Essential ORTHOPAEDICS
(Including Clinical Methods)

MAHESHWARI & MHASKAR

Promoted by
Knee & Shoulder Clinic
New Delhi
Essential Orthopaedics
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(Including Clinical Methods)

SIXTH EDITION

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Dedicated to

My patients
for giving me an opportunity to make a difference in their lives

and

My family & friends
for continuous support at all times
FOREWORD TO THE FIRST EDITION

This book written by Dr Maheshwari, is designed to introduce the trainee doctor and the young surgeon to orthopaedic surgery as he will meet it in the developing countries. There have been many good books on orthopaedic surgery and trauma written by experienced authors from Britain and America but their exposure to the real orthopaedic problems experienced in developing countries has often been limited, and today the difference in presentation of orthopaedic surgery in these countries and the presentation of conditions in developing countries like India and Africa is so different that this book, written by a surgeon with a good grounding of clinical experience in India, is most appropriate for the trainee from the developing countries.

I have read a number of chapters and have been impressed with the simple text and clarity with which the different conditions are explained.

Dr Maheshwari has visited my centre in Nottingham for a period of two months. I was impressed with his clarity of thought and his depth of understanding of orthopaedic conditions. I anticipate that this book will be one of many that he writes in future years and is likely to be a major contribution to orthopaedic training in developing countries.

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Preface to the Sixth Edition

It is always a tough call to update a basic book such as this. Challenge is, not cross the boundary of ‘what is relevant’, keeping in mind the audience the book is catering to. The book has been screened to ensure that not a single word is more than what is required at a UG level. The basic format of the book has been kept as it is—easy readability and well-illustrated text.

We did a survey and asked some bright undergraduate students as to what they would like added to this book. Based on their feedback, we have added a chapter on imaging in orthopaedics and updated information on currently relevant topics of joint replacement and arthroscopic surgery.

All chapters have been reviewed, and changes in photographs made wherever required. A part of the text is elaborated.

Hope the book will continue to enjoy the popularity amongst students.

J Maheshwari
Vikram A Mhaskar
What was the thought behind this book in 1993

As an undergraduate, though exposed to orthopaedics only for a short period, I was impressed by the ease with which I could understand the wonderful texts I studied. The problems were that their contents did not exactly meet the requirements of an undergraduate, and most of these books, written by authors from developed countries, did not provide adequate information about diseases peculiar to tropical and underdeveloped countries. Above all, I thought that the concepts could be presented with still more clarity, and improved by way of presentation.

This feeling continued to haunt me everytime I was called upon to teach undergraduates. A couple of years later, an experience at home helped me give a practical shape to this feeling. My wife, who was preparing for PG entrance examination, expected me to teach her orthopaedics. I tried out my ideas on her, and the result was extremely gratifying. Soon after, many more such occasions of teaching undergraduates gave me further opportunities for refining the material. It was on the request of the students that I decided to give it the shape of a book.

The book is primarily addressed to undergraduates and those preparing for the postgraduate entrance tests. General practitioners, particularly in the early stage of their practice would find it useful reference. It would enable nurses and physiotherapists to understand the basic concepts in orthopaedics. Junior postgraduates would find it an enjoyable reading.

Following are the salient features of the book:

1. Most chapters begin with a brief review of the relevant anatomy. This is because by the time a student comes to clinical departments, he has forgotten most of the anatomy he had learnt in the dissection hall.
2. While discussing treatment of a condition, a brief mention of principles is made first, followed by various methods and their indications. This is followed by treatment plan; a practical plan of treatment which is either being followed or can be developed in an average hospital. A brief mention of recent developments is also made.
3. The book has three additional chapters. These are "Approach to a Patient with Limb Injury", "Approach to a Patient with Back Pain", and "Recent Advances in Treatment of Fractures". The first two present a practical approach to handling these frequently encountered emergencies, and the third chapter updates the reader with the latest in this rapidly developing field. Due emphasis has been given to aspects of rehabilitation, considering the recent recommendations of Medical Council of India for including ‘rehabilitation’ in undergraduate curriculum.
4. Simple line diagrams have been used to supplement the text. Most of them have been developed by myself while teaching the undergraduates. Simplified line diagrams, rather than photographs, enable students understand the basic concepts better.
5. Self-explanatory flow charts are made use of wherever they would help to develop a concept in decision-making.
6. Tables have been used liberally. These serve two purposes: Firstly, they present the text matter in a concentrated form and allow review at a glance. Secondly, they permit quick and easily understandable comparison between related conditions.
7. Necessary information on instruments and implants commonly used in orthopaedics has been provided as an appendix, purely considering the requirement of such knowledge for final professional examination.

New Delhi
September 1993

J Maheshwari
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DEFINITIONS

Arthritis is an inflammation of a joint. It is characterised by pain, swelling and limitation of joint movement. The cause may be purely a local pathology such as pyogenic arthritis, or a more generalised illness such as rheumatoid arthritis.

Arthralgia is a term used for pain in a joint, without any associated signs of inflammation.

CLASSIFICATION

From the clinical viewpoint, arthritis can be divided into two types: (i) monoarthritis; and (ii) polyarthritis. Some common causes of the two types are given in Table–34.1.

Table–34.1: Types of arthritis

<table>
<thead>
<tr>
<th>Monoarthritis</th>
<th>Polyarthritis</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pyogenic arthritis</td>
<td>Rheumatoid arthritis</td>
</tr>
<tr>
<td>Tubercular arthritis</td>
<td>Rheumatic fever</td>
</tr>
<tr>
<td>Haemophilic arthritis</td>
<td>Juvenile chronic polyarthritis</td>
</tr>
<tr>
<td>Secondary osteoarthritis</td>
<td>Primary osteoarthritis</td>
</tr>
<tr>
<td>Gout - sometimes</td>
<td>Seronegative spondarthritis</td>
</tr>
</tbody>
</table>

RHEUMATOID ARTHRITIS

Rheumatoid arthritis is a chronic non-suppurative inflammation of the synovial joints diagnosed as per the criteria laid down by American Rheumatism Association in 1987 (Table–34.2).

AETIOPATHOLOGY

Aetiology: The exact aetiology is not known. Following factors have been thought to play a role in causation of the disease:

Table–34.2: New diagnostic criteria for rheumatoid arthritis (1987)

- Morning stiffness
- Swelling of three or more specified joints
- Swelling of joint(s) in the hands and wrist
- Symmetrical swellings
- Rheumatoid nodule
- Rheumatoid factor positive
- X-ray changes – erosion or unequivocal peri-articular osteopenia
- If four or more of these are present, it is rheumatoid arthritis

Sensitivity of these criteria 93 per cent
Specificity of these criteria 90 per cent

- A genetic predisposition is strongly suspected because of certain histocompatibility markers associated with it (HLA-drw4/HLA-DR1).
- Agents such as mycoplasma, clostridium and some viruses (EB virus) have been implicated in its aetiology.

It is now believed that rheumatoid arthritis results from exposure of a genetically predisposed individual to some infectious agent. This leads to autoimmunity and formation of immune complexes with IgM antibodies in the serum. These immune complexes are deposited in the synovial membrane and initiate a self-perpetuating chronic granulomatous inflammation of the synovial membrane.
Pathology: Initially the synovium becomes oedematous, filled with fibrin exudates and cellular infiltrates. There is an increase in synovial fluid. As the inflammation persists, the synovium gets hypertrophied and surrounds the periphery of the articular cartilage to form a pannus. The articular cartilage loses its smooth shiny appearance. The pannus extends over the cartilage from the periphery and burrows into the subchondral bone. With further progress of the disease, the cartilage becomes worn off and the bone surfaces become raw. The joints get deformed, initially because of severe muscle spasm associated with pain, but later due to fibrosis of the capsule and other soft tissue structures.

In some cases, adhesions develop between apposing layers of pannus, leading to fibrous ankylosis, and later bony ankylosis. In an advanced disease, the joint capsule gets distended by the hypertrophied synovium and synovial fluid, and the ligaments supporting the joint are stretched, resulting in subluxation of the joint. Osteoporosis develops in the bones adjacent to the diseased joint. Peri-articular tissues, notably tendons and muscles become oedematous and infiltrated with cells, and may rupture spontaneously.

The course of the disease varies from patient to patient. In some, it is no more than a mild arthritis which totally recovers; in others it may be a severe, chronic debilitating disease, ultimately ending up in deformities. A typical case has a history of spontaneous remissions and exacerbations. Some of the factors known to precipitate an attack are physical exertion, psychological stress, infections and occasionally, trauma.

Stages of rheumatoid arthritis: From clinical viewpoint rheumatoid arthritis can be divided into three stages:

1. Potentially reversible soft tissue proliferations: In this stage, the disease is limited to the synovium. There occurs synovial hypertrophy and effusion. No destructive changes can be seen on X-rays.
2. Controllable but irreversible soft tissue destruction and early cartilage erosions: X-rays shows a reduction in the joint space, but outline of the articular surfaces is maintained.

3. Irreversible soft tissue and bony changes: The pannus ultimately destroys the articular cartilage and erodes the subchondral bone. The joint becomes ankylosed usually in a deformed position (fibrous ankylosis). It may be subluxated or dislocated.

Associated changes: In rheumatoid arthritis there is sometimes evidence of diffuse vasculitis. The most serious lesions occur in the arterial tree; which may be mild non-necrotising arteritis, or severe and fulminant arteritis akin to polyarteritis nodosa. The latter is fatal.

Diagnosis

Clinical features: It occurs between the age of 20 to 50 years. Women are affected about 3 times more commonly than men. Following presentations are common:

a) An acute symmetrical polyarthritis: Pain and stiffness in multiple joints (at least four), particularly in the morning, mark the beginning of the disease. This may be followed by frank symptoms of articular inflammation. The joints affected most commonly are the metacarpophalangeal joints, particularly that of the index finger. Other joints affected commonly are as given in Table–34.3.

Table–34.3: Joints affected in Rheumatoid arthritis

<table>
<thead>
<tr>
<th>Common</th>
<th>Less common</th>
<th>Uncommon</th>
</tr>
</thead>
<tbody>
<tr>
<td>• MP joints of hand</td>
<td>• Hip joint</td>
<td>• Atlanto-axial joint</td>
</tr>
<tr>
<td>• PIP joints of fingers</td>
<td>• Temporo-mandibular joint</td>
<td>• Facet joints of cervical spine</td>
</tr>
<tr>
<td>• Wrists, knees, elbows, ankles</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

b) Others: The onset may be with fever, the cause of which cannot be established (PUO), especially in children. Sometimes, visceral manifestations of the disease such as pneumonitis, rheumatoid nodules etc. may antedate the joint complaints.

On examination, one finds swollen boggy joints as a result of intra-articular effusion, synovial hypertrophy and oedema of the periarticular structures. The joints may be deformed (Table–34.4).
Table–34.4: Deformities in Rheumatoid arthritis

<table>
<thead>
<tr>
<th>Joint</th>
<th>Deformities</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hand</td>
<td>• Ulnar drift of the hand</td>
</tr>
<tr>
<td></td>
<td>• Boutonniere deformity</td>
</tr>
<tr>
<td></td>
<td>• Swan neck deformity</td>
</tr>
<tr>
<td>Elbow</td>
<td>• Flexion deformity</td>
</tr>
<tr>
<td></td>
<td>• Early - flexion deformity</td>
</tr>
<tr>
<td></td>
<td>• Late - triple* subluxation</td>
</tr>
<tr>
<td>Knee</td>
<td>• Equinus deformity</td>
</tr>
<tr>
<td></td>
<td>• Hallux valgus, Hammer toe, etc.</td>
</tr>
<tr>
<td>Ankle</td>
<td>• Equinus deformity</td>
</tr>
<tr>
<td>Foot</td>
<td>• Hallux valgus, Hammer toe, etc.</td>
</tr>
</tbody>
</table>

* Flexion, posterior subluxation and external rotation.

Joints of the hand show typical deformities as shown in Fig-34.1. There may be severe muscle spasm. Range of motion of the joints may be limited. In later stages, the joints may be subluxated or dislocated. There may be fever, rash and signs suggestive of systemic vasculitis. The rash in rheumatoid arthritis is typically non-pruritic and maculo-papular on the face, trunk and extremities.

Extra-articular manifestations of rheumatoid arthritis: Although, rheumatoid arthritis is primarily a chronic polyarthritis, extra-articular manifestations are very common, and sometimes govern the prognosis of a case. These are given in Table–34.5.

Investigations: Following investigations are useful:

1) Radiological examination: This consists of X-rays of both hands and of the affected joints. Following features may be present (Fig-34.2):
   - Reduced joint space
   - Erosion of articlar margins
   - Subchondral cysts

2) Blood: It shows the following changes:
   - Elevated ESR
   - Low haemoglobin value
   - Rheumatoid factor (RF): This is an auto antibody directed against the Fc fragment of immunoglobulin G (IgG). RF can belong to

Table–34.5: Extra-articular manifestations of Rheumatoid arthritis

<table>
<thead>
<tr>
<th>Syndrome</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>Vasculitis</td>
<td>• Digital arteritis</td>
</tr>
<tr>
<td></td>
<td>• Raynaud's phenomenon</td>
</tr>
<tr>
<td></td>
<td>• Fever, skin lesions, chronic leg ulcers</td>
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<tr>
<td></td>
<td>• Peripheral neuritis (mononeuritis multiplex)</td>
</tr>
<tr>
<td></td>
<td>• Necrotising arteritis involving coronary, mesentric or renal vessels</td>
</tr>
<tr>
<td>Rheumatoid nodule</td>
<td>• Commonest site - olecranon</td>
</tr>
<tr>
<td></td>
<td>• Other sites - dorsal surface of forearm, tendoachilles</td>
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<tr>
<td>Serositis</td>
<td>• Lung and pleura - pleurisy, parenchymatous nodules, Caplan's syndrome,</td>
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<td></td>
<td>• Honey comb lung</td>
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<tr>
<td></td>
<td>• Heart - cardiomyopathy, pericarditis</td>
</tr>
<tr>
<td></td>
<td>• Eye - iridocyclitis</td>
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<tr>
<td></td>
<td>• Nervous system - peripheral neuritis, carpal tunnel syndrome</td>
</tr>
<tr>
<td>Others</td>
<td>• Anaemia</td>
</tr>
<tr>
<td></td>
<td>• Felty's syndrome</td>
</tr>
<tr>
<td></td>
<td>• Sjogren's syndrome</td>
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<td>• Amyloidosis</td>
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</tbody>
</table>

Fig–34.1 Deformities in rheumatoid arthritis

Fig–34.2 X-rays of both hands, AP view, showing juxta-articular rarefaction
any class of immunoglobulins i.e., IgG-RF, IgM-RF, IgA-RF, or IgE-RF, but commonly done tests detect only the IgM type of RF. It can be detected in the serum of the patient by the following tests:

- **Latex fixation test:** This is an agglutination test where the antibodies are coated to latex particles. Positivity in titres more than 1/20 is significant. Sensitivity is 80 per cent.

- **Rose-Waaler test:** In this agglutination test sheep’s red blood cells are used as a carrier. Sensitivity is 60 per cent.

All patients with positive rheumatoid factor do not have rheumatoid arthritis. Conversely, all patients with rheumatoid arthritis do not have a positive rheumatoid factor. It is the constellation of signs and symptoms, titre in which the RF is positive, presence or absence of other positive tests, etc, which determine whether the patient has rheumatoid arthritis or not.

- **Synovial fluid examination**—See Table 22.2 on page 177.

- **Synovial biopsy:** This can be obtained arthroscopically or by open methods.

**DIFFERENTIAL DIAGNOSIS**

Rheumatoid arthritis must be differentiated from the following diseases:

a) **Systemic lupus erythematosus (SLE):** In SLE, the joint involvement is not symmetrical; nor are ankylosis and erosions common. Absence of anti-nuclear antibody factor (ANF) is in favour of rheumatoid arthritis, although its presence does not confirm SLE. It is present in 25 per cent cases of rheumatoid arthritis, though in low titres.

b) **Osteoarthritis:** This occurs in older patients. There is complete lack of the systemic features of rheumatoid arthritis such as fever, weight loss, fatigue etc. Distal inter-phalangeal joints are often involved. Duration of morning stiffness, joint swelling, ESR etc., are less compared to rheumatoid arthritis.

c) **Psoriatic arthropathy:** Characteristic skin and nail lesions may be present. Distal inter-phalangeal joints are usually involved. Rheumatoid factor is negative.

**TREATMENT**

**Principles of treatment:** Aims of treatment are as follows:

a) **Induction of remission and its maintenance:** Disease activity is brought under control by drugs.

b) **Preservation of joint functions and prevention of deformities** during the activity of the disease and thereafter, by physiotherapy and splinting.

c) **Repair of joint damage** which already exists, if it will relieve pain or facilitate functions. It sometimes requires surgical intervention e.g., synovectomy.

**Methods of treatment:** Above mentioned goals can be achieved by medical and orthopaedic treatment.

**Medical treatment:** Medical treatment essentially consists of anti-rheumatic drugs. These consist of: (i) non-steroidal anti-inflammatory drugs (NSAIDs); (ii) disease modifying anti-rheumatic drugs (DMARDs); and (iii) steroids. For details please refer to a Medicine textbook.

**Orthopaedic treatment:** Orthopaedic treatment aims at prevention of deformity, preservation of joint functions and rehabilitation. It falls essentially into non-operative and operative methods of treatment.

**Non-operative methods:** These consist of the following:

- **Physiotherapy:** This consists of: (i) splintage of the joints in proper position during the acute phase; (ii) heat therapy – wax bath, hot water fomentation for symptomatic relief; (iii) joint mobilisation exercises to maintain joint functions; and (iv) muscle building exercises to gain strength.

- **Occupational therapy:** Role of occupational therapy is to help the patient cope with his occupational requirements in the most comfortable way, by modifying them.

- **Rehabilitation:** Role of rehabilitation is to improve the functions of the patient with the help of devices like braces, walking aids etc.

**Operative methods:** Surgical treatment of rheumatoid arthritis can be divided into: (i) preventive surgery; (ii) palliative surgery; (iii) reconstructive surgery; and (iv) salvage surgery.
• **Preventive surgery:** This is done to prevent damage to the joint and nearby tendons by the inflamed, hypertrophied synovium. It consists of synovectomy of the wrist, knee and MP joints.

• **Palliative surgery:** This is done in situations where general condition of the patient does not permit corrective surgery, but some relief can be provided by limited surgical procedures such as bone block operations, tendon lengthening etc.

• **Reconstructive surgery:** This has revolutionised the rehabilitation of patients with deformed and painful joints. It includes tendon transfers, interposition arthroplasties and total joint replacement. With improvement in surgical techniques and better design of artificial joints, it is now possible to replace practically any joint of the body. The joints where total replacement is most popular are the hip, knee and metacarpophalangeal joints.

**Plan of treatment:** Management depends upon the stage of the disease, as discussed below:

1. **Potentially reversible** soft tissue proliferation, where drug therapy constitutes the mainstay of treatment.
2. **Controllable** but irreversible soft tissue destruction and early cartilage erosion, where a combination of drug therapy and orthopaedic treatment is required.
3. **Advanced stage of** joint destruction with subluxation or dislocation, where primarily surgical treatment is necessary. Drugs alone are of no use at this stage.

Plan of treatment in these three stages is as given in Table–34.6.

**Prognosis:** Following factors decide the outcome of a patient diagnosed to have rheumatoid arthritis.

**Table–34.6: Staged therapy in rheumatoid arthritis**

<table>
<thead>
<tr>
<th>Stage</th>
<th>Medical</th>
<th>Surgical</th>
<th>Physiotherapy</th>
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<tbody>
<tr>
<td>Stage I</td>
<td>DMARDs*</td>
<td>Synovectomy</td>
<td>Joint mobilisation</td>
</tr>
<tr>
<td></td>
<td>NSAIDs**</td>
<td></td>
<td></td>
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<tr>
<td>Stage II</td>
<td>NSAIDs</td>
<td>Soft tissue repair</td>
<td>Splints</td>
</tr>
<tr>
<td></td>
<td>DMARDs</td>
<td>Arthroplasty</td>
<td></td>
</tr>
<tr>
<td>Stage III</td>
<td>NSAIDs</td>
<td>Arthroplasty (joint replacement)</td>
<td>Splints and</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Arthrodesis</td>
<td>walking aids</td>
</tr>
</tbody>
</table>

* Disease modifying anti-rheumatic drugs  ** Non steroidal anti-inflammatory drugs

• **Natural history of the disease:** It is well known that rheumatoid arthritis is a disease with variable natural history. It may be fulminant i.e., damaging joints quickly and producing deformities in spite of best care, or more usually a disease with persistent course punctuated with remissions and exacerbations. It is not possible to predict the precise nature of the disease in a particular patient.

• **Sex and age at onset:** Women of child bearing age with predominant upper extremity involvement have a progressively severe disease. Males, with sparing of upper extremity, where onset of disease is under the age of 30 years, show less severe disease.

• **Type of onset:** It is generally believed that insidious onset disease progresses to have more severe disease.

• **Anaemia:** Anaemia is associated with progressive rheumatoid arthritis. Also, it is believed that unresponsiveness of anaemia to oral iron therapy is a bad prognostic indicator.

• **ESR and C-reactive protein:** High levels are associated with more erosive arthritis.

• **Rheumatoid factor:** A positive rheumatoid factor is associated with more progressive disease. High titres of rheumatoid factor, appearing early in the disease, carry a bad prognosis.

• **Radiological erosions:** Presence of erosions within 2 years of onset of the disease, is a bad prognostic indicator.

• **Histopathological changes:** A case with synovial proliferation, with increased number of synovial cells with DR antigen, carries bad prognosis.

**ANKYLOSING SPONDYLITIS**  
**Marie strumpell disease**

Ankylosing spondylitis is a chronic disease characterised by a progressive inflammatory...
stiffening of the joints, with a predilection for the joints of the axial skeleton, especially the sacro-iliac joints.

AETIOPATHOLOGY

The exact aetiology is not known. A strong association has been found between a genetic marker—HLA-B27 and this disease. Whereas, the incidence of HLA-B27 is less than 1 per cent in general population, it is present in more than 85 per cent of patients with ankylosing spondylitis.

Pathology: Sacro-iliac joints are usually the first to get involved; followed by the spine from the lumbar region upwards. The hip, the knee and the manubrium-sterni joints are also involved frequently. Initially synovitis occurs; followed later, by cartilage destruction and bony erosion. Resultant fibrosis ultimately leads to fibrous, followed by bony ankylosis. Ossification also occurs in the anterior longitudinal ligament and other ligaments of the spine. After bony fusion occurs, the pain may subside, leaving the spine permanently stiff (burnt out disease).

CLINICAL FEATURES

Presenting complaints: This is a disease of young adults, more common in males (M : F=10 : 1). The following clinical presentations may be seen:

a) Classic presentation: The patient is a young adult 15-30 years old, male, presenting with a gradual onset of pain and stiffness of the lower back. Initially, the stiffness may be noticed only after a period of rest, and improves with movement. Pain tends to be worst at night or early morning, awakening the patient from sleep. He gets better only after he walks about or does some exercises.

b) Unusual presentations: Patient may occasionally present with involvement of peripheral joints such as the shoulders, hips and knees. Smaller joints are rarely involved. Sometimes, a patient with ankylosing spondylitis may present with chronic inflammatory bowel disease; the joint symptoms follow.

On examination it is found that the patient walks with a straight stiff back. There may be a diffuse kyphosis. Following clinical signs may be present:

- Stiff spine: There may be a loss of lumbar lordosis. Lumbar spine flexion may be limited.
- Tests for detecting sacro-iliac involvement: Following tests may be positive in a case with sacro-iliac joint involvement:
  - Tenderness, localised to the posterior superior iliac spine or deep in the gluteal region.
  - Sacro-iliac compression: Direct side to side compression of the pelvis may cause pain at the sacro-iliac joints.
  - Gaenslen’s test: The hip and the knee joints of the opposite side are flexed to fix the pelvis, and the hip joint of the side under test is hyperextended over the edge of the table. This will exert a rotational strain over the sacro-iliac joint and give rise to pain (Fig-34.3a).
  - Straight leg raising test: The patient is asked to lift the leg up with the knee extended.

Fig–34.3 Tests for sacro-iliac joint affections
This will cause pain at the affected sacroiliac joint.

- **Pump-handle test:** With the patient lying supine, the examiner flexes his hip and knee completely, and forces the affected knee across the chest, so as to bring it close to the opposite shoulder (Fig-34.3b). This will cause pain on the affected side.

- **Tests for cervical spine involvement:** In advanced stages, the cervical spine gets completely stiff. The Fle’che test may detect an early involvement of the cervical spine.

- **Fle’che test:** The patient stands with his heel and back against the wall and tries to touch the wall with the back of his head without raising the chin. Inability to touch the head to the wall suggests cervical spine involvement.

- **Thoracic spine involvement:** Maximum chest expansion, from full expiration to full inspiration is measured at the level of the nipples. A chest expansion less than 5 cm indicates involvement of the costo-vertebral joints.

**Extra-articular manifestations:** In addition to articular symptoms, a patient with ankylosing spondylitis may have the following extra-articular manifestations:

a) **Ocular:** About 25 per cent patients with ankylosing spondylitis develop at least one attack of acute iritis sometimes during the natural history of the disease. Many patients suffer from recurrent episodes, which may result in scarring and depigmentation of the iris.

b) **Cardiovascular:** Patients with ankylosing spondylitis, especially those with a long standing illness, develop cardiovascular manifestations in the form of aortic incompetence, cardiomegaly, conduction defects, pericarditis etc.

c) **Neurological:** Patients may develop spontaneous dislocation and subluxation of the atlanto-axial joint or fractures of the cervical spine with trivial trauma, and may present with signs and symptoms of spinal cord compression.

d) **Pulmonary:** Involvement of the costo-vertebral joints lead to painless restriction of the thoracic cage. This can be detected clinically by diminished chest expansion, or by performing pulmonary function tests (PFT). There may also occur bilateral apical lobe fibrosis with cavitation, which remarkably simulates tuberculosis on X-ray.

e) **Systemic:** Generalised osteoporosis occurs commonly. Occasionally, a patient may develop amyloidosis.

**INVESTIGATIONS**

**Radiological examination** (Fig-34.4): In a suspected case, X-rays of the pelvis (AP), and dorso-lumbar spine (AP and lateral) are required. Oblique views of sacro-iliac joints may be required in early stages to appreciate their involvement. Following changes may be seen on X-ray of the pelvis:

- Haziness of the sacro-iliac joints
- Irregular subchondral erosions in SI joints

![Fig–34.4 X-rays showing changes in ankylosing spondylitis. (a) X-ray of the pelvis, AP view, showing bilateral SI joint and hip involvement (b) X-ray of the lumbar spine, Lateral view, showing calcification of the ligaments](image)

- Sclerosis of the articulating surfaces of SI joints
- Widening of the sacro-iliac joint space
- Bony ankylosis of the sacro-iliac joints
- Calcification of the sacro-iliac ligament and sacro-tuberous ligaments
- Evidence of enthesopathy – calcification at the attachment of the muscles, tendons and ligaments, particularly around the pelvis and around the heel.

X-ray of the lumbar spine may show the following:

- **Squaring of vertebrae:** The normal anterior concavity of the vertebral body is lost because
of calcification of the anterior longitudinal ligament.

- Loss of the lumbar lordosis.
- Bridging ‘osteoophytes’ (synodesmophytes).
- Bamboo spine appearance.

In the peripheral joints, X-ray changes are similar to those seen in rheumatoid arthritis, except that there is formation of large osteophytes and peri-articular calcification. Bony ankylosis occurs commonly.

**Other investigations:** These are the following:
- ESR: elevated
- Hb: mild anaemia
- HLA-B27: positive (to be tested in doubtful cases)

**DIFFERENTIAL DIAGNOSIS**

In early stages, ankylosing spondylitis may be confused with other disorders, as given in Table-34.7.

**OTHER RHEUMATOLOGICAL DISEASES**

**Gout**

Disturbed purine metabolism leading to excessive accumulation of uric acid in the blood — an inherited disorder; or impaired excretion of uric acid by the kidneys. The result is accumulation of sodium urate crystals in some soft tissues. Tissues of predilection are cartilage, tendon, bursa.

Patient, usually beyond 40 years of age, presents as (i) arthritis – MP joint of the big toe being a favourite site; onset is acute, pain is severe; (ii) bursitis – commonly of the olecranon bursa; or (iii) tophi formation deposit of uric acid salt in the soft tissue. Confirmation of diagnosis – urate crystals in the aspirate from a joint or bursa, high serum uric acid levels.

Treatment – NSAIDs, uricosuric drugs, uric acid inhibitors.

**Pseudogout**

*Sodium pyrophosphate* crystal deposition

Symptoms like those of gout

Meniscus calcification may be seen on X-rays of the knee.

Treatment - NSAIDs.
Psoriatic arthropathy
Presentation is like rheumatoid arthritis—a polyarthritis, distal IP joints of hands involved (unlike rheumatoid arthritis, where these are spared). Classic skin lesions help in diagnosis. Treatment is by steroids.

Alkaptonuric arthritis
(Ocronosis)
An inherited defect in enzyme system involved in metabolism of phenylalanine and tyrosine. As a result, homogentisic acid is excreted in patient’s urine. As a long term result, it accumulates in the cartilage and other connecting tissues. Joint symptoms occur after 40 years of age. Spine and shoulder joint are commonly affected. There may be evidence of pigment deposit in the sclera. Homogentisic acid is present in the urine, and results in the colour of the urine turning dark brown on standing (due to oxidation of homogentisic acid on exposure to air). X-ray—disc space calcification, peri-articular calcification in large joints. Treatment same as that for osteoarthritis.

Haemophilic arthritis
Occurs due to a number of bleeding disorders. Occurs in males. Joints affected commonly are knee, elbow and ankle. May present as acute or chronic haemarthrosis. There are other manifestations of bleeding disorders. X-ray—non-specific signs including bone resorption, cyst formation, osteoporosis, widening of intercondylar notch in the knee. Treatment—rest during acute stage along with factor VIII supplementation or other deficient factor replacement. In the chronic stage, physiotherapy, bracing etc. are required. Deformities may be corrected by conservative or operative methods.

Neuropathic arthropathy
(Charcot’s joint)
These are changes seen in a neuropathic joint, where repeated strain on a joint due to loss of sensations leads to severe degeneration. Clinically, the joint manifests as painless effusion, deformity or instability. The X-ray changes are those of severe osteoarthritis but without much clinical findings like pain, muscle spasm etc. Treatment is difficult. Bracing is usually advised for some joints. Fusion of the joint may be required.

**TREATMENT**

*No specific therapy* is available. Aim is to control the pain and maintain maximum degree of joint mobility. This can readily be achieved by life long pursuit of a structured exercise programme. In some cases surgical intervention is required.

**Conservative methods:** These consist of: (i) *drugs*—NSAIDs are given for pain relief; Indomethacin is effective in most cases; long acting preparations are preferred; (ii) *physiotherapy*—this consists of proper posture guidance, heat therapy and mobilisation exercises; (iii) *radiotherapy*—in some resistant cases; and (iv) *yoga therapy*.

**Operative methods:** Role of operative treatment is in correction of kyphotic deformities of the spine by spinal osteotomy, and joint replacement for cases with hip or knee joint ankylosis.

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**What have we learnt?**

- There are two types of arthritis: inflammatory and degenerative.
- Rheumatoid arthritis is a chronic polyarthritis of inflammatory nature, typically affecting peripheral joints.
- Orthopaedic management of rheumatoid arthritis is aimed at prevention of deformity, correction of deformity and joint replacement.
- Ankylosing spondylitis occurs in young men. Treatment is aimed at physiotherapy.