and tuberculosis is the most common and most virulent opportunistic infection associated with HIV disease. In some African countries, the number of reported tuberculosis cases has doubled or even
triplled from 2001 to 2003 because of the spread of HIV/AIDS. Indeed tuberculosis coupled with HIV disease is “the cursed duet”.

Tuberculosis behaves differently in HIV patients; in HIV-negative patients, only 3–5% of tuberculosis is skeletal but in HIV-positive patients about 60% of the cases involve bone.

Concomitantly there has also been a significant increase in the incidence of disseminated Mycobacterium avium-intracellulare complex (MAIC) infection, which occurs almost exclusively in patients with a more severely decreased CD4-lymphocyte count below 100 cells/mm$^3$ (50–200 cells/mm$^3$) which, taken with the recent resurgence of chemoresistant Mycobacterium tuberculosis variants, has raised concerns worldwide and suggests the need to prepare for the re-emergence of spinal tuberculosis. This has not occurred so far as there are very potent chemotherapeutic agents effective in HIV-positive patients and the surgical techniques to manage, aided by the fact that tuberculosis infectivity in HIV-positive patients is less than that of the HIV-negative patients (although Mantoux skin test positivity rate in diagnosis is lower in HIV-positive patients than that of the HIV-negative patients) and the surgical techniques to manage, aided by the fact that tuberculosis infectivity in HIV-positive patients is less than that of the HIV-negative patients (although Mantoux skin test positivity rate in diagnosis is lower in HIV positive patients than that of the HIV-negative patients).

Management of spinal tuberculosis

Management of spinal tuberculosis has changed significantly in the second half of the 20th century. Even as late as 1970s the primary goal of the management was to save the patient’s life by curing the disease rather than prevention and/or correction of spinal deformity and there were no surgical techniques to treat what were seen as patients’ aesthetic demands which became more marked since the early 1980s.

Antituberculous drugs changed everything. Specific chemotherapeutic agents alone could cure not only active tuberculosis, but also helped in the recovery from paralysis. Antituberculous drugs made surgery safer but operative treatment was reserved for:

- failure of drug therapy;
- recrudescence of the disease;
- Pott’s paraplegia that did not resolve after 4–6 weeks of chemotherapy;
- involvement of the spinal cord;
- other complications.

However, there remain a significant number of issues in the management of spinal tuberculosis that up to now have not received much attention in spite of their significant clinical importance.

This paper seeks to address these issues in diagnosis and management in particular

- newer diagnostic techniques including non-culture laboratory methods;
- new chemotherapeutic protocols with regard to the latent tuberculosis;
- hyperbaric oxygen therapy;
- surgical options for the spinal deformity and associated neural involvement;
- instrumentation;
- implant removal time;
- the fate of the instrument-immobilized joints.

Staging of the disease

Disease staging must be of practical use in management. There is no universally accepted staging system. Kumar’s did not satisfy clinicians’ requirements. Thus terms such as early and late, early and advanced, and kyphotic and non-kyphotic are used in staging the disease, as is vertebral body loss expressed as a percentage and severity of the deformity (Table 1). A satisfactory system is still awaited.

Diagnostic issues

The insidious onset, the lack of early constitutional symptoms and local signs of spinal tuberculosis militate against early diagnosis and there is no single diagnostic test which can detect all cases of tuberculosis. With the increasing incidence of tuberculosis of all types throughout the world, physicians and surgeons must exercise a high index of suspicion to achieve early diagnosis. Thus clinicians typically rely on a battery of methods and diagnosis can take from several days to many weeks and involve expensive, invasive and complex procedures.

The diagnostic techniques include observation and investigation of clinical signs and symptoms, the use of various imaging techniques, and laboratory methods.

**Table 1** Staging of spinal tuberculosis (Moon, 1992).

<table>
<thead>
<tr>
<th>Disease progress</th>
<th>Size of lesion</th>
<th>Deformity</th>
</tr>
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<tbody>
<tr>
<td>Disease advancement (stages)</td>
<td>Degree of body destructive loss</td>
<td>Degree of kyphosis</td>
</tr>
<tr>
<td>Stage I: minimally (very early)</td>
<td>&lt;20% (2/10)</td>
<td>12.2°</td>
</tr>
<tr>
<td>Stage II: early</td>
<td>26–50%</td>
<td>12.2–22.25°</td>
</tr>
<tr>
<td>Stage III: relatively (moderately)</td>
<td>51–100%</td>
<td>22.26–39.0°</td>
</tr>
<tr>
<td>Stage IV: far advanced</td>
<td>101–200%</td>
<td>39.1–72.5°</td>
</tr>
</tbody>
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Note: percentage and degree of kyphosis in cases of conservative treatment is calculated by Rajasekaran’s formula ($Y = 3.5 \times 30.5 \times \text{degree of body destruction}$).
individuals are negative to TST throughout life, despite repeated exposure to the tubercle bacilli. Additionally the sensitivity decreases in immuno-compromised patients for whom accurate diagnosis of latent tuberculosis infection is essential. In terms of specificity, TST is influenced by BCG vaccination and non-tuberculous mycobacterial infection.

**Imaging**

Imaging techniques such as simple radiographs, bone scan, CT and MRI are useful but not diagnostic; e.g. when disc and/or end-plate destruction with surrounding soft tissue swelling is observed on simple radiographs spine infection should be suspected.

Diffusion-weighted MRI has been found to have limited usefulness for differentiating spinal infection and malignancy.

**Laboratory**

Laboratory aids to diagnosis include:

- complete blood counts including total lymphocyte and CD4 lymphocyte (helper-inducer T-cell) counts, ESR and CRP;
- smear and/or culture;
- histology;
- detection of specific antigen;
- metabolic products;
- patient’s antibody response and detection of antibody to *M. tuberculosis*;
- DNA sequence polymerase-chain reaction (PCR) of *M. tuberculosis*.

The diagnostic gold standard has traditionally been the isolation of *M. tuberculosis* by smear and/or culture from clinical samples e.g. aspirates and tissue specimens, and the typical histology. However, the tubercle bacilli is difficult to culture due to its fastidious growth requirements and slow rate of growth. Hence the need for the development of various laboratory methods.

There are three diagnostic non-culture laboratory tests:

- immunological tests; antigen and antibody;
- metabolic product detection such as extracorporeal interferon-\(\gamma\) test, and
- amplification of DNA of *M. tuberculosis* by PCR.

Lymphocytes secrete interferon-\(\gamma\) when T-lymphocytes are exposed to tubercle bacilli making extracorporeal interferon-\(\gamma\) test for latent tuberculosis particularly noteworthy. Two ex-vivo interferon-\(\gamma\) (IFN-\(\gamma\)) assay kits have been marketed recently; Quanti-FERON® TB Gold [Cellestis limited, Carney gie Victoria, Australia, QFT-G] and T-SPOT, TB [Oxford Immunotec, Oxford, UK, T-SPOT].

The former uses the ELISA technique and T-SPOT uses enzyme-linked immunosorbenet spot (ELISPOT) technique. Both use *M. tuberculosis*-specific antigens, ESAT-6 and CFP-10 as stimulants. As BCG does not contain ESAT-6 and CFP-10, these assays are more specific than TST in the diagnosis of tuberculosis infection. They have variable sensitivity, dependent on host factors and methods used. There are reports that T-SPOT is more sensitive than TST in immuno-compromised subjects.

These assays detect tuberculosis infection, but they cannot differentiate between latent tuberculosis infection and active tuberculosis. Therefore, when the TST and IFN-\(\gamma\) assay results are positive, differentiation of latent infection and active tuberculosis should be carried out on the basis of the clinical history, chest and skeletal imaging studies and tissue studies.

The other major non-culture test is the molecular diagnostic test, PCR, which amplifies the DNA of *M. tuberculosis*. A primer pair targeting a 123 base pair (bp) segment of the repetitive sequence IS6110 of the *M. tuberculosis* complex is used, which covers *M. tuberculosis*, *M. africanum*, *M. bovis*, *M. canetti* and *M. microti*.

This technique represents an exciting development. It has been used as a marker to monitor response to treatment and has been shown to provide rapid information on drug resistance and clonality in epidemiological investigation of outbreaks.

**Histology**

The histological lesions caused by *M. tuberculosis* in AIDS and anti-TNF-\(\alpha\) treatment patients range from the classical caseating granulomas, to more frequently a non-specific chronic inflammatory reaction without necrosis. Thus it is important to use acid-fast staining in non-specific inflammatory lesions to rule out *M. tuberculosis* infection. PCR techniques utilizing microdissection of the tissue specimen is another diagnostic option and is one of the most advanced diagnostic techniques.

To summarize, despite recent advances, there is yet no 100% accurate diagnostic technique. The author’s preference, in cases of diagnostic delay, is for image-guided needle bone biopsy. This is a simple and safe procedure allowing bacteriological and histological assessment of aspirates and bone specimens.

**Management**

**Chemotherapy**

Chemotherapy is the mainstay of treatment of spinal tuberculosis. There are six main anti-tuberculous drugs (see below). With the increasing incidence of drug-resistant tuberculosis worldwide, it is very important to know the bacterial sensitivities before commencing chemotherapy. By culture of aspirate or tissue specimens, sensitivity tests of the cultured tubercle bacilli against each drug can be ascertained. Non-culture laboratory techniques are also utilized.

**Pyrazinamide**

Pyrazinamidase activity is assessed by either

- rapid detection of mutation in Pnc A gene by PCR-CSCP in one to 2 days, or
- flowcytometric array with BACTEC TB 460 or BACTEC MGIT 960. Data are available 2 h after the initiation of testing procedures.
Streptomycin
Resistance is assessed by detection of 16S-RNA with ribosomal S12 protein and mutation in regions surrounding the nucleotides 530–912 in 16s-RNA, or by PCR, gyrase A (Gyr A) and ribosomal protein SR (rPSL) mutation.

Isoniazid (INH)
As this drug is a prodrug, being active only in the presence of an enzyme, Kat G gene mutation is studied.

Rifampicin, ethambutol and ciprofloxacin
The localized mutation in a RNA polymerase (rPOB) gene for rifampicin, mutation in embB codon 306 for ethambutol, and gyrase A and rPSL mutation for ciprofloxacin are shown.

While the ideal antituberculous chemotherapeutic regimen is based on drug sensitivity of cultured \( M. \) \textit{tuberculosis}, as already discussed, it is not always possible clinically to discover \( M. \) \textit{tuberculosis} by the direct smear and/or on tissue slide. Furthermore, it is difficult to culture tuberculosis bacilli successfully from abscess and tissue aspirate at all. Hence the development of non-culture diagnostic and drug sensitivity techniques.

To date, chemotherapy with currently available agents remains highly effective in extrapulmonary tuberculosis in children and adults, even in patients with concomitant HIV infection (Table 2), but, as with all chemotherapy, compliance with therapy is of the utmost importance. Measures, including directly observed therapy (DOT) to maximize compliance are essential as inadequate treatment and/or patient non-compliance are the most common causes of drug-resistant strains.

Controversial issues regarding chemotherapy are:

- drug combination formulae;
- duration of drug administration in surgical and nonsurgical management;
- when to reassess drug efficacy i.e. slow, inadequate and non-responder, and
- when to change the initial chemotherapy.

Because patients differ and each has varying disease severity, and is infected with different bacterial strains,
they respond differently to treatment. Thus a standard “accepted regimen” is not applicable to every patient. Table 3 sets out the regimens adopted as standard by most physicians.

It appears that short course chemotherapy (9 months) and intermittent chemotherapy for non-skeletal tuberculosis is as effective as old long course regimens of 12–18 months (Table 3). However, chemotherapy should be changed for non-responders i.e. in cases of serious clinical deterioration, notably the onset of paraparesis, untoward change in existing paraparesis or clear-cut radiographic extension of the spinal disease. In such cases misdiagnosis, drug resistance or improper combination of drugs has to be considered. For non-responders, initially the dose is increased for 3–4 weeks. If this fails, therapy should be changed from a 3 drug to 4–5 drug regimen.

Multi-drug resistant tuberculosis is defined on the basis of resistance to both INH and rifampicin. As the management for resistant tuberculosis is very complex, the best available expertise should be sought. However, it is best never to add a single drug to failing regimen, because it creates an ideal condition for the development of resistance to the new medication.

The WHO warned in 2004 that the super-strain tuberculosis bacilli which had emerged in Eastern Europe and middle Asia had 10 times higher rates of drug resistance compared to other areas. Around 80% of these “super strains” are resistant to 3–4 drugs. Hence consistent use of 2 or more effective agents at the time of initial chemotherapy is therefore essential. Drug resistance usually reflects the failure to choose an appropriate regimen and dosage or to ensure adherence to therapy (Table 4).

Thus treatment should be initiated with at least two effective drugs to which the patient has never previously been exposed. For those who had received incomplete treatment with multiple drugs in the past, initial therapy with as many as 9 drugs has been used until sensitivity results became available. Surgery has a role in the treatment of multidrug resistant tuberculosis if the infection is sufficiently localized to be amenable to resection.

Figure 1  (A) Preoperative radiographs of L2-3 tuberculosis in a 38-year-old male showing markedly narrowed disc space with mild left lateral tilt of L2 body on L3. (B) Post-anterior fusion of L2-3 showing iliac bone graft and incomplete correction of the tilted L2. (C) Nine months post-operative radiographs showing re-tilting of L2 body over L3 despite the good consolidation of fused L2-3 bodies. (D) Twelve months post-operative radiographs showing further consolidation of the fused L2-3 bodies, but no further tilting of L2. This is an example of under-correction of the scoliotic spine at the time of anterior fusion. Some over-correction with bone graft for tuberculous scoliosis is preferably recommended to prevent retitting.
The author’s experience is that chemotherapy cannot be shortened by anterior radical surgery.\textsuperscript{10,15}

For latent tuberculosis, combined anti-TNF-\(\alpha\) therapy, hyperbaric oxygen and vasodilator drug therapy (PGE\(_2\): Opalmon) are recommended by the author to awake the dormant bacilli, because dormant bacilli do not respond even to sensitive drugs\textsuperscript{10,15} (Fig. 1), (pyrazinamide has been reported to kill a population of semidormant tubercle bacilli not affected by other antituberculous drugs).

**Concomitant rheumatoid arthritis**

It must not be forgotten that immune suppressors e.g. steroids and TNF-\(\alpha\) blocker ([TNF-\(\alpha\) inhibitors] etanercept (infliximab, adalimumab)) can activate latent tuberculosis by inhibiting lymphocytic and macrophage activity. Thus for rheumatoid arthritis and related disease patients with latent tuberculous infection, anti-TNF-\(\alpha\) treatment should be started at least after 3 weeks of anti-tuberculous chemotherapy, and for rheumatoid patients with active tuberculosis TNF-\(\alpha\) agent therapy should be started at least after 2 months of anti-tuberculous chemotherapy (Fig. 1).

As most cases of tuberculosis after biologic therapy represent reactivation rather than new onset disease, screening before TNF-\(\alpha\) blocker therapy is critical to prevent such relapse. However during anti-TNF-\(\alpha\) therapy, post-operative rigor or fever other bacterial infection must be excluded.\textsuperscript{14,16}

**Extra- or intra-dural extra-medullary or intra-medullary tuberculoma**

There has been debate as to whether cord tuberculoma is a medical or surgical condition, because of the grave sequelae of surgical removal. The author recommends combined medical treatment after diagnostic biopsy; anti-tuberculous chemotherapy and anti-TNF-\(\alpha\) blocker treatment (subcutaneous injection of etanercept, 25 mg twice weekly up to 6–8 weeks) that has degranulating effect. This can obviate the need for total excisional surgery for tuberculoma.

**HIV and spinal tuberculosis**

Two key points to remember are that the chemotherapeutic effect is the same for HIV positive and negative patients nor does host immuno-competence influence the neural outcome in Pott’s paralytic patients. However, HIV infected patients appear to be more prone to adverse reactions to antituberculosis agents than HIV negative individuals.\textsuperscript{15}

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**Figure 2** (A) Preoperative radiographs of active tuberculosis of L2–3 in a 32 year old female, managed with anterior radical surgery and triple chemotherapy. (B) Immediate postoperative radiographs showing regained disc height with two iliac bone grafts and restored lumbar lordosis. (C) Two month post-operative radiographs showing the re-collapsed disc space and absorbed bone graft. (D) Four months post-operatively with further corporeal destruction with graft collapse. (E) Radiographs 2 years and 10 months post-operatively showing solid fusion of L2–3 with mild kyphosis.
The new HIV protease inhibitors have great potential for HIV patients with spinal tuberculosis. The author’s view is that primary prophylactic use of INH (300 mg/day for 12 months) for the HIV patients is more cost-effective in preventing tuberculosis recurrence than triple chemotherapy after tuberculosis infection. HIV positive patients with tuberculosis must be treated initially with at least three agents, the ultimate regimen being chosen according to culture and sensitivity testing. However, long-term suppressive therapy is not recommended.

Other drug therapies

The use of the immunopotentiators such as oral levamisole and vaccines (BCG and DPT) is recommended as is combined use of NSAIDs with antituberculous agents, despite their gastrointestinal side effects because they are effective in alleviation of back pain and might prevent bony destruction attributable to non-specific synovial membrane inflammation and inhibit or minimize the bone resorption by prostaglandin E2 in the early course of disease (4–6 weeks).

Surgery

Chemotherapy alone cannot treat bone and joint destruction. Thus despite effective conservative treatment, surgery still has an important role in the management of spinal tuberculosis, but it cannot solve every problem!

Surgery can have a place in the management of cold abscess, paraplegia and spinal deformity. In most cases, small and even medium-size cold abscesses rapidly resolve spontaneously with chemotherapy alone. In the author’s series, evacuation of abscesses did not alter patients’ general condition, and very often resulted in a persistent draining sinus. Routine drainage even for large abscesses is not recommended.

A further factor to be considered is the patient’s aesthetic demand regarding spinal deformity that surgeons have to

Figure 3  (A) Preoperative radiographs of tuberculosis of T12-L-1 in a 2-year-old girl showing destruction of disc and vertebral bodies of T12 and L1 with relatively good bony contact. (B) Immediate postoperative radiographs showing the interspinous fixation wire between T12-L1. (C) Radiographs at 25 weeks post-operatively showing no further body destruction. (D) Radiographs at 1 year and 6 months post-operatively showing the consolidated T12-L1.
take into account. Hence the need to prevent or minimize spinal deformity.

Hyperalimentation may be necessary to restore patients from a preoperatively physically debilitated state to their pre-morbid nutritional status. The goal should be to achieve a serum albumin level > 3g/dl, an absolute lymphocyte count > 800/mm$^3$, and a 24-h urine creatinine excretion > 10.5 mg in men and > 5.8 mg in women.

**Predicting the final kyphotic angle**

A formula to predict the final kyphotic angle in the ambulant chemotherapy group, proposed by Rajasekaran and Shanmugasundaram in 1987 has been widely used.\(^3\) Accuracy has been reported to be as high as 90%, but Jain et al.\(^{36-38}\) in 2004 reported 77.64% accuracy. The author’s view is that the discrepancy was due to inaccurate estimation of initial vertebral body loss.\(^3\) Because accurate estimation of the pre-treatment vertebral body loss is impossible discrepancy of the final kyphosis reaches 6.1° (30.5 x 0.2). Additionally the formula is only applicable to adult patients.

**Spinal fusion**

Hong Kong surgeons stress that surgical fusion is essential to heal spinal tuberculosis, and if fusion has not been achieved, some of those cases are untreatable.\(^9,10,33,34,39\) My view is that bony and mixed replacement of the intervertebral space are not always synonymous with clinical healing, and that anterior radical surgery in adults to obtain cure through intercorporeal fusion is not justified in every case, because spontaneous fusion can be obtained by modern chemotherapy alone, and that cure can be also obtained without fusion.\(^6,8,21,27,30,39\) Fusion is desirable, but is not essential for every patient.\(^22,35\) In particular ‘block spine’ after anterior fusion in children is not desirable, as it accelerates the tuberculous kyphosis during growth.\(^40,41\)

It has become clear that the only advantage of anterior radical surgery is a degree of deformity correction, a reduced tendency for deformity progression and stabilization of the diseased unstable segment. The author’s experience suggests that because of graft failure the procedure was not always successful in preventing kyphosis progression and/or correcting pre-existing kyphosis (Figs. 1 and 2) and that it is unwise to rely solely on the anterior radical surgery to correct the spinal deformity and to maintain correction. Because of this, the author uses posterior instrumentation surgery in addition to the anterior surgery.\(^9,10,23,25\)

Huang et al. reported that video-assisted thoracoscopic surgery for tuberculosis of the dorsal spine was effective and safe.\(^42\) The current author’s view is that the procedure has significant practical limitations (Figs. 3 and 4).

**Stabilizing instrumentation surgery**

Timing of surgery has been an issue. Patients’ demands differ, even with the same severity of the disease at the same level at presentation. Some are more concerned about spinal deformity than others who choose the conservative treatment despite being well informed regarding disease prognosis.
anterior graft, prevented graft collapse, and speeded graft incorporation.\textsuperscript{9,23}

They utilized the British Medical Research Council (MRC) grading (mild, moderate and severe) of dorsolumbar kyphosis. Since 1990, they have recommended posterior instrumentation, using Rajasekaran and Shanmugasundaram’s residual kyphosis predicting formula\textsuperscript{37} e.g. the patient with >70\% destructive vertebral body loss in the dorsolumbar spine will heal with >30° residual kyphosis. According to Rajasekaran a vertebral body loss of 0.75 in the dorsal and dorsolumbar region and 1.0 in the lumbar region is an indication for surgery.\textsuperscript{37,43} Between 1997 and 2002, Moon\textsuperscript{27} recommended posterior instrumentation surgery to restore the sagittal alignment when there was vertebral body loss >40\% and final residual kyphosis >20°. By doing so the author attempted to prevent development of the compensatory lumbar hyperlordosis and adjacent segment disease in cases of the conservative treatment. More recently, he has aimed to surgically restore the normal spinal alignment.

The types of vertebral body collapse and development of kyphosis in the tuberculous spine differ at the various levels of the spine; cervical, dorsal, dorsolumbar and lumbar. In the dorsal and dorsolumbar spines the pattern of vertebral collapse is kyphotic, whereas that in cervical and lumbar spines is rather vertical (telescoping)\textsuperscript{27,43} It follows that the indications for posterior instrumentation in the cervical and lumbar spines are different from the dorsal and dorsolumbar spines.

On theoretical grounds posterior instrumentation surgery is recommended for very early disease, but latterly patients’ high aesthetic demands have tended to drive decision making rather than pure surgical indications. Sadly surgeons are gradually becoming cosmetic surgeons of the musculoskeletal system. Informed consent is essential!

Surgery for dorsolumbar tuberculosis with non-rigid kyphosis

Jain et al.\textsuperscript{38} recommended operative stabilization of the kyphosis in the active stage of the disease when the patients had an initial vertebral body loss of two without giving a

\begin{figure}[h]
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\caption{(A,B) A 6-year-old boy with tuberculosis of T_{12}-L_{2}. (C) Lateral radiograph immediately after posterior segmental stabilization with two Rush nails and segmental wiring showing some correction of kyphosis. (D) Radiograph after anterior interbody fusion. (E) One year 9 month post-operative radiograph showing the fused block spine (T_{12}-L_{2}). (F) Two years 3 months after removal of the instrument showing the fused block.}
\end{figure}
The author’s view is that their recommendation was related to the late development of neurological compromise, rather than directly to the kyphosis. He does not agree, particularly in relation to aesthetic and neurological problems.

Posterior stabilizing/corrective spinal surgery is most effective in the management of the active progressive kyphosis and established residual non-rigid kyphosis (Figs. 5–8), but kyphosis correction when there is the destructive body loss, will produce a bony gap in the anterior column. This gap should be reconstructed by an anterior strut graft,23,25 otherwise the posterior instrumentation will ultimately fail secondary to anterior column re-collapse.

Corrective spine surgery for severe rigid kyphosis

Non-paralytic young patients tend to have very high aesthetic demands.23,25 This has led to the development of safe and effective corrective surgical procedures for the unsightly kyphosis,38,40,44,45 but each patient must be considered individually.

In healed severe tuberculous kyphosis at cord level, the bony anatomy is severely deformed and the cord in this area is ischemic, deformed and less mobile. It is often trapped in the deformed, narrow, hypoplastic canal and is adherent to the dura.29,36 There are several possible surgical procedures, ranging from single-stage to multistage procedures for rigid tuberculous kyphosis (Figs. 9–11)23,25,45,46 including posterior closing wedge osteotomy, decancellation procedure, and hemi- or total spondylectomy as a single stage operative procedure.15

While correction is possible, surgery is high risk, technically challenging and dangerous with high complication rates. Any decision to operate must weigh the high complication and re-operation rates against anticipated improvement. Therefore, an informed consent discussion should include the patient and his or her support group. Risks and complications must be fully understood, and the patient will ultimately choose between those risks and his/her quality of life. The surgeon should offer options and probability, but it is the patient who must bear the consequences of that decision.

Spinal column shortening

The ideal size of the longitudinal spondylectomy—en-bloc hemi- or total spondylectomy—in the correction of the
spinal deformity in relation with the cord function has rarely been discussed, and there is no consensus view. In the normal spine, cord length, spinal canal and anterior spinal column length are equal, while in the idiopathic adolescent scoliosis the anterior column length is longer than the canal length. Therefore, if the scoliotic spinal column is corrected by longitudinal distraction alone, the cord will be stretched, which may result in paralysis. When en-bloc total spondylectomy is performed for a spine tumour, the preoperative relationship of the cord and spinal column length should be maintained. Sometimes the spinal column can be lengthened or shortened at the time of anterior column reconstruction and instrumented stabilization; in the former the cord will be stretched and in the latter the cord will be axially compressed which results in cord ischemia leading to paralysis.

Kawahara et al.\(^47\) reported that the dural kinking was not observed in any patients undergoing a column shortening procedure; 22.2% (7 mm ± 4–10 mm) of the corpectomy defect in the 40 patients. In none was neurological degradation observed postoperatively. However, 2 of the 3 preoperative Frankel B paraparetics did not recover, while one recovered to Frankel E. Seven out of 13 preoperative Frankel C paraparetics improved to E, 5 improved to D, and in one there was no improvement. Six Frankel D paraparetics improved to E in all. They concluded that 20% column shortening in spinal tumours might be safe.

Kobayashi et al.\(^24\) reported the results in dogs of longitudinal column shortening surgery. Up to 7.2 mm shortening produced no morphological change in the dura mater or spinal cord. If the shortening was between 7.2 and 12.5 mm the cord remained straight in spite of ’ruffling’ of the dura mater. Above 12.5 mm of shortening, the cord became kinked. Abnormal evoked potentials were recorded with column shortening of 15 and 20 mm, causing incomplete paralysis of the hind limbs in 1 of 3 and 3 of 4 dogs, respectively. With column shortening of 5, 10, 15 and 20 mm cord blood flow changed to 146%, 160%, 102% and 93% of the preoperative blood flow, respectively. Column shortening less than 15 mm significantly increased the diameter of the anterior spinal artery.

Figure 7 (A, B) Preoperative radiographs of a 29-year-old female with tuberculosis of T\(_{10–T_{12}}\) and wedge shaped destroyed T\(_{11}\) body and narrowed discs. (C, D) Radiographs after Harrington rodding between T\(_{9–L_{1}}\) and anterior fusion showing the two iliac grafts between T\(_{10–T_{12}}\) and good correction of kyphosis. (E, F) Three month post-operatively radiographs showing good consolidation of the grafts with minimal increase in kyphosis. (G, H) Two years after initial surgery and removal of Harrington rods showing complete consolidation of the grafts.

M.-S. Moon
while shortening over 15 mm decreased the anterior flow. They concluded that the safety limit for column shortening was 12.5 mm (62.5%).

Tanaka et al. reported their results of the cranial half shortening spondylectomy (14–23 mm, mean 20 mm) of the L1 body for adult patients with tethered cord syndrome;
there were symptomatic improvements after spinal column shortening. The procedure was found safe and effective, but they did not discuss spondylectomy size.

**Tuberculous kyphosis in children**

In children spontaneous intercorporeal fusion rarely occurs during chemotherapy. It occurs later after the cure of the tuberculosis at lower rates than in adults. This has not been satisfactorily explained. Study of BMP and anti-BMP factors in the tissue around the tuberculous lesion may clarify the cause. In most children there was a gradual increase of spinal deformity in spite of disease cure, when the growth plates were destroyed in either unfused and fused vertebrae. When the unfused segment was unstable, the deformity progressed more rapidly than that of the fused block vertebrae.

The unsightly hunchback residual kyphosis can be distressing to patients, their parents and surgeons alike. Initially in the early 1970s the author used posterior interspinous wiring and fusion. This was followed by interspinous wiring and cementation to stabilize the involved segment and to arrest the posterior spinal element growth. Both techniques failed because of loosening of wires and cement (Figs. 3 and 4), and they were succeeded by posterior instrumentation (Fig. 5). Posterior instrumentation in the management of the pediatric tuberculous kyphosis can contribute to growth correction of the kyphosis by growth arrest of the posterior column when applied before age 10–11 years. Thus in children, posterior instrumentation is thought to be the best procedure with or without posterolateral fusion, utilizing several tether devices.

**Quadriplegia or paraplegia with or without kyphosis**

There are three main causes of paralysis:

- cord compression by abscess and granulation tissue;
- cord compression by sequestrum and the posterior bony edge of the vertebral body at the level of the kyphosis;
- bony canal stenosis of the deformed spine above the level of the kyphosis.

The management of Pott’s paralysis and mild, moderate or severe kyphosis is still controversial. This author made treating the paralysis the priority, and did not aim to correct the kyphosis simultaneously.

There are two approaches to manage paralysis, conservative medical or surgical. The timing, whether immediate or delayed, for decompression surgery under chemotherapeutic cover is still debated. Slowly progressive paralysis due to abscess and granulation tissue and in the early stage can be effectively managed by chemotherapy alone which cannot only cure the disease but also the neural deficit without surgical decompression regardless of the severity of kyphosis. However, if the paralysis does not improve within...
maximum of 4–6 weeks after commencement of chemotherapy, decompression surgery should be undertaken.

When there is acute onset paraplegia with rapidly worsening neurology by bony cord compression and fibrosis, and chronic cord compression within the narrow canal surgical decompression is indicated. In the author’s experience, posterior instrumentation surgery alone regardless of the disease stage and/or severity of deformity can hasten the neurological recovery without direct decompression surgery by stabilizing the unstable diseased segment (Fig. 12).

**Extent of stabilization and timing of implant removal**

Long rodding immobilizes normally mobile segments and may later cause these segments to stiffen and degenerate (Figs. 5, 6, 8, 11). Therefore the author normally removes the rods after solid intercorporal fusion about 9 months postoperatively to remobilize these instrumented segments and over the last 15 years has carried out short rather than long segment posterior instrumented stabilization before anterior fusion.

In children with unacceptable tuberculous kyphosis, posterior instruments should be left until maturity to maximize gradual growth correction of the deformity due to the tethering effect of the implants.

**Bacterial response to implant and chemotherapy**

Until implants such as titanium cages, screws and rods, P.V.P and ceramic cups in the tuberculous focus were found to be harmless, there was concern that the use of such materials would hinder the healing of the tuberculosis, or cause arrested or quiescent infection to flare up. This has been shown not to be a concern.

*M. tuberculosis* has biologically very specific behavioral characteristics which differ from pyogenic bacteria. A good understanding of these characteristics is vital as is knowledge of bacterial response to implanted material and chemotherapy. *M. tuberculosis* lesions represent a planktonic form which reproduces slowly, producing minimal adhesion molecules and slime, and occasionally becomes dormant. Thus, *M. tuberculosis* has little tendency to adhere to implants that can be safely used in tuberculous lesions, but they do form a nidus to perpetuate tuberculosis infection. However,
HIV positive patients, the use of biomaterials in the infected foci is inappropriate, because they are susceptible to a variety of other opportunistic infections. Additionally, bacteria adhere less to titanium than stainless steel, because titanium has a less electrochemically active oxide surface compared to stainless steel, which has a higher surface free energy ($>40 \text{ mN/m}$) and hence more available ligands for binding with cells both bacterial and tissue competing for the biomaterial surface. It follows that stainless steel implants should not be used for spinal stabilization.

Conclusions

Spinal tuberculosis is treatable with the presently available therapeutic agents and surgical techniques. Chemotherapeutic agents are the mainstay of curative treatment with surgery supplementary, i.e., spinal tuberculosis is essentially a medical condition. Surgery has a place in those patients with structural and/or neurological complications.

References


