

ANKYLOSING SPONDYLITIS

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1. HISTORY

- This has been an important decade for our understanding of AS and a fitting occasion to review its history.
- Von Bechterew's report in 1893 may not clearly represent AS.
 Krebs, Scott, Forestier concurrently described sacroilitis after spinal roentgenology evolved in the 1930, and Robert and Forestier drew attention to the characteristic syndesmophyte on radiographs shortly thereafter.
- Elegant clinical, epidemiologic and family studies by Moll, Haslock, MacRae, and Wright in the 1960s and 1970s showed the inter relationship among AS, Reiter's syndrome, psoriatic arthritis, and enteropathic arthritis and led to the concept of the seronegative spondyloarthropathies.
- The irish physician Bernard Conner provided the 1ST pathogenic description and drawing of AS in1963 based on an ankylosed skeleton unearthed in a French cementery.

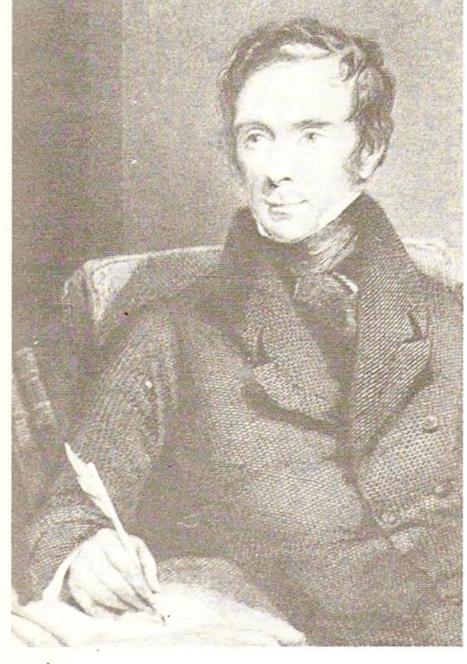


Fig. 1.7 Sir Benjamin Brodie (1783–1862) (from Pettigrew, 1838).

Sir Benjamin Brodie (1850) gave perhaps the best clinical description of the diseases.



Fig. 1.8 Charles Fagge (1838–1883) (from Rolleston, 1936; by courtesy of the Oxford University Press).

> Charles Fagge recorded a man of 34 who had ankylosis of the neck and dorsal spine. The macerated spine showed complete destruction and ossification of the apophyseal joints, hips and costovertebral joints. The upper lobe of the lungs showed Bronchiectasis. 2 yrs later Sturge in 1879 detailed a man of 26 with a fixed spine and chest starting at the age of 18 with back pain, urethritis and iritis.

- In 1973, Brewerton et al and Schlusstien et al discovered the genetic association with HLA-B27, which subsequently solidified the classification and broadened the spectrum of the spondylo arthropathies.
- The genetic sequence of the doned HLA-B27 gene was established in the late 1980s and the crystal structure of the molecule with its peptide binding groove was described in 1991. in 1990, Hammer et al introduced the human HLA-B27 gene into a transgenic rat which spontaneously developed disease resembling spondylo – arthropathies, thus producing the strongest evidence for the direct participation of HLA-B27 in the pathogenesis of AS.
- In 1994, these same investigators found that germ- free B27 transgenic rats failed to develop arthritis, thus emphasizing the additional requirement for bacteria in causing these disease.

2. DEFINITION AND TERMINOLOGY

- Ankylosing spondylitis(AS) { from the greek angkylos meaning "bent" and spondylos meaning "spinal vertebrae"} is an inflammatory diseases of unknown etiology characterized by prominent inflammation of spinal joints and adjacent structures leading to progressive and ascending bony fusion of the spine.
- Peripheral joints are less often affected, but hips and shoulders may become involved in 1/3rd of cases, and inflammatory lesions of extra- articular organs, such as the eye and heart, may occur. AS is clinically, radiographically, epidemiologically, and genetically related to a family of arthritides termed the "seronegative spondyloarthropathies."

> What are seronegative spondyloarthropathies?

AS is correctly categorized as a prototype of a spectrum of diseases sharing in common the presence of a specific histocompatability antigen, and lack of rheumatoid nodules, that are collectively known as seronegative (referring to the absence of serum Rh factor) spondylo arthropathies.

These include Reiter's syndrome, psoriatic arthritis, Rheumatiod arthritis, arthropathy resulting from Ulcerative colitis and Crohn's disease, as well as those from systemic gastrointestinal bacterial infection such as Yersinia, Shigella and Salmonella, which cause enteric arthritis.

Other members of this group include SAPHO. (Synovitis, ache, pustulosis, hyperosteosis, osteitis) syndrome, ache associated arthritis and Whipple's diseases.

3. EPIDEMIOLOGY-

Prevalence –

The incidence of patients with clinical manifestation of AS is two to three per thousand with an increased male to female ration of 2:1 to 10:1. the sacroilitis associated with AS is more prominent in males, and the disease is typically more progressive. Women tend to have more peripheral joint involvement and are sometimes misdiagnosed as having seronegative Rh arthritis. More often that not, men demonstrate a greater proportion of the spinal form of AS. In American and African blacks the overall prevalence of AS is approximately one- fourth that of Caucasians. Interestingly, although blacks do not have the B27 antigen, they do have the related B7 antigen. Although the prevalence rate among native Japanese is less than that of Caucasian Americans, the prevalence in North American Indians is very high, approaching 20% of their total male population.

Familial association –

 A logical approach to the presentation of epidemiological data is via familial association family studies of AS have shown aggregation of clinical disease, but with no clear pattern of inheritance.

Bremner et al found the proportion of 1st degree relatives with clinical disease to be 4% of 76 probands (63 males and 13 females) and applying the extended CIOMS (Rome) criteria, the proportion rose to 7%. These author also carried out a family study to determine the proportion of 1st degree relatives with asymptomatic radiological sacroilitis. 128 males and 60 females were X- rayed of whom 20 males and 5 females showed bilateral sacroiliac abnormalities, i.e. 13.3%. It is likely that had more female been x-rayed the male: female ratio of abnormalities would be narrowed.

earlier family studies showed that the frequency of AS among the relatives of patients with spondylitis was from 23 times higher to 30 times higher (Stecher, 1955) than in relatives of non spondylitic patients.

Falconer(1965) estimated the heritability of the liability to AS to be 70.0%, based on the prevalence of clinical disease in relatives, and when based on the prevalence of sacroiltis with or without manifestations of AS he calculated a liability of 72.5%.

Sex incidence –

AS has often been referred to as a disease of young men although recognized to occur in women. The sex ratio of adult males to female has been reported in a wide range varying from 10;1 (West 1949, Wright and Moll, 1973).

Hill and Bodmerl(1926) confirmed by finding that the evolution of AS in women with regard to both clinical and radiographic features is slower than in men, leading to a delay in diagnostic averaging nearly 10 year after onset, compared with just under 3 yrs in men.

When a presumptive diagnosis of AS is made, based on a characteristic pattern of history, Clinical features, ESR and the presence of HLA B27, the male:female sex ratio becomes as close as 2:3:1.

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In juvenile patients suffering from chronic polyarthritis, the sex ratio of those who carry the HLA B27 antigen is also close, and was found to be 9:7 male to female out of a group of 40 females and 26 males reported by Hall et al (1975). The male: female ratio of noncarrier was 17: 33.

- Ethnic and geographical distribution-
- Epidemiology in its broadest sense implicate the world, and it is both interesting and important to establish whether different races in their different environments suffer from the same condition, or whether is significant variation.
- There has been relatively little reliable information on the prevalence of clinical AS from around the world. West (1949) calculated a prevalence of one in 2000 in Bristol, UK and more than 20 yrs later Wright and Moll (1973) gave a frequency of 1 in 200 males and 1 in 2000 females in UK.

- The prevalence of AS Netherland was estimated to be 0.12% of the population over 15 yrs of the age(de Blecourt, 1973).
 Julkunmen (1962) reported a frequency in Finland of about 0.2% of the population over 15 yrs of age.
- Mendelck (1969) recorded his estimate of the population over 15 yrs of age suffering from AS in the Lebanon to be 1.3 %. Gomor et al (1974) quoted a frequency of 0.54% based on a district survey in Budapest, Hungary.

- HLA B27 antigen and ankylosing spondylitis -
- The antigen b27 is inherited as autosomal dominant with complete penetrance, but it is not the sole determinant affecting susceptibility in the spondyloarthritides - the other factor or factors being as yet unknown.
- It has been shown that about 7% of Caucasian controls carry the HLA B27 antigen, whereas it is present in over 90% of patients with AS and in about 50% of them 1st degree relatives.
- The striking association between the HLA B27 antigen and AS in Caucasians serves as a valuable diagnostic and aid. Studies in other ethnic groups indication a similar association, despite a wide variation in the occurrence of the antigen among different races.

- Testing more widely for HLA B27 specificity in various areas of japan, none was found, which highlights the specificity of HLA-B27 in the patients with AS. In other words, all persons in the spondylitis study carrying the b27 antigen proved to have the diseases, thus underlining the strong relationship between its and HLA-B27 in the Japanese, although only 2/3rd of those with spondylitis carried the antigen compared with about 90% of Caucasians.
- It must be remembered that the majority of b27 positive individuals do not develop AS and also that apparently identical spondylitis does occur in B27 –ve patients.
- Calin et al (1977) investigated the pima Indian of Arizona by clinical, radiological and HLA- typing, and included females as well as males. Radiological sacroilitis was used as a marker for AS. 100 male and 93 females over the age of 20 yrs who had pelvic radiographs were HLAB 27 was present in 57% of males but in only 9% of females.

HLA- B27 AND ANKYLOSING SPONDYLITIS(AS) IN CAUCASIAN POPULATIONS-

POPULATION

PERCENTAGE

> Frequency of HLA-B27 in normal individuals

8 – 14%

> Frequency of HLA-B27 in AS patients

> 90%

> Prevalence of AS in populations

0.2 – 1.4%

4. GENETICS -

 The disease occurs 30 times more commonly in the relatives of patient than in the general population. Recent research indicates a 90% or more relationship of AS with certain blood group, associated with the white blood cell (WBC).

• The WBC group involved in AS is known as HLA-B27 and is inherited, so the people born with this group have a much greater chance of developing this condition and at least 90% of the AS patients have positive HLA B27 antigen is lower among blacks, and it appears that clinical AS is less common in this group than the in white population.

Early studies in AS –

- Individual pedigrees have been described with two or more individuals with AS. The occurrence of disease in parents and occasionally in children, as well as in siblings of patients, has suggested the possibility of a major gene with autosomal dominant effect. (Riecker, neel, 1950).
- Investigations have been subsequently conducted to determine the frequency of diseases in close relatives. Whittinghill et al (1959) found that 1.3% of all relatives studied and 4.8% siblings had spondylitis compared with 0.11% in a control series.
- Julkunen (1962) found 1.9% in relatives and 0.19% in controls.
 There was aggregation of AS in families to a degree greater than would be expected by chance.

There is some evidence (West 1949, Neel and Test, 1950), that familial aggregation is greater with a female proband, giving some support to the idea that many genes contribute to susceptibility, females requiring a greater accumulative of such genes in order to develop disease.

• Emery and Lawrence (1967) examined the degree to which the relative frequencies in relatives in with major dominant or recssive inheritence and with a polygenic model. It was thought that the relative frequency of radiological sacroilitis found in siblings was most consistent with the latter hypothesis.

> Twins studies -

The evidence here consists of isolated reports of monozygotic twins. Concordant or discordant for the disease, it is possible to find reports of 18 such pairs of twins in the literature of which 13 were concordant and 5 discordant. Two discordant pairs were aged 48 and 52 yrs respectively, making it unlikely that the unaffected twin would subsequently develop disease. (Eastmond and Woodrow, 1972).

• The only definite conclusion to be drawn from these reports is that monozygotic twins may be discordant for AS.

Segregation of B27 and spondylitis –

• If the AS gene is B27 itself, 1 would except that the independent segregation of B27 and manifestations of spondylitis in families would occur but rarely. It is therefore to be anticipated that occasionally two separate genetic mechanisms for spondylitis, one possibly associated with B27 and the other not, could be segregating in a family. This might give the impression that a genetic recombination between B27 and a linked gene for spondylitis had occurred.

Evidence from B-27 negative spondylitis —

- If the AS gene were not B 27 but a gene at locus linked with the HLA B locus, it follows that most if not all instances of spondylitis in B27 – negative in individuals could in fact be genetically the same AS gene.
- It would follow that familial aggregation of B27 negative spondylitis would be observed to a degree comparable with that for B27 positive patients. So, far the evidence is against this (Vander linden 1977).
- In a personal series of 18 B27 negative patient the only instance of a sibling also had ulcerative colitis. Similar families have been described by Hochberg, Bias and Arnett(1928).

• The evidence quoted shows that B27 negative patients with inflammatory bowel disease not uncommonly get sacroilitis or spondylitis. Evidence has been put forward (Eastmond and Woodrow, 1977) that in some b 27 negative patients with spondylitis and chronic peripheral arthritis, the latter had features resembling those seen in psoriatic arthritis. It was thought possible therefore that some example of B27- negative spondylitis might result from the presence of gene for psoriasis in the absence of skin involvment.

5. ETIOLOGY

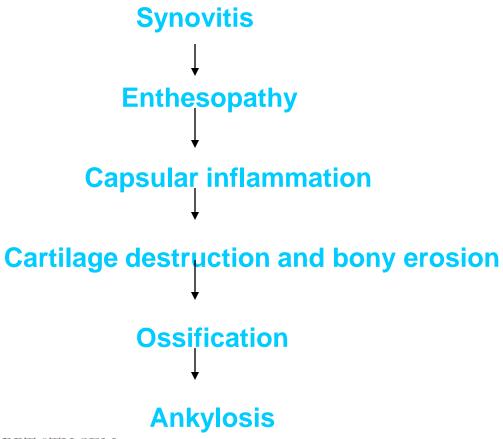
- In 1970's a +ve association between the HLA B27 histocompatibility antigen and AS was discovered, suggesting that the risk of disease is strongly, although not absolutely related to genetic risk.
- There is a 20 times greater incidence of AS among relatives with known AS, and these relatives have a much higher incidence of the b 27 antigen than the normal population. Whether this antigen is the primary cause or whether it acts as a receptor for an infective or environmental agent that triggers the disease is unknown.
- Although the etilogy of AS is unknown, several theories exist that may account for the pathogenesis of this disease. An infectious etilogy is populated with Klebsiella, a ubiquitous organism, incriminated as a possible triggering infection before onset of the disease, although definitive proof is lacking.

 In addition to the HLA B27 link, raised level of complement in active products, certain anti globulins, and circulating immune complex suggests an immunologic role in the pathogenesis of AS.

 Conceivably, an infectious agent might interact with B27 positive cells to render them auto antigenic, leading to an autoimmune state.
 The pathogenesis of AS may in fact be a complex interplay between genetic, autoimmune, antigenic and infectious variables.

6. PATHOLOGY AND PATHOGENESIS

The pathology of AS include the following process-



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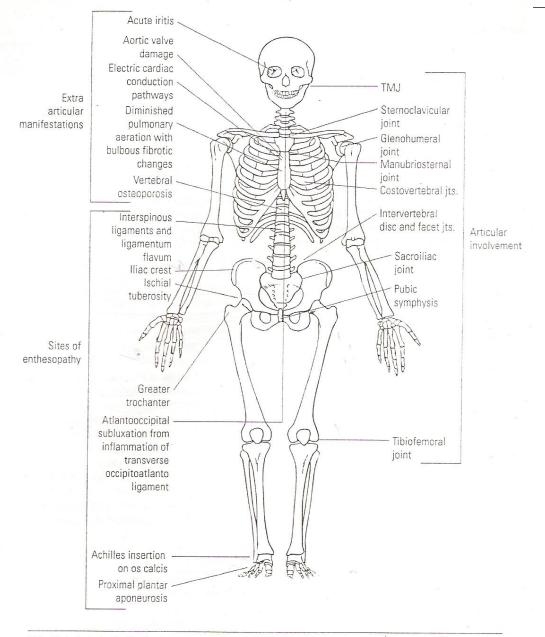


FIG. 56-1 Pathologic distribution of *ankylosing spondylitis*. Pathology includes articular involvement, enthesopathic sites, and extraarticular manifestations.

- The distinctive pathologic feature of AS is the involvement of the enthesis, the site of insertion of the ligaments and jt. Capsule into the bone. Enthesopathy may cause bony erosions at the enthesis.
- The reparative process that follows the initial inflammatory phase is characterized by ossification at these sites. This enthesopathic process is the basis for many of the findings –

for ex:- heel pain and spurs, syndesmophyte formation, and squaring of the vertebral bodies.

- The recent demonstration of persisting bacterial antigens and possibly dormant, but viable, micro-organisms in the peripheral jts. Of patients with reactive arthritis leads to speculation concerning bacterial antigens and micro-organism in the spinal jts in AS.
- To date, no such studies have been reported, probably because of the difficulty in obtaining the necessary pathologic material to study, and the lack of candidate bacteria for which to search.

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 Additionally, elevated serum level of IgA antibodies to the causative bacteria in reactive arthritis have been reported that probably reflect increased mucosal immunity to persisting infection.

 Recent searches for increased serum levels of the antibodies to a variety of bacteria in AS patients showed increased levels of IgA antibodies to Klebsiella or E coli in 12%, while there were no associations with the bacteria known to trigger reactive arthritis.

 Recent studies of synovial fluid in reactive arthritis never demonstrated that mononuclear cells proliferate specifically to the organism which triggered the arthritis, and bacteria specific T cell responses.

SPONDARTHRITIDES

- There is an increased frequency of spondylitis in patients with certain other condition such as ulcerative colitis, Crohn's disease, psoriasis, Reiter's disease and acute anterior uveitis, and it is important top study this association both individuals and in population in case further light may be thrown on aetiological factors, whether genetic or environmental.
- Huaux et al (1972) studied patients with Crohn's disease and found that Crohn's disease associated with spondylitis showed a higher frequency in those with B27 (33.3%) than in normal Caucasian population, although the relationship was not as strong as as seen with pure ankylosing spondylitis (80.6%). None of the patient with Crohn's disease was HLA B27 +ve.

 Bluestone et al (1975) found that 67% of their patients with both chronic inflammatory bowel disease and spondylitis were B27+ve whereas the antigen was absent in patients with peripheral arthropathy alone. Iritis was present in 6 out of 12 patients with spondylitis, compared with one out of 33 without spondylitis.

Roux et al (1977) studied 108 Caucasian patients with psoriasis and were able to separate the associated arthritis and were able to separate the associated arthritis into 3 distinct patterns. These was a central from closly related to ankylosing spondylitis, of which 50% were B27+ve, whereas the peripheral form of inflammatory arthritis even with sacroilitis was associated with B27 +ve in 9%, and in peripheral inflammatory arthritis without sacroilitis.

8. EXTRA- ARTICULAR MANIFESTATIONS -

- 1. OCCULAR
- 2. CARDIAC MANIFESTATION
- 3. PULMONARY
- 4. RENAL
- 5. NEUROGENIC
- 6. GASTROINTESTINAL TRACT

1. OCCULAR - About 25% - 30% of patient suffering frim AS experience acute attacks, usally unilateral iritis. Typically symptoms include the sudden onset of occular pain, redness and photophobia. Unless inflammation is promptly suppressed, debris may accumulate in the ant. Chamber causing pupillary and lens dysfunction and blurring of vision.

2. CARDIAC MANIFESTATION — About 2-5% of patients develop inflammation of the proximal aorta, resulting in aortic valve incompetence. other cardiac lesion include conduction defects, cardiac myopathy and pericarditis.

- 3. PULMONARY —lung involvement in AS is unusual. Despite a diminised chest expansion due to costovertebral jt. Fusion, patients with AS rarly have significant reduction in total lung and vital capacities because diaphragmatic function is not imparied. Bilateral apical pulmonary fibrosis occurs in approximately 1% of patients, usually after many years of disease, and cavitation mimicking TB.
- **4.RENAL-** secondary amyloidosis complicates the course of AS and other spondyloarthropathies in 1 to 3% of cases, and occurs more commonly in Europe than in the US.

proteinuria, often in the nephrotic range, is the usual presentation, and progression to renal failure is common Proteinuria, with or without renal failure impairment, may also indicate the presence of IgA nephropathy, which is of considerable inter in view of serum elevation of IgA in AS patients.

5. NEUROLOGIC- Besides cervical spine fractures and dislocation, a slowly progressive cauda equina syndrome may appear late in the diseases course. Usual symptoms include sensory loss in lumbar and sacral dermatome and less often, lower extremity weakness and pain, and loss of urinary and rectal sphincter tone.

MRI is the most reliable means of demonstrating the characteristic enlarged dural sacs and arachnoid diverticulaqm and excluding other pontentially surgically correctable myelopathies. These may be be an increased frequency of multiple sclerosis in AS patients, but this has not been proven by definitive epidemiology studies.

6. GASTRO INTESTINAL TRACT- asymptomatic areas of both macro and microscopic inflammation in the proximal colon and terminal ileum have been demonstrated by ileocolonoscopy in up to 60% of patients with AS. Rarely, patients with long standing AS will develop typical symptoms and findings of inflammatory bowel diseases.

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9. CLINICAL FEATURES -

➤ The sacroiliac joint is often the 1st joint of the axial skeleton to be affected by inflammatory disorders because AS is a disease of the spine and axial skeleton, and is charcterized by a caudal to cephalad migration, it is not surprising that the sacroilitis, often perceived as pain in the low back is the very 1st manifestation of AS.

The most characteristic and the commonest early complaints are:-

- a) LOW LUMBAR BACKACHE.
- b) ACHES IN THE BUTTOCKS.
- c) EARLY MORNING STIFFNESS.
- d) RESTRICTION OF CHEST MOVEMENT.
- e) BONY TENDERNESS.
- f) PERIPHERAL JOINT INVOLVEMENT.

- a) LOW LUMBAR BACKACHE- This may occur as the 1st symptom in AS, but more often it follows the more typical buttock ache. Pain may center at the lumbosacral spine but may also occur in the buttocks and hips. Asked for site for initial pain, the patient usually draws his hand from his buttock down the back of the thigh on one or both sides, less often he draws his hand sideways across the lower lumbar spine to indicate his initial discomfort several months later the whole area becomes painful and the discomfort may also extend up the spine.
- b) ACHE IN THE BUTTOCK- AS is usually gradual and undramatic in onset. Romanus and Yden (1955), in an excellent description of the pain of sacroilitis, emphasize that the pain is deep in gluteal region, is often described as a dull ache, but even if severe is difficult for the patient to localize accurately. It may extend upwards toward the iliac creast down the thigh, towards the trochantric region, and occasionally towards the groin.

15-11-2018 A coughi sneeze or sudden twist of the upper part of the body may cause a sharp transient exacerbation of this pain.

- c) EARLY MORNING STIFFNESS- Due to ankylosis of the vertebral bodies, the movement of the spine is reduced or diminished, thus the condition of the stiff back develop. Patient can neither bend forward nor can he bend backward. The longer the time spent in bed are the deeper the sleep is worse he is in the early morning, and heavy sedation for insomnia aggravates these symptoms. This painful stiffness involves primarily the lumbar are sacroillic regions but may soon involve the dorsal and cervical spine and also, in more severe cases the hips. It may last for 1,2 or more hours, or even for most of the morning until eased by movement are excessive.
- d) RESTRICTION OF CHEST MOVEMENT- Due to the involvement of the costovertebral joints and manubriosternal joint, the restriction of the chest expansion occurs. Discomfort and thoracic restriction restriction is noticed by the patient when he over breathes and attempts full expansion of his rib cage.

- AS diaphragmatic movements are full and normal, only considerable exertion involving much over breathing produces this awareness of restricted movt. Of the chest wall, but occasionally it is a major early symptom, and the patient may be mistakenly reffered to a chest or cardiac clinic. The antero-lateral distance between the lower costal ribcage and iliac creast is reduced so diminished and the patient stands with hips and knees flexed.
- e) BONY TENDERNESS- Bony tenderness may be one of the early feature of AS, the patient noting that his ant. Chest wall over sternum, ribs and cartilages has become tender on pressure. The patient may also complain of pain and swelling of the sternomanubrial and sterno-clavicular joints, which may also become very tender on pressure.

Tenderness over the pelvic brim may cause discomfort at night and interfere with sleep, as turning onto iliac creast cause pain are wakes the patient. H SEM

f) PERIPHERAL JOINT INVOLVEMENT- In severe AS, inflammation gradually spreads upto the spine and is reflected in the patients inability to move easily. His movements becomed so restricted that patient finds it difficult, as well as painful to bend either forwards, sideways or to twist around. Instead of moving his head to look behind, the patient turns his whole body around, and when there is a need to pick up an object from the floor, then he carefully lower himself by bending his knees rather than his spine.

10. INVESTIGATION-

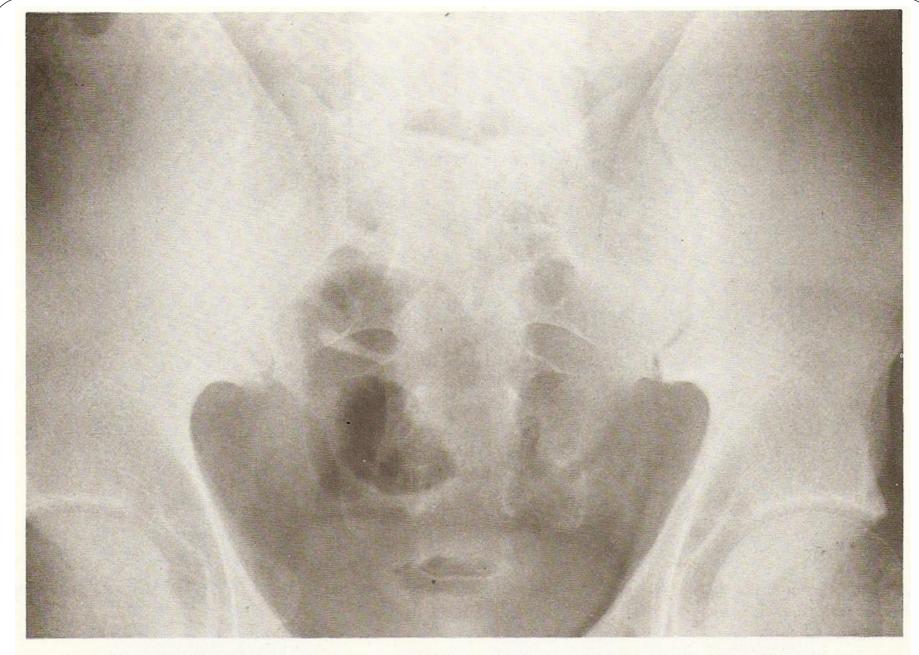
- * RADIOLOGIC
- + LABORATORY
- Radiographic- Characteristic radiological changes may be seen at various site and by there radiographs we can confirm the diagnosis features of different sites are-
- In sacro-iliac joints-

Radiologically the SI jts. are staged as:-

- 0- unequivocally normal.
- 1- possible abnormal.
- 2- Definite marginal sclerosis.
- 3- Definite erosions and sclerosis.
- 4- complete obliteration.



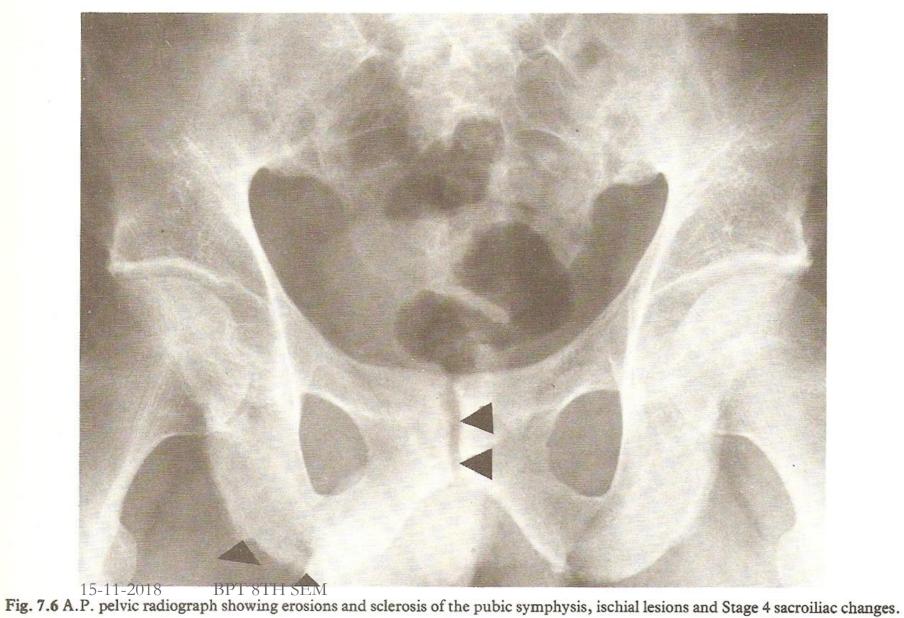
15-11-2018 BPT 8TH SEM Fig. 7.1 A.P. pelvic radiograph showing the normal appearance of the sacroiliac joints.



15-Fig 2012 A.P. pelvic radiograph showing definite marginal sclerosis of the sacroiliac joints (Stage 2).



Fig. 7.3 A.P. pelvic radiograph showing definite erosion of bone and marginal sclerosis (Stage 3)



- Sclerosis of the ilium and sacrum on either side of the joint.
- Haziness of the joint margins which later on show erosion.
- Narrowing of the joint space which may progress to fusion .
- When ankylosis is complete, the periarticular sclerosis fades sometime leaving the evidence of the previous jt. line, known as "GHOST JOINT".
- Sacroilitis is usually bilateral but may be unilateral or asymmetrical, early in the disease.



Fig. 7.9 Vertebral discitis in a patient with ankylosing spondylitis.

• In spine-

Initial stage-

- Squarring of the vertebral bodies.
- Syndesmophyte formation usually first seen at the thoracolumbar level.
- Arthritic changes and later apophyseal joint fusion occur.
 which is best seen in cervical spine.
- Atlanto- axial subluxation
- Calcification of the paraspinal ligament.
- Erosion surrounded by sclerosis at vertebral body margin (i.e called Romanus sign.)

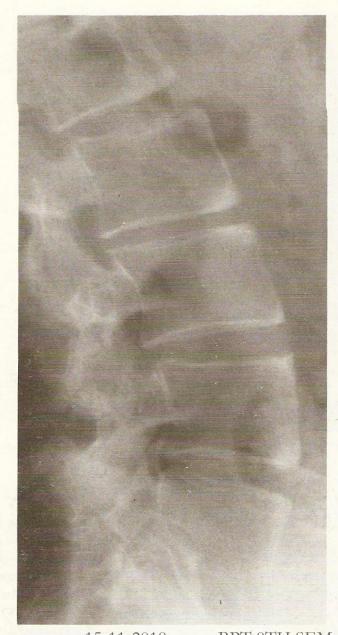


Fig. 7.7 Lateral radiograph of lumbar spine showing vertebral squaring with sclerosis at the attachment of the anterior portion of the annulus fibrosus.

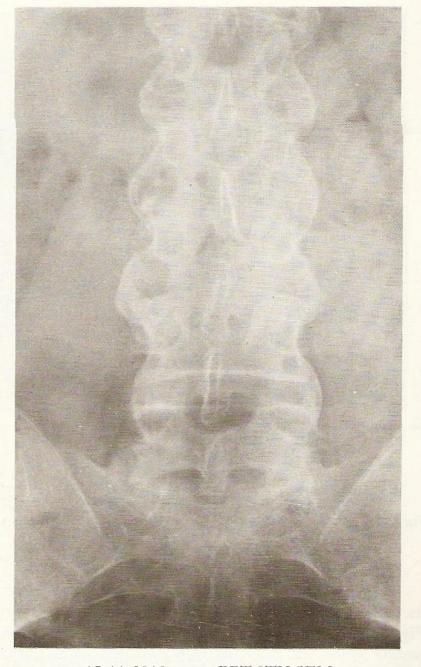


Fig. 7.8 A.P. radiograph of the lumbosacral spine in advanced ankylosing spondylitis. The spinal ligaments are calcified giving an appearance similar to a piece of bamboo.

In Advanced stage-

- The characteristic "bamboo spine" results from syndesmophyte or paraspinal ligament calcification around a normal disc space.
- Spondylodiscitis may develop in the lower thoracic or upper lumbar segments. Erosive changes in the ant. Vertebral bodies adjacent to the disc, which becomes progressively destroyed and causes angulation of the spine.
- Ligamentous attachments (ligamentous bony junction) inflammation and secondary ossification at these junction in areas such as the pelvis, the greater trochanter of the femur, plantar fascia and the achilles tendon leads to proliferation bony margins and whiskery spicules.



Fig. 7.10 Lateral radiograph of cervical spine showing obliteration of the upper apophyseal joints. A surprisingly large range of movement was available at the solitary articulation at C6-7.



Fig. 7.11 A.P. radiograph of the kned in advanced ankylosing spondylitis showing erosions and bony fusion of the superior tibio-fibular joint.

Peripheral joints:

Initially, the X-ray appearance of proximal joints in AS may resemble RA. However there is a greater tendency in AS to central articular erosion and proliferative newborn formation in peri-articular tissue with bony ankylosis.

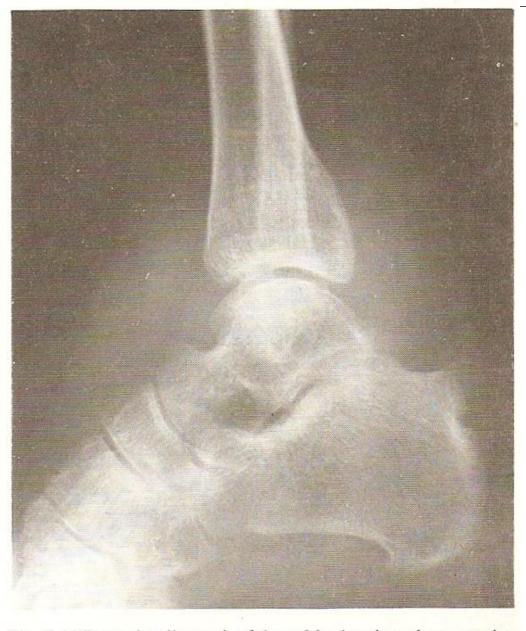


Fig. 7.12 Lateral radiograph of the ankle showing a bone erosion in the region of the Achilles bursa, periosteal calcification at the calcaneal attachment of the plantar fascia and obliteration of the calcaneo-talar joint.

- LABORATORY certain laboratory tests done for the confirmtion to make the diagnosis of AS are as follows-
- A. ESR- the sedimentation rate is generally raised in proportion to the inflammatory activity in about 70% of patients.
- B. HLA- B27 TEST- this is positive in about 95% of patients.
- C. HAEMOGLOBIN- normochromic or normocytic anaemia may occur but in contrast to RA, patients with active disease often have a normal haemoglobin and blood firm.
- D. SYNOVIAL FLUID- contains a moderate no. of mononuclear leucocytes in contrast to the increased polymorphonuclear leucocyte count of RA fluid.

- E. RHEUMATIOD FACTOR- rheumatoid factors are absent.
- F. NUCLEAR SCAN- Technitium sternous pyrophosphate bone scans, can often detect areas of active inflammation in AS, before standard radiographic changes are present.
- G. PULMONARY FUNCTION TEST- Pulmonary function tests in patients with thoracic involvement usually shows diminished vital and total lung capacity, increased residual volume and functional residual volume.

11. COMPLICATION OF AS-

- A. ATLANTO AXIAL SUBLUXATION
- **B. CAUDA EQUINA SYNDROME**
- C. PATHOLOGIC FRACTURE
- D. REDUCE CHEST EXPANSION
- E. ROMANUS LESIONS.
- F. IRIDOCYCLITIS.

A. ATLANTOAXIAL SUBLUXATION –As AS progresses cephalad, increased stress at the craniocervical junction may occur due to solid column of bone inferior to the atlanto axial and atlanto occipital joints. Atlantoaxial instability is caused by inflammation of the transverse atlantooccipital –atlantoligament.

The 1st and 2nd cervical vertebrae are respectively known as the atlas and axis. The atlas resembling no other vertebral, lacks a vertebral body and is a ring of bone that carries the skull while the axis forms a pivot around which the atlas and skull may rotate. Thus nodding occurs at the atlantooccipital joint wheras rotation occurs at the atlantoaxial joint.

The combination of this stress coupled with attritional effects of inflammation of the transverse ligaments and bony attachments associated with AS may cause severe pain, instability, ant. subluxation and even dislocation in which the atlas mayslides forward on the axis and impinge upon the spinal cord by odontiod process.

- B. CAUDA EQUINA SYNDROME- This results from encasement and stenosis of nerve roots in deformed vertebral bone causing bedwetting, impotence, diminished bladder and rectal sensation, buttock or lower extremity pain, or an absent ankle reflex.
- C. PATHOLOGIC FRACTURE- AS the pelvis, spine and thorax become fixed into a single rigid bony union, the axial and proximal appendicular skeleton lose the energy dissipating capability afforded by the joints and soft tissue comprising the musculoskeletal system. In addition, the lessened activity accelerate the rate of osteoporosis in the skeletal system. The lack of trunk mobility increases the risk for falls and pathologic fracture.
- D REDUCED CHEST EXPANSION –. Fibrosis of the upper lobesof the lungs was a not uncommon finding in AS in pre-war years due to apical pulmonary tuberculosis.

Recently Gacad and masaro(1974) described 3 such patients with fibrocavited pumonry diseases, 2 yielding cultures of mycobacterium fortuitum. In most cases reported, sputum cultures have been negative, but in 2 of the authors patients tubercle bacilli were eventually isolated and treatment given successfully. The chest expansion decreses, usually being less than 2 inches initially, and gradually progresses to a mere ½ inch. This is due to the involvement of the costochondral joints. The spinal mobility is decrease considerably.

E. ROMANUS SIGN- Romanus lesions are relatively uncommon inflammatory lesions affecting the edge of 1 or more vertebrae, adjacent to the disc. They are often painful. Radiographically they appear a erosive defects which closely looks like infective laricens.

F. AMYLOIDOSIS- Amyloidosis occurs as a rare complication of ankylosing spondylitis. Cruickshank(1960) found it in 6% of his autopsy series, the patients all dying of uraemia from this complication.

G. CRICOARYTENOID ARTHRITIS- These is rare in Rh but is even more rare in AS. Two of the author's patients, both with severe advanced Ankylosing disease developed this as a late complication (Wojtolewski et al, 1973) and a similar patient was reported by Berender and Miehlke (1973). Two of the 3 patients develop severe dyspnoea necessitating tracheostomy.

12. DIAGNOSTIC TEST-

CLINICAL CRITERIA-

Low back pain and stiffness for more than 3 months, which is important by exercise and unrelieved by rest. Limitation of chest expansion, lumbar spine movement in both sagittal and frontal planes.

RADIOLOGICAL CRITERIA-

Defined as radiological criteria and is associated with at least one clinical criteria. Calin et al (1977) analysed the factors in the clinical history that lead to a positive diagnosis of AS.

The 7 most important factors were-

- 1. Onset of back discomfort before the age of 40.
- 2. An incidious rather than sudden onset.
- 3. Persistance for more than 3 years.
- 4. Associated with early morning stiffness.
- 5. Improved with exercise.
- 6. Limitation of chest expansion.
- 157.1-12mitation of lumbar spinal motion in both saggital and frontal plane.

There is also another different criteria for the diagnosis and that is "NEW YORK CRITERIA" given by Bennet and Wood.

CRITERIA FOR DIAGNOSING ANKYLOSING SPONDYLITIS ROME, 1961

CLINICAL CRITERIA-

- 1.Low back pain and stiffness for more than 3 months, which is not relived by rest.
- 2. pain and stiffness in thoracic region.
- 3. limited motion in lumbar spine.
- 4. limited chest expansion.
- 5. history or evidence of iritis or its sequelae.

RADIOLOGIC CRITERIA-

6. roentgenogram showing bilateral sacroiliac changes.

BPT 8TH SEM

NEW YORK ,1966

DIAGNOSIS

- 1. limitation of motion of the lumbar spine in all three planes- anterior fexion, lateral flexion and extension.
- 2. pain at the dorsolumbar junction or in the lumbar spine.
- 3. limitation of chest expansion to 1 inch(2.5cm) or less measured at the level of the 4th intercostal space.

GRADING OF RADIOGRAPHS- 0- normal,1- suspicious,2- minimal sacroiliitis, 3- moderate sacroiliitis, 4- ankylosis.

DEFINITE AS

- 1. Grade 3-4 bilateral sacroiliitis with at least one clinical criterion.
- 2. Grade 3-4 bunilateral or grade 2 bilateral sacroiliitis with clinical criterion 1 or with both clinical criteria 2 and 3.

PROBABLE AS BPT 8TH SEM

Grade 3-4 bilateral sacroiliitis with no clinical criteria.

MODIFIED NEW YORK CRITERIA

- 1. Low- back pain of at least 3 months duration improved by exercise and not relieved by rest.
- 2. Limitation of lumbar spine in sagittal and frontal planes.
- 3. Chest expansion decresed relative to normal values for age and sex.
- 4. Bilateral sacroiliitis, grade 2-4.
- 5. Unilateral sacroiliitis, grade 3-4. definite AS if unilateral grade 3 or 4 or bilateral grade 2-4 sacroiliitis and any clinical criteria.

13. DIFFERENTIAL DIAGNOSIS-

The differential diagnosis of AS represents a clinical challenge. Many patients weith AS remain undiagnosed or are incorrectly diagnosed with neurosis, mechanical back pain, or seronegative Rh arthritis, and inappropriately receive bed rest, improper exercise, or undergo a myelogram. The clinician most bear in the mind that lab tests are of limited diagnostic use. The B27 antigen is the single best laboratory clue as it is present in 95% of all white spondylitics. Diagnosis is suggested by patient history and examination, and corroborated by positive radiographs. If 3 or more features are present, a possibility of AS is strongly considered.

- Condition that must be distinguished from AS include-
 - 1. Rh ARTHRITIS
 - 2. PROLAPSED INTERVERTEBRAL DISC (PID)
 - 3. REITER'S SYNDROME
 - 4. SEPTIC SACROILITIS
 - 5. PSORIATIC ARTHRITIS
 - 6. OSTEOARTHRITIS
 - 7. OSTEITIS CONDENSANS ILII.
 - 8. LUMBOSACRAL DISC DISEASE
 - 9. DIFFUSE IDIOPATHIC SKELETAL HYPEROSTOSIS.
 - 10. OLIGOARTICULAR ARTHRITIS

- 1. Rh ARTHRITIS when comparing AS and Rh arthritis we note more differences than similarities between both spondylarthropathies.
- Those tissue affected by AS are primarily composed of dense connective tissue, where as rheumatoid arthritis has a predilection for inflammation of loose connective tissue.
- AS is distinctly different from Rh arthritis as a distinct clinical and pathologic entity in that it is predominant in young men, demonstrates absence of Rh factor and nodules, and has a predilection for the axial skeleton. Spinal involvement is total and ascending with AS, but only confined to the cervical spine in patients Rh arthritis.
- In AS the distribution is racial, a positive family history is frequent, it is more frequently diagnosed in males, it involves the SI joint, aortic regurgitation may be present, and it has no nodules.

- In contrast, with RA distribution is worldwide, a family history is rare, it is more common in female, the SI joint is not involved, there is no aortic regurgitation, and nodules are typically present.
- In AS the main presenting clinical problems are gross fixed deformities, where as in Rh arthritis, the main problem are local jts. destruction and instability.
- 2. PID In PID the mode of onset is sudden where as in AS it is insidious. The site of pain in PID is usually lumbar where as in AS it is lumbar buttocks, thoracic.

<u>Features</u> <u>AS</u> <u>PID</u>

- Effect of rest incresing stiffness decresing pain
- Effect of exercise decresing pain and stiffness incresing pain
- Inspection BPT Susually no scoliosis scoliosis often present

- 3. REITER'S SYNDROME- Reiter's syndrome classically presents as a triad of arthritis, uveitis and urethritis. Patients may also develop hyperakeratotic skin lesions of the palms and sole around the nails, whereas hyperkeratosis may also occur.
- The 2 recognized form of Reiter's syndrome include the dysentric form in which bowel inflammation is caused by different infectious agents and the sexually transmitted form. Both form of Reiter's syndrome may manifest in the spine as a particular type of syndesmophyte that is uniquely different from that of AS or the spurring associated with osteoarthritis of the spine.
- In Reiter's syndrome bony outgrowth occurs form the lateral and ant. Surfaces of the vertebral bodies where as in AS osteophytes grow from the margins of the vertebral bodies.

- 4. SEPTIC SACROILITIS This is a form of infectious arthritis caused by a pathologic microbe infecting synovial tissue. The most commonly implicated pathogen is staphylococcus.
- Patient will complain of acute sacroiliac joint pain and stiffness, a warm and tender joint, and fever and chills that are often present. Because the sacroiliac joint may be rapidly destroyed if this condition is not promptly treated, acute bacterial arthritis is considered a medical emergency. Suspected septic sacroilitis should undergo needle aspiration and culture.
- 5. PSORIATIC ARTHRITIS Psoriatic arthritis differs from AS in that it is more common in females, whereas the syndesmophytes tend to be asymmetric.
- The distal IP joints are especially affected as they undergo excessive destruction and become swollen and distorted in what is called anthritis mutilans. The toes may also exhibit a sausage-like swelling.

- 6. OSTEOARTHRITIS Osteoarthritis of the sacroiliac joint does occur although radiologic involvement is limited to the lower portion of the joints. This is not the case with AS in which radiologic involvement involves the entire joints.
- 7. OSTEITIS CONDENSANS ILII this is a sclerotic condition of 1 or both iliac surfaces articulating with the sacrum. This condition, common to females, may be asymptomatic in multiparous women, or may present as back pain in both men and women runners secondary to leg length discrepancy or running along a banked surface secondary to shearing action across the sacroiliac joint.
- This condition is radiographically characterized by sclerosis of the sacoiliac jt that differs from AS in that sclerosis is wedge- shaped and confined to the iliac side of the joint so that the joint margin is more clearly demarcated. This is not the case in with AS.

- 8. LUMBOSACRAL DISC DISEASE- Lumbar disc disease is a mechanical rather than inflammatory condition whose clinical features include acute onset, local muscle spasm, neurodeficit, local tenderness, and asymmrtrical loss of motion.
- Lumbosacral nerve entrapment is worsened by exercise and made better by rest. These features are not present with AS, which is characterized by an opposite symptomatic pattern in which pain intensifies with rest and improves with exercise.
- SLR is normal, whereas onset is insidious. In addition, with AS lateral flexion is usually abnormally limited, and typically unaffected by lumbar disc. Finally, the tenderness in AS is diffuse, whereas diminished range of motion is symmetric.

- 9. DIFFUSE IDIOPATHIC SKELETAL HYPEROSTOSIS (DISC)- DISC is synonymous with ankylosing hyperostosis and Forestier disease, and results from an abnormal tendency of the body to ossify excessvely taut, fibrous connective tissue, typically occuring as ligaments, joint capsules, and tendons of the spine.
- This condition closely resembles and even clinically mimics AS, and differs only in that it occurs in elderly males greater than 50 years of age.
- Radiologically evident ligamentous calcification is limited to the cervical and lower thoracic spine but spares the sacroiliac and vertebral facet joint.
- With this condition, ossification of the anterior longitudinal ligament does occurs, whereas it does not occur in AS. In addition, the ESR is normal and there is no link to the B27 antigen.

10. OLIGOARTICULAR ARTHRITIS – Oligoarticular arthritis of the lower extremities, typically as heel pain, is often the early presentation of juvenile AS in older boys before evidence of radiographic sacroilitis. Almost all children are B27 positive and this may mimic and often be confused with Rh arthritis. However, with time the child develops the more typical feature of the adult form of AS.

14. MANAGEMENT-

CONSERVATIVE MANAGEMENT.

- PHARMACOLOGICAL MANAGEMENT.
- PHYSIOTHERAPY MANAGEMENT.
 - a. Physical examination
 - b. Rehabilitation in acute stage.
 - c. Rehabilitation in subacute and chronic stage

2. SURGICAL MANAGEMENT.

1.CONSERVATIVE MANAGEMENT-

A. <u>PHARMACOLOGICAL MANAGEMENT-</u> the role of the drugs is to relieve pain and inflammation to maintain posture and allows an effective physical therapy or exercise programme.

ANTI- SPONDYLITIC DRUGS-

- D- pencillamine- It has been tentatively tried Scharf and Nahir(1976) wrote of a case of AS with severe peripheral arthritis. The peripheral jt. Improved markedly after 4 months treated with penicillamine and subsequently settled completely.
- Levamisole- It was tested by Rosenthal, Trabert and Muller(1976) in a long term open study of 13 patients with AS. The mean periodof observation was 22 weeks. 7 of 13 improved subjectively, but only in 4 there was an increasing in spinal movement.

- Asprin, indomethacine and other anti inflammatory drugs are more effective in AS, particulary, for relieving early morning stiffness.
- > Sulphasalazine may actually help to control the underlying inflammation. It is principally used when there is a lot of active inflammation, especially in the earlier stages of the disease.
- Phenylbutazone afford good relief of acute symptoms but has a predictable bone marrow toxicity. Hart(1954) rated phenylbutazone as the most useful drug in AS, both in the early painful phase and also later in the disease.
- Other non- steroidal antiinflammatory drugs such as naproxen, sulindoc, piroxicam and tolmetin are reported to offer comparable pain relief.

PHYSIOTHERAPY MANAGEMENT-

PHYSICAL EXAMINATION-

A. SACROILIAC JOINT (SI JOINT)

There are certain simple tests, which may be positive if the SI joints is affected.

- a. Early signs include local tenderness over the SI joints and tenderness with paraspinal muscles spasm at lumbar and sacral vertebral levels.
- b. Provocative testing.
- c. Pelvic compression test.
- d. Pump handle test.
- e. FABER'S test.
- f. Gaenslen's test.
- g. SLR test.

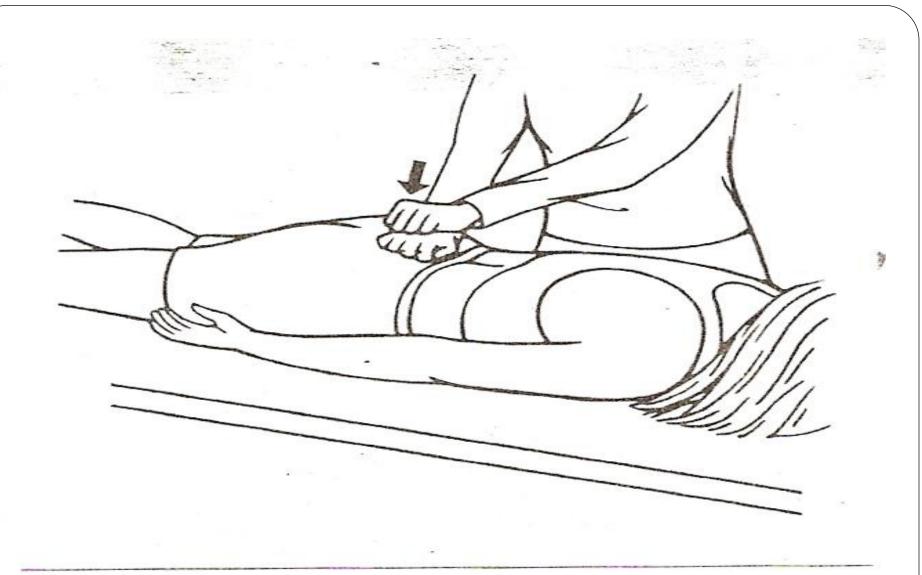


FIG. 56-4 Provocative testing of an inflamed sacroiliac joint may provoke pain by applying direct pressure over the sacrum with the patient lying prone.

BPT 8TH SEM

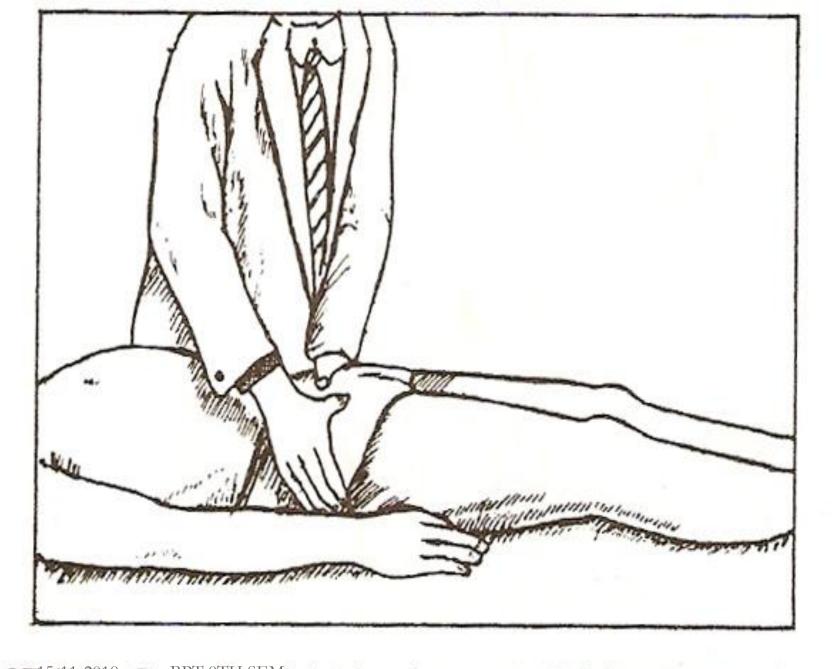


Fig. 27.17B: Sacroillac joint involvement: Pelvic compression test

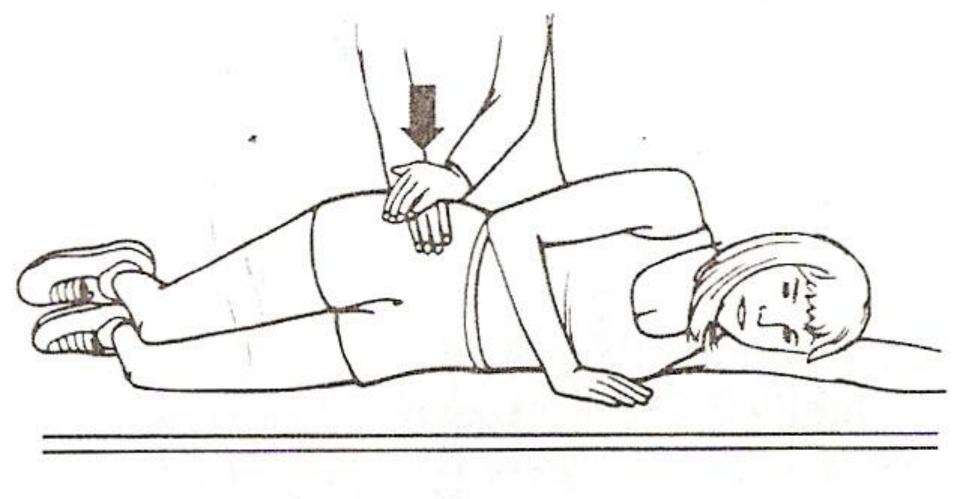


FIG. 56-5 The sacroiliac joint may be assessed by lateral compression of the pelvis.

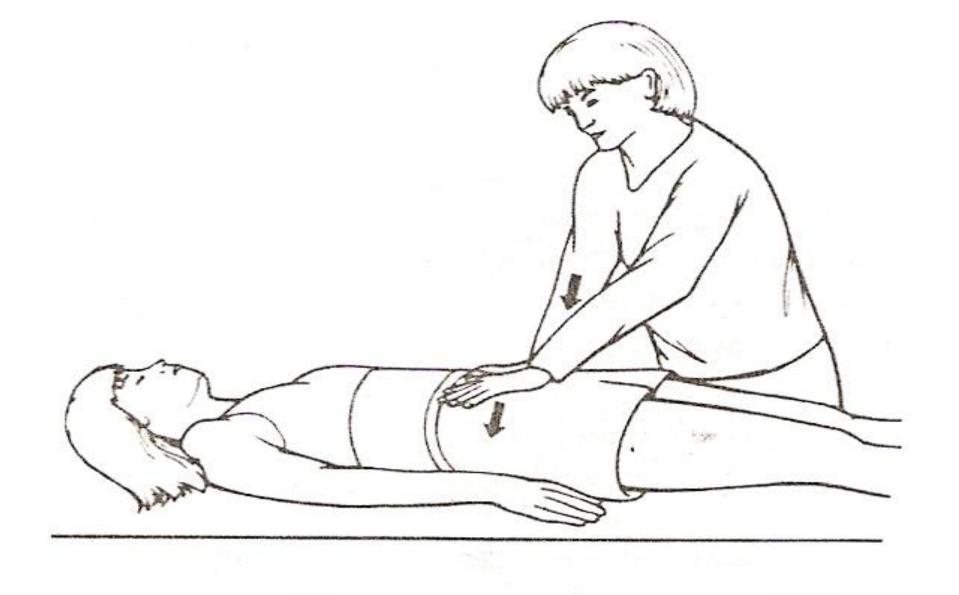


FIG. 56-6 Pelvic springing may be performed with the hands over the antellograsuperforming may be performed with the hands over the antellograsuperforming may be performed with the hands over the antellograsuperforming may be performed with the hands over the antellograsuperformed with the hands over the antellogram as the patient lies supine.

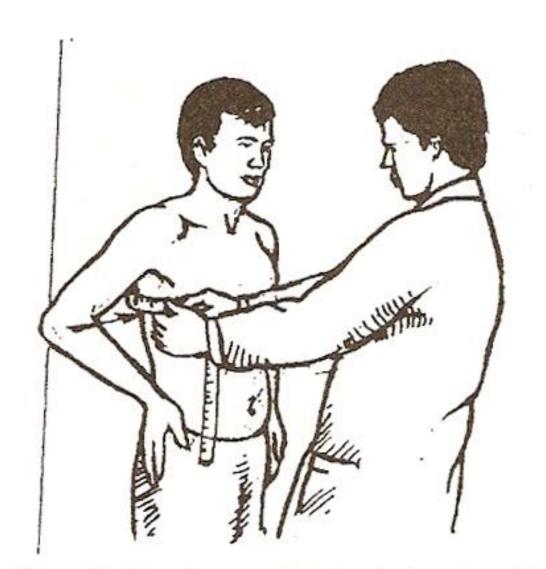


Fig. 27.16: Assessment of chest expansion in ankylosing spondylitis

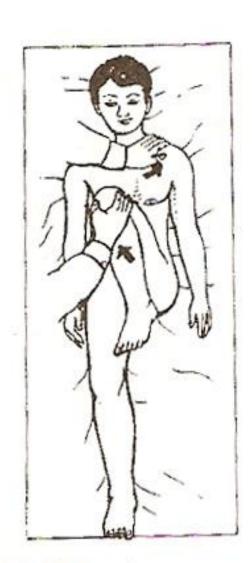


Fig. 27.17A: Sacroiliac joint involvement: Pump handle test

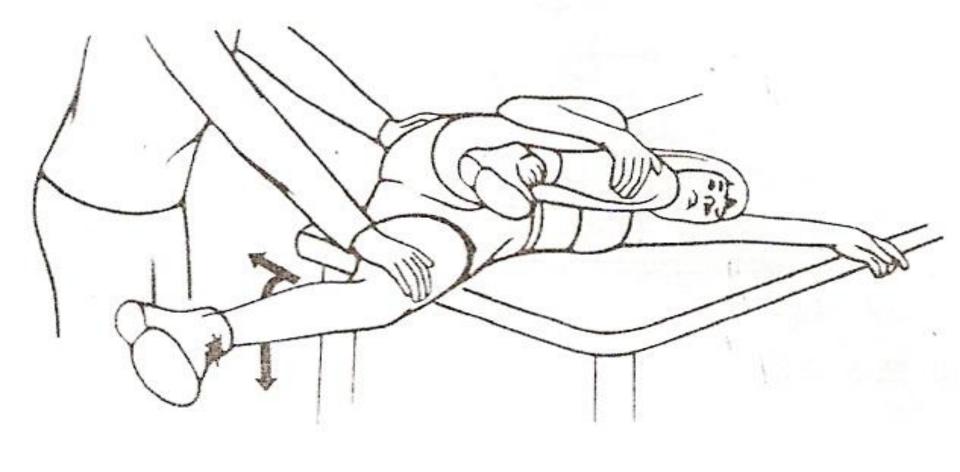


FIG. 56-7 Gaenslen's test. The patient lies supine over the edge of the examining table with the knees flexed and with one buttock over the edge. The patient then drops the unsupported leg off the table. This maneuver elicits pain in the contralateral sacroiliac joint by stretching it.



Fig. 27.18: BPT 8TH SEM Sacroiliac joint involvement: Fabre test

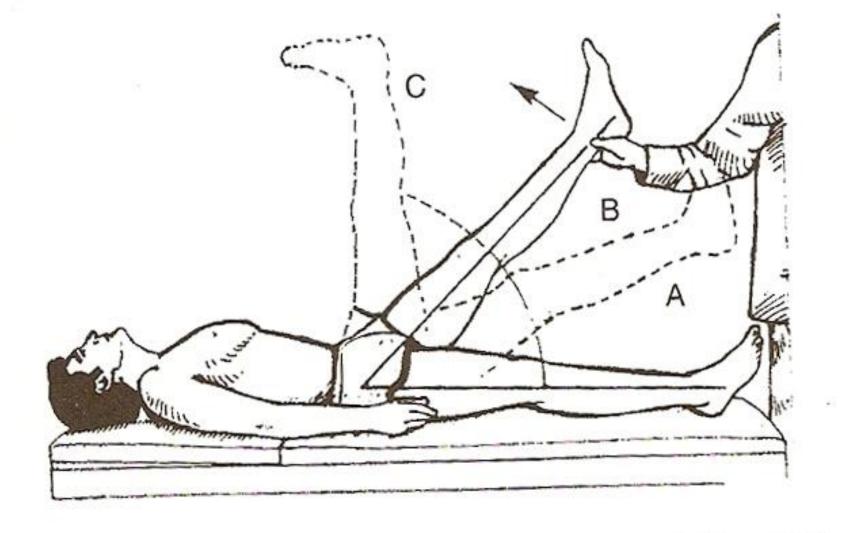
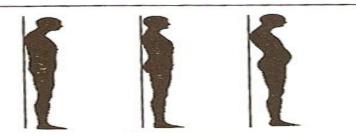


Fig. 18.10: Interpretation of SLR test (passive) (A) Upto 30°, sciatic nerve not under stretch, (B) Between 30-70°, nerve is stretched by prolapsed disc, hence positive (C) Pain between 70-90°, is not due to disc prolapse but due to S1 joint arthritis

- B. SPINE- Loss of spinal motion (lateral flexion, forward flexion and extension) can be detected quite early in most cases and several manoeuver can be employed to detect and then follow such changes.
 - a. Fleche test
 - b. Thoracolumbar flexion extension
 - c. Lateral flexion(on both sides)
 - d. Thoracic movt.
 - e. Combined hip and spinal flexion.



Inspection With the patient in standing position look for postural abnormalities.

The figure from left to right depict thoracic hyperkyphosis, normal spine and exaggerated lumbar lordosis (Fig. 18.11).

Fig. 18.11



Fig. 18.12A

Fig. 12B



1. To test flexion

Instruct the patient to bend forwards as much as possible at the waist.

Normal flexion is 80° or fingertips 3 to 4 inches from the floor (Figs 18.12A and B).



Fig. 18.13



Fig. 18.14

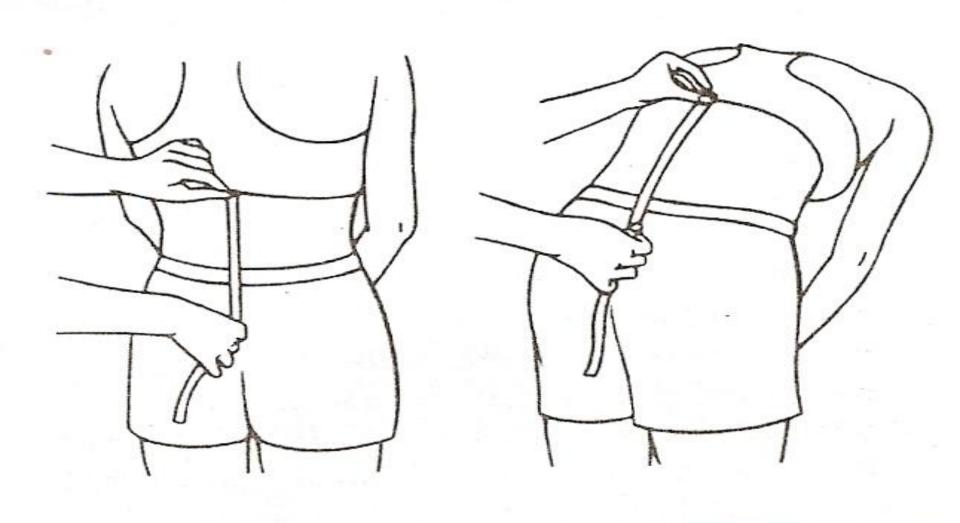
2. Lateral flexion

Instruct the patient to bend to the left and to the right as far as possible.

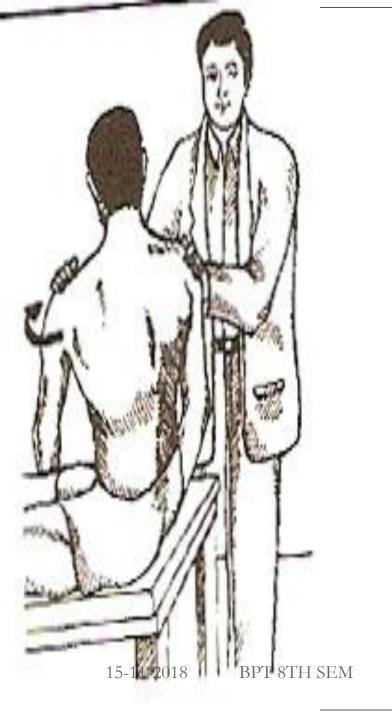
Normal range is 35° on each side (Fig. 18.13).

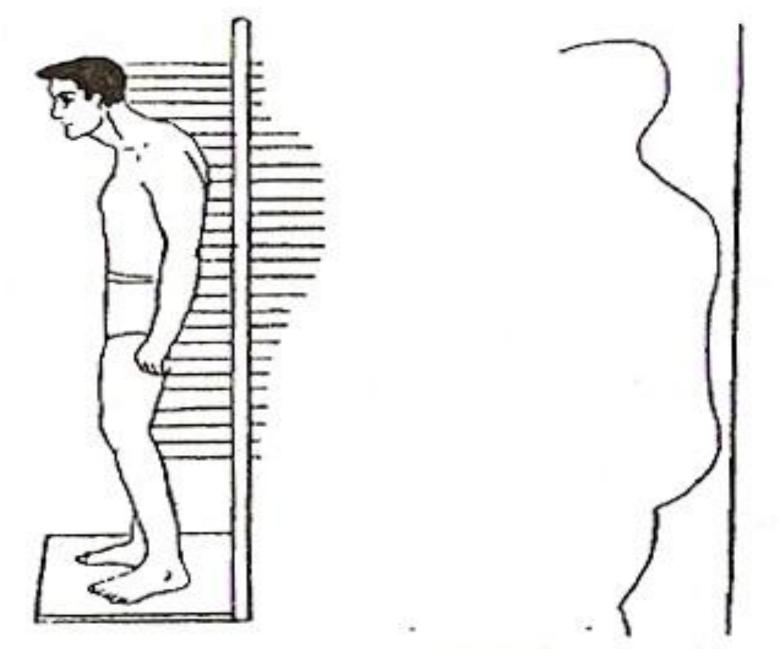
3. Extension

Instruct the patient to bend at the waist as far backward as possible. Normal range is 20 to 30° (Fig. 18.14).



15-11-2018 FIG. BP56 SEM easurement of forward flexion.





BPT 8TH SEMSPONDYLOMETER

- The patient stands on the wooden base with his back to the upright, care being taken to ensure that of each measurement the medial malleoli of the tibia are at a fixed distance and knees as straight as far as possible. So in most erect posture can be assumed.
- The vertebral spinous processes are central over the tip of the rods. The rods are then adjusted so that they just touch the spinous process. There are some fixed or important areas where the line of measurement is passed, they are bony points.
- The distance of the pioteruria of the rod from the upright are plotted on the graph paper. By the plotting of the anterior posterior curves of the spine, we get the spondylograph.

C. EXAMINATION OF PERIPHERAL JOINT INVOLVEMENT- the jts. are examined for active and passive range of motion, done with the help of goniometer. Commonly involved jts. are hip, knee and shoulder.

D. MEASUREMENT OF POSTURAL DEFORMITY- there are marked visible deformity present at the spine. In this, loss of the secondary spinal curve occurs, 1st the lumbar lordosis is lost and then the cervical lordosis and increase of the thoracic curves which results in rounded spine.

A simple instrument known as <u>SPONDYLOMETER</u> is used to the deformity.

PHYSIOTHERAPY MANAGEMENT-

Regular physiotherapy is very essential in the management of a patient of AS and only physiotherapist is the person who can help the patient to fight with the disease.

AIMS OF PHYSIOTHERAPY TREATMENT-

- 1. To control pain.
- 2. To maintain the mobility of the affected joints.(spine, thorax etc)
- 3. Increase the chest expansion and vital capacity.
- 4. To prevent or correct the deformity.
- 5. Postural correction.
- 6. To maintain and improve the physical endurance.
- 7. General advice to the patient.

- **REHABILITATION IN THE ACUTE STAGE-**
 - A. GENERAL EXERCISES
 - **B. BRETHING EXERCISES**
 - C. POSTURE CORRECTION
- A. <u>GENERAL EXERCISE</u>- Rest is essential, but as soon as the pain has subsided with the use of phenylbutazone, exercises in sling suspension, mat work and hydrotherapy are started.
 - At this stage no resistance is given. The warmth and the buoyancy of the water are invalkuable in encouraging gentle activity.
 - Slings can also be used to achieve either passive or active stretching of different muscle groups. This helps to prevent shortening and consequent deformities.

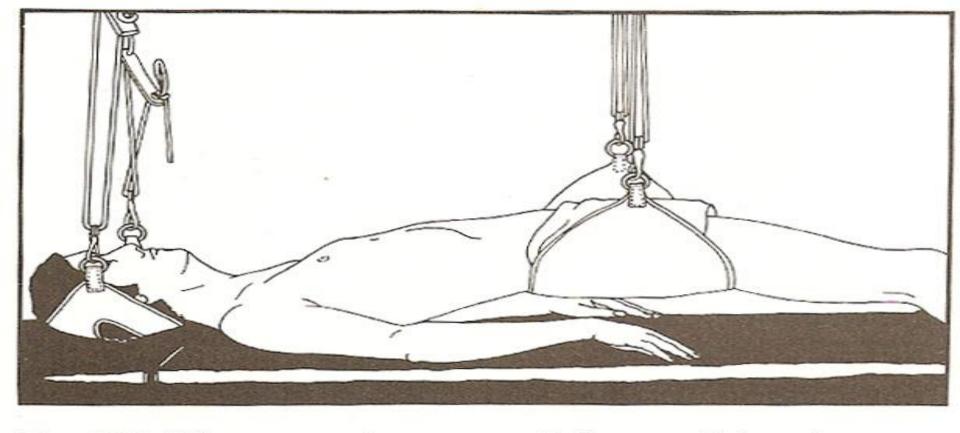


Fig. 17.1 Sling suspension to stretch flexors of the spine. should should relaxation techniques be taught in total suspension but the patient should be taught how to relax on the mat or in the gymnasium, as well as in bed.

B. BRETHING EXERCISES- breathing exercises should be practised at all stages of the treatment and are prescribed not only as a means of maintaining or improving vital capacity but also to help the mobility of the joints of the thorax. Diaphragmatic, abdominal and segmental breathing are taught.

C. POSTURAL CORRECTION- All postural training should be preceded by a warm-up phase of activity when the patient is given mobilizing and stretching exercises in order to prepare his muscles and joints to adapt to the new position they may be expected to adopt. mobility exercises should be of such a nature that the demand a maximal range of the movement from the area s being mobilized.

- The fundamental starting positions we use(Colson,1956) are crook lying, lying feet pressed against wall, back support kneeling, kneeling, standing. It is not always easy for the patient to adopt some of these positions but by slightly modifying them it is usually possible to get a resonable starting position.
- Once a satisfactory starting position has been achieved isometric contraction are taught for gluteal and abdominal muscles.
- Pelvic tilting is then practised to obtained and hold the correct inclination, then sub-costal opening, each position being held for a few seconds. Scapulae are placed in good position by outward rotation of the shoulders. Each position is held for a movt. or two before relaxaing. Finally the patient attempt to press the back of the head against the mat without making a double chin or without pointing the chin.

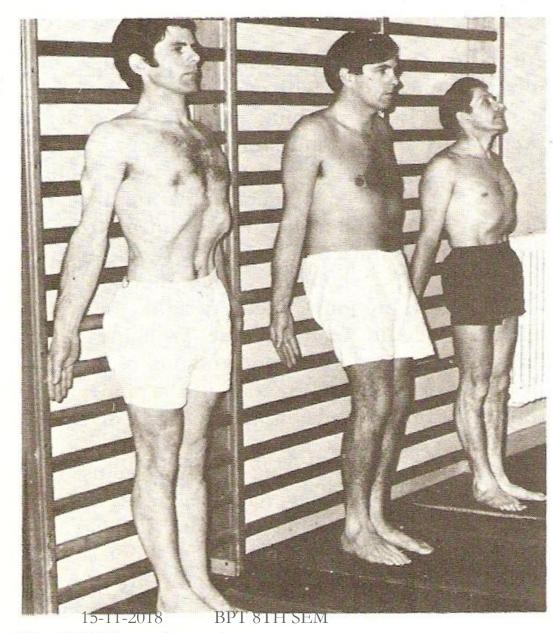


Fig. 17.2 Postural exercise.

REHABILITATION IN THE SUBACUTE AND CHRONIC STAGE

- A. GENERAL EXERCISES
- **B. MOBILITY EXERCISE**
- C. HYDROTHERAPY
- A. GENERAL EXERCISES- in this stage strengthening exercises are added and breathing, mobility and postural exercises are continued. Games or recreational activities play an important part at this stage, e.g badminton, relay races.
- B. MOBILITY EXERCISES- all the free exercises for all the joints esepcially shoulder, hip spine should be done in this phase along with thoracic cage mobility exercises.

STRENGTH-BUILDING EXERCISES (in pairs using 3-foot ash sticks) Starting Position Exercise 'Coffee grind' 1 Face partner holding one stick in each hand 2. Face partner holding Pull and push one stick in each hand 3. Face partner holding Stick wrestle same stick Stand back to back Rotate to pass stick to a vard apart partner. Rotate in opposite direction to receive stick Extend trunk 5. Lie prone with partner supporting ankles. Hold stick behind neck 6. Lie on back with partner Sit up to reach stick supporting ankles. Hold to feet stick above head Lie on back with partner 7. Chin the stick straddled over you. Both hold stick. 8. Lie on back with partner Span (arch trunk) kneeling above your head facing you. Both hold stick 9. Lie on side with stick Bend trunk to side held behind neck and partner supporting ankles 10. Row Sit opposite partner both with legs straight and apart. Both hold stick 11. Sit back to back with Bend forward to pull partner both with legs partner into span

straight and apart. Both hold stick above head

stoop and with legs apart. Hold stick with partner supporting it

Warm up

Hold stick in both hands Hold stick in both hands

Stand with forward

12.

2. 3. Stand with slegs apart SEM and stick behind neck Stand with legs apart

and stick behind neck

(arched trunk) position

Press trunk towards floor

and backwards

side (lateral flexion)

and upwards

Step over stick forward Swing arms forwards

Rotate from side to side Flex trunk from side to

	Starting position	Exercise
1.	Stand with legs apart	Swing arms downwards, forwards, and sideways
2.	Stand with legs apart	Swing arms forwards while rotating trunk sideways
3.	Stand with legs apart and arms on hips	Bend trunk forward from side to side
4.	Stand with legs apart	Bend forwards and try to touch toes
5.	Stand with legs apart and hands behind neck	Alternate knee raising and trunk bending to touch left knee with right elbow and right knee with left elbow
6.	Stand with feet together and hands by side	Swing arms forwards above head and alternate placing leg backwards
7.	Stand with hands behind neck	Alternate left and right trunk circling
8.	Stand with hands behind back	Alternate left and right head circling
9.	Stand with a lax stoop	Press arms backwards
10.	Sit with legs straight and apart and arms held sideways	Reach to touch opposite toe with alternate hands
11.	Sit with legs straight and apart and arms held sideways	Rotate trunk from one side to another
12.	Kneel with trunk prone	Tilt pelvis
13.	Kneel with trunk prone	'Pluto' sniff
14.	Kneel with trunk prone	Klapp crawl*
15.	Kneel with trunk prone	Raise and lower opposite arm and leg and alternate
16.	Kneel with trunk prone	With left arm under chest swing arm to the left

with trunk rotating.

Repeat using right arm

^{*} Klapp crawls are a series of exercises done in the horizontal kneeling position. Specifically, they are used as a treatment for spinal defects, especially scoliosis. (It is said that Dr Rudolf Klapp [1873–1949] thought out these exercises while observing his dog's back when it was walking.)

C. HYDROTHERAPY- Hydrotherapy, in the real sense refers to the therapeutic use of water. Recreational activities in water are a good method of encouraging movement whilst distracting the patient mind from his condition, and such pursuits as swimming and playing games with floats should be practiced.

Therapeutic effects of use of water is related to-

- a. The relief of pain and muscle spasm
- b. The maintance or increase in range of motion of the joints.
- c. The strengthening of weak muscles and an increase in tolerance to exercise.
- d. The improvement of circulation.
- e. The maintance and improvement of balance, co-ordination and posture.

USEFUL SPECIFIC EXERCISES IN THE POOL

Shallow end

55	Starting position	Exercise	
1.	Kneel with trunk prone and hands on steps	Keep hands still, lower hips to steps and raise to	
2.	Kneel with trunk prone and hands on steps	Alternate raising legs to touch chest with knee	
3.	Kneel with trunk prone and hands on steps	Alternate raising legs back- wards (hip extension)	
4.	Kneel with trunk prone and hands on steps	Combine exercises 2 and 3	
5.	Kneel with trunk prone and hands on steps	Raise and lower alternate arms	
6.	Kneel with trunk prone and hands on steps	Raise and lower opposite arm and leg	
7.	Lie prone on steps with hands on top step	Raise and lower alternate legs. Raise and lower both legs	
8.	Crouch with hands on steps	Keep hands on steps and stretch and bend legs	
9.	Lie on side on steps	Raise and lower top leg. Raise and lower both legs	
10.	Kneel with trunk vertical and legs apart with hands on hips	Rotate trunk from side to side	
11.	Kneel with trunk vertical and legs apart with hands by side	Bend trunk from side to side	
12.	Kneel with trunk vertical and legs apart with hand behind back	Repeatedly extend back	
13.	Stand with stoop and and hands clasping side rail	Press trunk to water and relax	
14.	Lie on back on steps with legs apart	Rotate trunk to cross legs over one another. Alternate	
15.	Lie on back on steps with legs together	Move closed legs side to side	

RECREATIONAL ACTIVITIES

Relay races

Draw braid over body*

Relay race Arch ball†

Relay race Tunnel ball‡

4. Relay race Alternate arch and tunnel ball

5. Relay race Pass ball with alternating trunk rotation

Minor games

 Standing basket ball shooting Two teams lined up same distance from basket. First to score 21.

- 2. Sitting volley ball
- 3. Padder tennis SEM

220 ANKYLOSING SPONDYLITIS

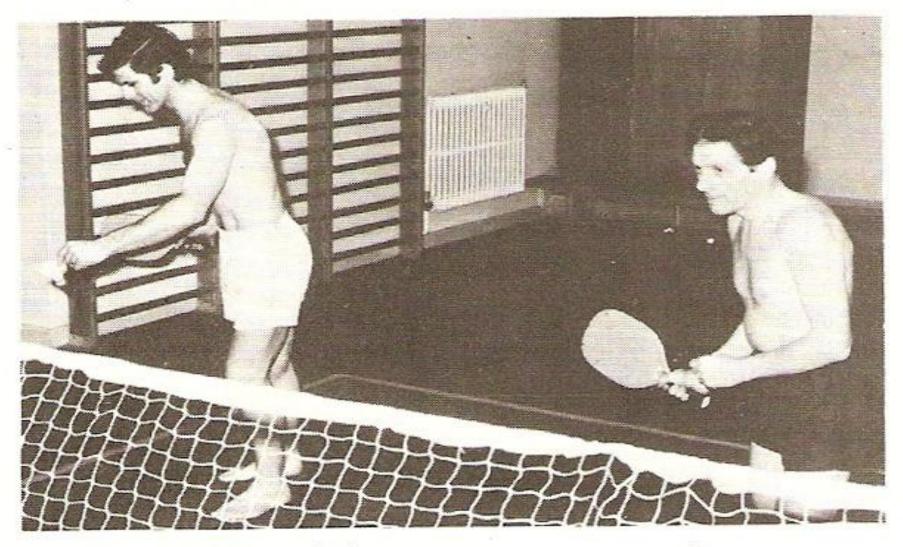


Fig.15-117203 Padder badminton.

EXERCISES RECOMMENDED FOR HOME USE Using towel Starting position Exercise 1. Stand with feet together Step over towel and back holding towel in both hands 2. Stand with feet together Swing both hands above holding towel in both head and down hands 3. Stand with legs apart and Rotate trunk from side towel held behind neck to side Stand with legs apart and 4. Bend trunk forwards and towel behind neck recover 5. Stand with legs apart and Flex trunk from side to towel held behind neck side (lateral flexion) Using dining room chair Starting position Exercise 1. Sit high on chair Bend forward to touch toes Sit high on chair with 2./ Alternate raising knee to hands behind neck touch opposite elbow 3. Sit high on chair with Flex trunk from side to legs apart side reaching down with hand Sit high facing back of Rotate trunk from side chair with legs apart to side and hands behind neck Lie with crooked back Drop knees alternately holding chair legs Lie on back with back Span (arch trunk) of wrist resting on chair seat 7. Lie on back with feet Attempt to sit up and resting on chair seat touch knees Lie on back with feet Press heels into seat of resting on chair seat chair and attempt to raise buttocks Stand facing chair 9. Alternate placing foot on chair seat. If strong enough do step ups Crouch with hands on 10. Stretch and bend legs. If chair seat necessary assist with arms. Hold on to chair throughout exercise

	Using dining room table or bed				
	Starting position	Exercise			
Į.	Lie on back with back and buttocks on bed or table and feet on floor	Flex hips by passive stretching			
2/	Lie prone with abdomen and hips on bed or table and feet on floors over	Alternate raising and lowering legs, then raise			
3.	Lie on side on bed with feet on floor and top leg forward	Raise and lower both legs			

ADVICE TO PATIENT OF AS-

- 1. The patient must do exercise everyday.
- 2. Patient is advised to sleep on a firm mattress with the use of small pillow or without pillow.
- 3. Spend some time in prone lying daily.
- 4. Always be conscious of posture, especially in leisure activities including sitting, for ex-reading, watching TV.
- 5. Pillow under the knees should be avoided for all time to avoid flexion deformity.
- 6. Avoidance of excessive spinal strain.

SURGICAL MANAGEMENT- In advanced and severe AS, causing painful deformities or loss of function, surgery may be needed.

The indication s to preceed to surgery in the individual patient must be clearly defined. These indications are-

- 1. Degree of deformity at any level which interferes with the function.
- 2. the presence of pain and encroachment on intra- thoracic and intra-abdominal viscera with associated ribs/ pelvis impingement, both sufficient to cause drastic impairment of general health.

- The most successful operation is restoring pain free movement to the stiffened hip jts. and make the quality of the life better. Total hip replacement of both the hip is done. The operation is called "Betroth operation".
- Total knee replacement, cervical and lumbar osteotomies to relieve severe spinal kyphosis, condylar resections of the ankylosed tempro-mandibular joints. And stabilization of atlanto axial subluxation have also been performed.
- While operating the spinal segments or vertebral column there may be the danger of causing neurological damage and may result in paraplegia or quadriplegia.

15. PROGNOSIS-

- Several long-term studies indicate that the prognosis of AS is good in the majority of the patients. Only 10 to 20% became significantly disabled over long periods, and 85 to 90% are able to pursue full-time employment, despite progression to severe spinal restriction in approximately one- half.
- A predictable pattern of the disease usually emerges after the 1st 10 years. Hip disease which typically begins early and in the youngest patients, is surest indicator of poor functional outcomes.
- Mortality from the disease itself occurs in less than 5% of the patients, most commonly from cervical fractures and dislocations, spondylitic heart disease, and amyloid nephropathy.

THANK YOU...