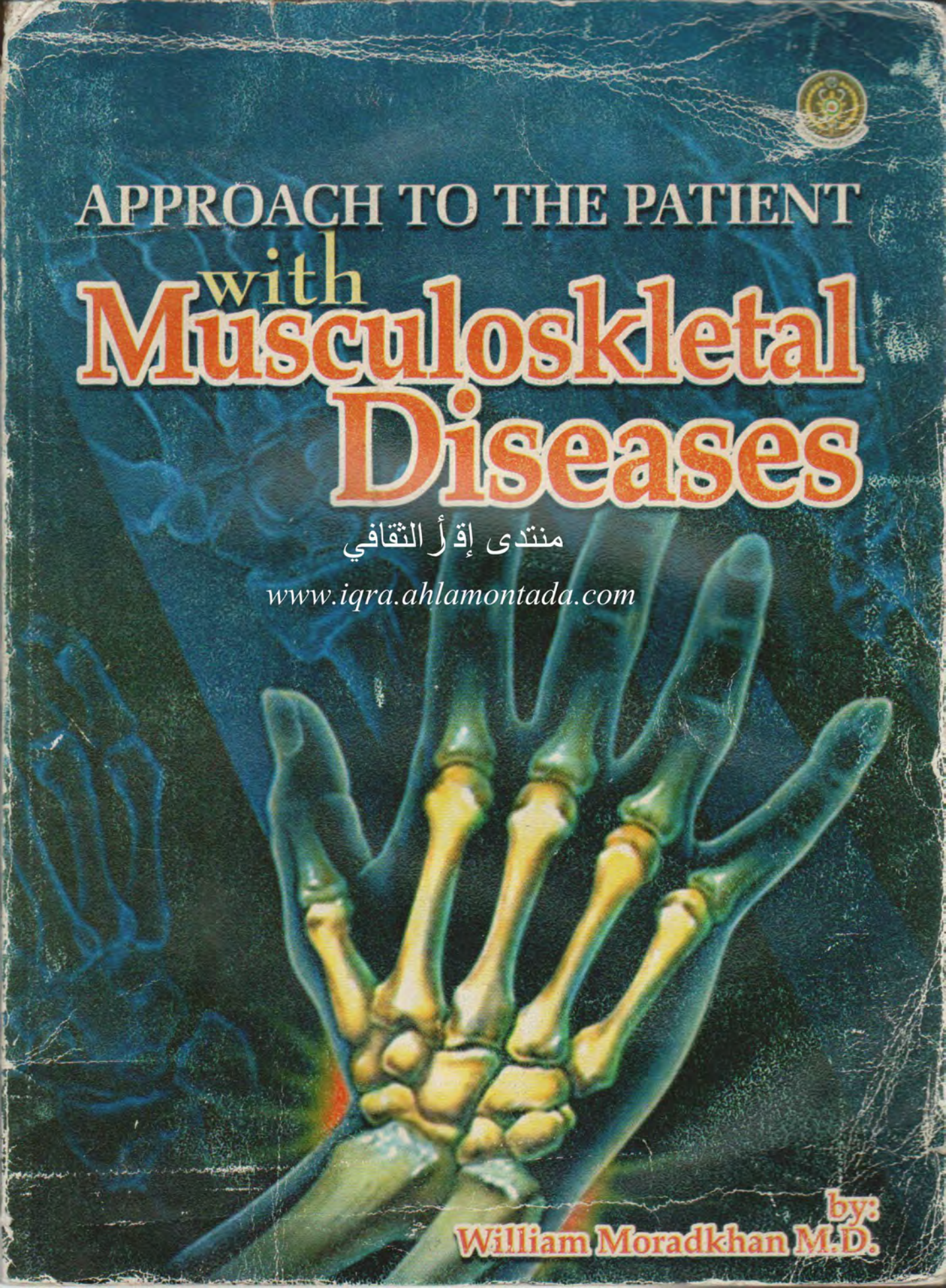
APPROACH TO THE PATIENT with Musculoskletal Diseases



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First Edition

Approach to the patient with MUSCULOSKELETAL DISEASES

Anthology by: William Moradkhan

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PREFACE

As it's title suggests, the aim of this book is to provide internists, family practitioners with concise practical information to approach the patient with musculoskeletal disorders by targeting steps.

Musculoskeletal problems are exceedingly common in clinical practice. The frequent combined involvement of musculoskeletal and other systems in disease requires all doctors to have a certain basic level of rheumathological expertise.

To approach the patient step by step the history and physical examination skills are there for of paramount importance. Such a major emphasis on clinical skills is in keeping with the current philosophy on medical education.

William Moradkhan, M.D.

INTRODUCTION

Screening of the locomotor system should be included in any full general medical examination: many rheumatic diseases involve other systems and, conversely, many 'general medical' conditions (particularly endocrine, metabolic, and neoplastic) affect locomotor structures.

The present book, which is an anthology of several books and articles on Rheumatology, has been compiled in 5 chapters. Chapter 1 consists of two parts. The first part includes a detailed account of a goal-oriented approach to the rheumatic diseases. In the second part, a number of commonly observed patterns and their matching conditions are listed.

Chapter 2 gives a more detailed information (basic anatomy, pain patterns, method of clinical examination) to enable the determination of arthicular and Periarthicular problems at individual region.

In Chapter 3 the diagnostic data and the key points of a series of rheumatic diseases (connective tissue disorders) are briefly introduced.

Chapter 4 includes the diagnostic data and key points of a number of muscoloskeletal diseases which are localized in the bones.

Chapter 5 introduces the diagnostic data and key points of several painful syndromes, related conditions and miscellaneous disorders.

CHAPTER I

APPROACH TO THE PATIENT WITH MUSCULOSKLETAL DISEASES

The twentieth century witnessed great advances in the field of medicine. During this period, especially in the second half of the century, many health problems were overcome. Many contagious diseases were completely eradicated; others were controlled to a large extent. However, the Musculoskeletal disorders, which are the most common cause of the patients' visit to medical centers, were not efficiently dealt with. The significance of Musculoskeletal diseases may be recognized if we noticed the fact that the World Health Organization (WHO) has designated the first decade of the 21st century as the decade of campaign against such diseases.

Until not long ago, especially in the developing countries, little was known about the physiopathology of Musculoskeletal disorders. As a result, all the Musculoskeletal diseases, which consist of over 150 known varieties, would be considered either as Arthrosis or Rheumatism.

In the last two decades of the last century, research has made great contributions to our understanding of the physiopathology of these diseases. The result of these astounding advances were such that if appropriately approached, Musculoskeletal diseases may now be diagnosed and controlled easily before they can cause irreparable damage.

The Musculoskeletal disorders can be classified under three categories. The first group are those that are localized in a limited part of the Musculoskeletal system. The second category include the diseases that involve different parts of the system and cause functional disorders in the affected tissues. The third category goes beyond the Musculoskeletal system and affects other systems such as the cardiovascular system, respiratory system, the peripheral and central nervous systems, kidney, skin and the eyes. However, it should be born in mind that even a systemic disease might have a local manifestation in its early stages (such as Behcet's disease).

Usually the second and especially the third type of the above disorders would be accompanied by some constitutional symptoms such as fever, exhaustion and weight loss. In these

categories the joints may be involved in the form of Oligo-arthritis or Poly-arthritis. The complications may remain in the peripheral joints or may involve the axial joints as well. A kind of these diseases only involves the sinovial joints, another group causes complications in the small and larg joints in a symmetric manner. In some varieties of these diseases along with the joints, the adjacent tissues are also damaged.

The initiation of Musculoskeletal diseases may also take different forms. Some of the forms involve acute outbreaks of disease in which the symptoms appear in a matter of hours or days (such as in gout). Other forms develop gradually in a period of 6 weeks to 6 months.

The course of development in Musculoskeletal diseases is also of great importance for their diagnosis. Some of these diseases may cause damages to the involved tissues in a short period of time (e.g., Septic Arthritis). In other forms it takes longer for the disease to induce notable complications (e.g., Rheumatoid disease). Some of these diseases may not cause damages to the tissues even if they are left untreated. Still other forms may become intermittent (e.g., Mediterranean fever and rheumatic fever). The Musculoskeletal diseases may even become systemic (i.e., one or more organs other than the Musculoskeletal system may become involved) sometime in the course of development. Often, the type or the severity of the involvement in the non-Musculoskeletal organs will provide the clue for the diagnosis as well as the Prognostic and therapeutic protocols.

Since the symptomatic treatments (before the diagnosis) will often alter the natural course of the disease, it is recommended that the patients avoid medication before the diagnosis. The reliable clinical and epidemiological findings can help us to approach the disease through logical goal-oriented steps. There are various methods of approaching the patient, most of which are intuitive. One of the conventional methods will be introduced below.

After the patient's details (Name, age, sex, profession, and address) are recorded, the following points will be considered in the history of the disease:

1. the patient's main complaint(s),
2. the description of disease from the outset,
3. the previous diseases or patient's health condition ,
4. The health record of the close relatives,
5. The condition of other systems,
6. Systematic physical examination of the patient,
7. Diagnosis or the distinctive diagnosis (following the consideration of the symptoms and other data).
8. Paraclinical examinations.

This approach has some disadvantages. First, in this approach the questions in the history of the disease are not goal-oriented. Also, through this approach usually a strong diagnosis cannot be made and thus the physical examination and the paraclinical decisions based on the diagnosis would not be goal-oriented.

There are three paraclinical diagnostic approaches. The first include the tests that are used as diagnostic criteria. These tests are sometimes useful in determining the severity of the

complications as well as the prognosis. The second category consists of the tests that are used to determine the severity or the activity of the disease. The third group of tests is used to decrease or reject the feasibility of a certain disease. A major disadvantage of the latter approach is that the various tests carried out are not purposeful. This will result in the familiar scene of patients whose files are loaded with various tests and x-rays.

The approach adopted here (the method of approaching the patient in successive goal-oriented steps) is explained below.

The complaints of the patient lead us to the primary data, which in turn will be processed in the next stage by asking the following questions:

- When did the symptoms begin?
- How did the symptoms begin?
- How did the disease develop since your first symptoms?

Next, the processed data is synthesized, and that often takes us to one or two strong diagnoses. A simple example can help us to better understand the issue:

PART ONE

The patient is a 35-year-old housewife. She complains from the pain in the small joints of both hands, pain in both wrists and the left elbow as well as the pain in the knees and the small joints of the lower limbs. She reported that the pain had gradually begun from two months ago. It started from the upper limbs in a symmetric manner and had slowly developed into the lower limbs. The pain was more intense when she relaxed, especially in the mornings she was stiff for a couple of hours. She was counseled by the friends to take Diclofenac for 10 days, which had alleviated the pain.

According to the above data we have a female patient who is in the age of fertility and is suffering from a chronic rheumatic disease with the symptoms in the upper and lower limbs especially in the small joints in a symmetric manner. In approaching a chronic illness we should realize the way it began, its course of development, the complications it has caused, and the effect of the medication. This will help us to adopt the most appropriate measures to approach the disease not only in the diagnosis and treatment stages but also in proposing an appropriate lifestyle for the patient, including the job and family issues. Now, to conclude this part of the observation, the synthesis of the data with high sensitivity and specificity will lead us to the following possible diagnoses.

1- Rheumatoid arthritis (RA)
2 -Systemic lupus erythematosus (SLE)
3- Certain drug reactions
4- Psoriatic arthritis
5- Gout and pseudogout
6- Brucellosis (in endemic areas)

Comments: Chronic symmetric polyarthritis in hands and wrists in over 80% of cases represents RA. SLE may also present in this fashion. If the patient is taking procainamide, a beta-blocker, or quinidine, a drug reaction should be at least considered. Preceding, concurrent, and familial psoriasis should be noted and examination should include areas in which psoriasis may be hidden.
Remember that both gout and pseudogout may present with a pseudo-RA pattern.
Brucellosis caused by *B. melitensis* occurs frequently in endemic areas (Latin America, the Middle East, and Mediterranean countries). Musculoskeletal manifestations occur in over 50% of patients including rheumatoid-like peripheral arthritis or a spondylitic condition with unilateral Sij involvement.

For the diseases set forth so far in the observation, depending on the sensitivity and specificity level of the data, the following diagnostic scores can be taken into consideration:

1. **Rheumatoid Arthritis =80**
2. SLE =60
3. **Drug reaction =50**
4. **Psoriatic arthritis =55**
5. **Gout** and Pseudogout =50
6. Brucellosis in the endemic areas =50

PART TWO

In this part of the observation the investigation of the data will be goal-oriented.
In the case introduced above, the data concerning the past health and the family history does not support any of the above diagnoses.

Review of Other Systems:

- General conditions: the patient complains from exhaustion, fatigue, irregular fevers and weight loss (3 kilos in about 2 months).
- Skin and hair: during the last month her hair loss has intensified. She has taken antihistamine drugs due to an allergic bout of widespread rash. Also, since 3 months ago she has been using sun lotion because of sensitivity to the sunlight.
- Respiratory system: since last week she feels pain in her chest when she coughs, sneezes or breathes deeply. In the examination of other systems no evidence was found in support of any of the diagnoses.

Physical Examination:

- General: temperature: 38; pulse: 90 p.m.; breathing rate: 20 p.m.; blood pressure: 120/80.

- Skin and hair: there are hyper-pigmented and hypo-pigmented plaques on the chins and the base of the nose. There are erythematosus plaques on the extensor of fingers in the area of joint wrinkles. There is also considerable hair loss. No evidence of skin disease in the form of psoriasis was observed in the hidden areas.
- The Musculoskeletal system: the 3rd and 4th joints of PIP and MCP are inflamed without the signs of synovial hypertrophy in both hands. There is also sensitivity and limitation of movement in the wrists. There is arthralgias in the knees and the left ankle. There is no signs of involvement in the axial joints and inthesis.
- Respiratory system: deep breathing is painful. Listening to the lungs revealed friction rub in the left side of the thorax. In the examination of other systems no irregularity was observed.

Now, considering the goal-oriented data from the second part of the observation, we may add the strong diagnostic criteria (the symptoms of skin involvement and Pleural involvement) to one of the diagnoses (SLE).

The interpretation of all the data gathered so far from the first and second part of the observation, leaves room for only the following two diseases:

1. Systemic loopus with a score of 85
2. arthritis rheumatoid with a score of 60

PART THREE

In this part (the paraclinical measures) approaching the Musculoskeletal patient would be quite purposeful. Generally speaking, the laboratory tests can be helpful in one of the following goals:

1. Diagnosis of the disease,
2. A general evaluation of the activity of the disease,
3. An evaluation of the activity of the disease in a specific organ,
4. An assessment of the markers that determine the prognosis of the disease, and
5. An investigation of the effects of the medication (both therapeutic and side effects)

In order to achieve the above 5 goals the following paraclinical findings were used:

a. Biological tests: (W.B.C: 4000 with Lymphocytopenia, normo-chrome and normo-cyte anemia, and platelets count 8000).
b. Immunological tests (titer of the anti-nuclear anti body 3 times above the normal level. C3 and C4 had decreased).
c. Serological tests (ESR: 65, CRP++, RF+)
d. Urinalysis: the protein level of urine in 24 hours was 800 milligrams.
e. Chest X-ray reveals the signs of pleuritis in the left side.
f. Biopsy of the kidney shows the defuse proliferative glomerolonephritis.

The paraclinical results and all the symptoms and the data gathered so far from the observation and physical examination lead us to the diagnosis of SLE.

DISCUSSION AND CONCLUSION

Four centuries ago Francis Bacon said, anyone who wants to serve science should go and record all the facts that he has observed. He also used the metaphor of grapes for truth, which when compiled, the 'wine of knowledge' will flow. In the past century both the philosophers and scientists realized that a lot of the facts are useless or even misleading. The advancement of knowledge will not be achieved unless the facts are approached selectively in a purposeful plan. Haphazard compiling of different grapes, sour and sweet, will result in a 'bitter vinegar' instead of 'sweet wine'.

In the purposeful method of approaching the Musculoskeletal diseases, elucidated above, it was suggested that we should isolate the useful facts drawn from the first part of the observation and sketch the plan for hypothetical diagnosis of one or two diseases. This sketch will be used as a basis on which the other set of relevant data from the other two parts (observation, physical examination and the paraclinical data) would lie.

Pattern Recognition in Rheumatology

ACUTE ARTHRITIS (painful swollen warm joint)

- Septic arthritis
- Gout
- Pseudogout
- Pseudoseptic arthritis in rheumatoid or reactive arthritis
- intraarthicular fracture
- Hemarthrosis

Comments: The main move here is to aspirate the joint, handling the fluid as in synovial fluid analysis. A bloody SF means Hemarthrosis but gout and pseudogout may also produce Hemarthrosis. These should identified on routine polarizing microscopy. If no crystals are present place the hemoragic fluid in the refrigerator. Fat layering on the top (just like chicken soup) raises the possibility of intraarthicular fracture. A high leukocyte count renders the SF turbid in bacterial infection, gout, and pseudogout and Pseudoseptic arthritis. Presence of MSU and CPPD crystals indicates gout and pseudogout, respectively, but does not rule out concurrent bacterial infection. If no crystals are found, likely diagnosis is septic arthritis and pseudoseptic arthritis. Make sure a gram stain is done (and exhaustively inspected), and request anaerobic and aerobic cultures. In acute arthritis you must move fast but it helps to first go through the steps mentally. For example determine if you know how to enter that joint, if all of the required materials are there including two pairs of gloves, a lot of sterile gauze, extra syringes, thin and large-bore needles, and a sterile rubber stopper (to submit the syringe to bacteriology) that may not be in the try. As important as submitting the fluid is to personally follow it through or have someone in the laboratory you entirely trust to follow it through.

ARTHRITIS, FEVER, AND A RASH

- Rubella
- Parvovirus B-19
- Gonococcemia
- Meningococcemia
- Lyme borreliosis
- Secondary syphilis
- Adult acute rheumatic fever (ARF)
- Adult Kavasaki disease?
- Vasculitic urticaria
- Acute sarcoidosis
- Adult Still's disesae
- Familial Mediterranean fever (FMF)
- Hyperimmunoglobulinemia D and periodic fever syndrome

Comments: Small-joint arthritis, an erythematous macular and papular rash that starts in the face and generalizes within 24 hours, plus retroauricular and posterior neck nodes characterize rubella arthritis, which is particularly common in young women following immunization.

In parvovirus B-19 infection, which is a cause of polyarthritis in adults, the typical "slapped cheeks" rash seen in children is replaced by inconspicuous macules affecting the face or elsewhere. There may be several "lupoid" features to parvovirus infection including thrombocytopenia, positive ANA, and hypocomlementemia. Fortunately, these are only transient and few people are so tested. An imprudently requested ANA may raise great concern. Gonococcal arthritis, particularly polyarticular febrile cases, feature erythematous macules that evolve into hemorrhagic/necrotic pustules. Few lesions are usually present and they tend to involve the upper extremities. A rather similar clinical pattern occurs in meningococcemia, but here the rash consists of erythematous/petechial papules, sometimes confluent, that may present anywhere. Remember that acute meningococcemia and certainly chronic meningococcemia may not feature meningitis. The typical erythema migrans of Lime borreliosis is single erythematous maculae or papule that expands gradually, as it clears in the center, to a diameter of 15 cm or more. There are minimal constitutional symptoms at this stage. The true arthritis of Lyme borreliosis comes later with early generalized or late disease. The possibility of secondary syphilis should be raised when subacute arthritis associates with brownish red papules in the trunk or scaling papular lesions on palms and soles. The rare erythema marginatum of ARF is striking in its appearance: evanescent rashes in which individual lesions expand centrifugally while the center clears. A hot shower makes EM lesions striking. EM has been aptly compared by Taranta to smoke rings, such is its dynamism. Kawasaki disease is a condition of young children,

Although a few adult cases have been reported. There is a high fever, a polymorphous rash in the trunk that may disseminate, palmar and plantar erythema, brawny edema in the dorsum of hands and feet, a strawberry tongue, conjunctival irritation, and cervical adenopathy. Myocarditis in early disease and coronary artery aneurysms and myocardial infarction account

for most of the mortality in Kawasaki disease. Arthritis presents in the recovery period and affects large weight-bearing joints.
Vasculitic urticaria is another condition that features arthralgias or arthritis, fever, and a rash in which individual urticarial lesions persist for 24 hours to 3 days; An associated angioedema is frequent.
The typical skin lesion in acute Sarcoidosis is erythema nodosom. Skin inflammation may be intense, resembling cellulitis.
The last three conditions listed are intermittent conditions with several overlapping clinical features. Adult Still's disease is seen predominantly in young adults. The condition features high spiking fevers with a rapid return to baseline or below baseline once or twice daily, a discrete salmon-pink macular rash accompanying the fever with elements from 2 to 5 mm in diameter, predominantly in trunk and proximal extremities, and lymphadenopathy. There is high leucocytosis and the ESR is markedly elevated. Whereas in children systemic findings precede the arthritis by weeks or months, adult cases of Still's disease feature concurrent arthritis. Different from childhood cases, adult Still's disease has no association with Amyloidosis.
Familial Mediterranean fever (FM F) is an intermittent condition that affects individuals from Mediterranean ancestry and has its onset in late childhood or adolescence. The rare but clinically striking skin lesion of FMF is a very painful, angry-looking pseudoerysipela involving the distal legs. These lesions must be distinguished from the recurrent erysipelas that complicates congenital lymphatic channel hypoplasia. Characteristic manifestations in FMF include fever, large-joint arthritis, serositis, and peritonitis. Leucocytosis and ESR are markedly elevated. Untreated (colchicine), many FMF patients develop amyloidosis.
Hyperimmunoglobulin D syndrome is a rare recently described condition so far identified in Europe that begins in early childhood and features fever, lymphadenopathy, a macular or papular rash in the extremities, abdominal pain, and Arthralgias or arthritis of large joints. As in the previous two entities, leucocytosis and ESR are high. An elevated serum IgD (polyclonal) is characteristic of the condition. There is no association with Amyloidosis.

Palindromic rheumatism

- Palindromic rheumatoid arthritis (RA)
- Essential Palindromic rheumatism
- Crystal synovitis (gout,CPPD pseudogout, calcific periarthritis
- Lyme borreliosis, stages 2 and 3
- Sarcoidosis
- Whipple disease
- Acute rheumatic fever
- Reactive arthritis (rare)
- Adult still's disease
- Familial Mediterranean fever (FMF)
- Hyper immuonoglobulin D syndrome

Comments: Palindromic rheumatism is a fascinating condition in which different joints are affected in sequence, each becoming acutely inflamed for a few hours to a few days after which they once again become normal. It is indeed curious to see a joint that was normal 10

minutes ago and now is warm, tender, and progressively swollen, only to become normal a few hours later. Sometimes inflammation is extraarticular rather than arthicular.
Of a cohort of patients with Palindromic rheumatism, about half will have a positive Rf test. Of these, half will go on to develop typical RA within a few years; the others will continue experiencing Palindromic attacks.
Of the seronegative cases, some will turn out to have a definable process such as gout, pseudogout, and rarely basic calcium phosphate deposition disease. Sarcoidosis may feature Palindromic arthritis over a background of intrathoracic findings. Reactive arthritis occasionally features Palindromic arthritis. The condition is suggested by preceding enteric or urogenital infection, coexistent enthesopathy, as well as mucosal and skin changes.
Microbial conditions causing Palindromic arthritis include Lyme borreliosis and Whipple's disease. In the latter, relapsing arthritis may go on for years if not decades before lymphadenopathy, diarrhea, and weight loss develop.
Acute rheumatic fever differs from Palindromic rheumatism in that individual joints become involved every few days in an additive fashion plus the fever and cardiac findings.
Finally, the previously described adult-onset Still's disease, familial Mediterranean fever, and hyperimmunoglobuhn D syndrome are additional conditions that belong to the Palindromic rheumatism pattern. Thus, confronted with a patient with Palindromic rheumatism the task is, rather than applying a label, to identify the underlying condition. Joint aspiration is essential in these patients.

CHRONIC MONOARTHRITIS

- Mechanical derangement
- Neuropathic (Charcot,s) joint
- Rheumatoid arthritis (RA)
- Psoriatic arthritis
- tubeculous infection
- Foreign body synovitis
- Pigmented villonodular synovitis (PVNS)
- Synovial chondromatosis

Comments: Mechanically damage joints, for example osteochondritic knee or a knee that has suffered a complex meniscal/ligamentous injury, are typically unstable, exhibit coarse Crepitation, are chronically swollen from synovial proliferation and effusion, and swelling varies with the use of the joint. Radiographic changes are those of osteoarthritis with bony sclerosis, degenerative cysts, and asymmetric narrowing from cartilage loss. The erythrocyte sedimentation rate (ESR) is normal and joint effusions are typically noninflammatory with leukocyte counts of less than 3000/mm3 and a predominance of mononuclear cells.

In Charcot's joint, instability is greater, Crepitation coarser, and effusions often huge contrasting with a virtual absence of pain. Neurologic examination reveals neuropathy. Radiographically there is subluxation, lytic areas, and large ossified intracapsular bodies. It may resemble osteomyelitis or septic arthritis, but the ESR is normal and synovial fluid is noninflammatory with a variable amount of blood. Calcium pyrophosphate dehydrate crystals may be present.

In the remaining conditions arthroscopy and biopsy is the most efficient diagnostic procedure. Both RA and psoriatic arthritis may feature monoarthritis at disease onset look for psoriasis at hidden sites (behind ears, scalp, umbilicus, and intergluteal fold) and inquire about a family history of psoriasis. Synovial fluid is inflammatory in both conditions with WBCs ranging from 10,000 to 100,000 WBCs/ mm 3 and a predominance of polymorphonuclear cells.
The findings are similar in tuberculosis except that litic areas are often present on x-rays at initial evaluation.
Foreign body synovitis should be suspected from previous trauma such as a thorn puncture wound or kneeling on a cactus. Patients with PVNS have recurrent bloody or chocolate-colored effusions with WBC counts of less than 20,000/ mm3
Clinical and synovial fluid characteristics in PVNS and synovial hemangioma are often similar. Arthroscopy shows redundant, inflamed synoviom in rheumatoid, psoriatic, sarcoid, tubeculous, and foreign body synovitis. Histology allows identification of tuberculosis and Sarcoidosis. In foreign body synovitis the offending foreign material is usually identified in the synovectomy specimen. PVNS features marked synovial proliferation and tan-, yellow, or chocolate- colored nodules and villous proliferation. A histologic diagnosis of PVNs can only be accepted after mycobacterial and fungal infections have been ruled out.
Finally in synovial chondromatosis the joint is filled with cartilaginous bodies that are often ossified. The fluid is clear highly viscous and noninflammatory. Diagnosis and treatment are Arthroscopic.

ENTHESOPATHY

- Reactive arthritis
- Ankilosing spondylitis
- Psoriatic arthritis
- Bacteremia
- Viremia
- Fluorosis
- X- linked vitamin D-resistant rickets
- Etetrinate treatment
- Foestier's disease (DISH)
- fluoroquinolones treatment

Comments: Enthesis designates the insertional area of tendon into bone, enthesopathy is a disorder of the enthesis, and Enthesitis represents enthesial inflammation. Inflammation of the distal Achilles tendon, the plantar fascia at its attachment in the medial calcaneal tuberosity, and the Tibial insertion of the patellar tendon represent typical enthesitides and are seen in reactive arthritis, AS, and psoriatic arthritis. Associated findings in the former include Oligoarthritis and perhaps some of the mucocutaneous manifestations of the disease. In AS the patient will have spinal rigidity plus radiographic evidence of sacroiliitis. Patients with psoriatic arthritis and Enthesitis usually, but not always, have skin psoriasis. Interestingly, in viremias, such as in AIDS or hepatitis B, and bacteremias, such as in S. *aurous* infection, enthesial pain is frequent.
Certain metabolic disorders including vitamin D—resistant rickets, fluoride intoxication, and etetrinate treatment of psoriasis or severe acne often feature enthesial pain as well as ossification at insertional sites of tendons, ligaments, and joint capsule. Similar findings occur in DISH.

Finally, there is a little understood toxic effect of fluoroquinolones on tendons that includes pain, swelling, and sometimes rupture. The condition most often affects the Achilles tendons, but the Epicondylar insertion of wrist extensors (similar to tennis elbow) and the shoulder's rotator cuff may also be involved.

ARTHRITIS AND WEIGHT LOSS

- Severe rheumatoid arthritis (RA)
- RA with vasculitis
- Reactive arthritis
- RA or psoriatic arthritis or (AS) with Amyloidosis
- Cancer
- Enteropathic arthritis(IBD)
- HIV infection
- Whipple's disease
- Blind loop syndrome
- Scleroderma with intestinal bacterial overgrowth

Comments: When a patient with arthritis has severe weight loss, several possibilities are raised non-of them insignificant.

First, patients with severe RA may have a decrease in lean body mass of more than 10%. These patients, as any chronically starved patient, are at a risk of sudden death.

Patients with this degree of wasting may have systemic complications in particular systemic vasculitis manifested by palpable purpura, neuropathy or mononeuritis multiplex, or leg ulcers. Patients with uncontrolled reactive arthritis may enter a catabolic state that may take many months to recover from. Some of these patients, with joint findings overshadowed by wasting, adenopathy, and splenomegaly, may seem to have an evolving lymphoma until someone examine the joints, inspect the glans penis and the oral cavity, and request SI joint films, establishing the diagnosis. Another association of weight loss (unless the patient becomes nephritic) is secondary (AA) Amyloidosis complicating RA, psoriatic arthritis, and AS. I am not implying that Amyloidosis causes weight loss per se but rather that severe inflammation causes both the Amyloidosis (by sustained high serum levels of AA protein) and the weight loss.

Neoplasia, a well-known cause of weight loss, may under some circumstances cause arthritis, for example, in lymphomas. Thus, unexplained weight loss in an arthritic patient, particularly if adenopathy is present, should make the physician consider neoplasia. Another prominent cause of weight loss is inflammatory bowel disease, which in about 20% of patients lead to arthritis. There is well-known association between reactive and psoriatic arthritis with HIV infection. In some of the patients arthritis is recognized first. It is therefore important in the evaluation of reactive and psoriatic arthritis patients with progressive weight loss to consider a concurrent HIV infection.

Whipple's disease is rare, as attested by the author's experience of only 6 cases in a 30-year experience. Four of the six had a massive weight loss. All had arthicular symptoms but only two had diarrhea. The blind loop syndrome used to feature arthritis in addition to weight loss.

Finally, patients with scleroderma, a condition that may often cause arthritis in early disease, may have profound small-bowel motility disturbances that promote bacterial overgrowth and

malabsorption. A scleroderma patient who loses a lot of weight is likely to have this complication.

FIBROMYALGIA

- Fibromyalgia
- Osteomalacia
- Hypothyroidism
- viremias (HIV, other)
- bacteremias
- Narcotic withdrawal

Comments: chronic generalized pain and symmetric tenderness in a predetermined number of possible tender points characterizes Fibromyalgia.

Because osteomalacia may closely resemble FM, it is important in the evaluation of older patients with presumed fibromyalgia to inquire about dietary habits and request appropriate biochemical determinations.

Hypothyroidism is a great mimicker in Rheumatology and its clinical spectrum includes fatigue, diffuse soft tissue tenderness, and constipation. The author has picked up several cases of hypothyroidism masquerading, as FM. Thyroid-stimulating hormone (TSH) determination is one of the few legitimate tests in the evaluation of fibromyalgia. Fibromyalgia should not be diagnosed in acutely ill patients because fibromyalgic (and enthesial) tenderness is common in both viremia and bacteremia.

If you have the chance, examine FM points in-patients undergoing alcohol or opiate withdrawal. Reversible fibromyalgic tenderness is a major component in their muscoloskeletal pain.

PAINFUL, SWOLLEN HAND(S)

- Gout
- Pseudogout
- Rheumatoid arthritis
- Remitting seronegative symmetrical synovitis with pitting edema (RS3PE)
- Polimyalgia rheumatica (PMR)
- Mixed connective tissue disease (MCTD)
- Scleroderma
- Rupture of olecranon bursa
- Medsger,s syndrome (neoplasia)
- The puffy hand of drug addiction
- Reflex sympathetic dystrophy (RSDS)
- Eosinophilic fasciitis
- Sickle cell (hand-foot syndrome)
- Leprosy
- Factitial (the rubber hand syndrome)

Comments: conditions 1 through 5 produce hand edema in older people. Conditions 4 and 5 occur in older people. The condition is bilateral in RA, RS3PE, and PMR unless neurologic disease paralyzes one hand in which case the edema affects the good hand. Diffuse swelling

with a leathery feeling in both hands suggests MCTD and early scleroderma. In the latter, edema eventually goes away, as the skin becomes taut. Raynaud's is prominent in both conditions. Gout and pseudogout cause diffusely swollen hands in older patients with acute arthritis of the wrist, with or without digital synovitis. Rupture of the olecranon bursa which occurs predominantly in septic bursitis and RA, causes localized inflammatory edema that migrates distally along the ulnar border of the forearm reaching in some cases the dorsum of the hand. In medsger's syndrome edema affects predominantly the palmar side of both hands and is later replaced by a variant of dupuytren contracture. Drug addicts who use the dorsal hand for injection characteristically develop puffy hands. Burning pain and edema characterize the early stages of RSDS. This process is generally unilateral. In Eosinophilic fasciitis there is an indurated dorsal edema that typically spares the fingers. A puffy painful hand (and foot) caused by soft tissue and bone ischemia is a well- known feature of sickle cell crisis in small children. Lepromatous leprosy often causes bilaterally swollen hands. The diagnosis should be considered in immigrants from endemic areas. Additional skin lesions and neuropathy are usually present. Finally, disturbed individuals may self-induce hand or foot edema by applying a tight rubber band. A faint rubber band pressure mark is sometimes present on examination.

DISTAL-INTERPHALANGIAL (DIP) JOINT ARTHRITIS

- Osteoarthritis
- Nodal gout
- Psoriatic arthritis
- Multicentric reticulohistiocytosis

Comments: Almost everyone is aware that in a common form of OA there is a predilection for DIP involvement. The base of the distal phalanx becomes swollen and tender, which gives way, months later, to bony knobs known as Heberden's nodes.

Urate deposits may accumulate about Heberden's nodes. Acute crystal inflammation in this location is known as "nodal gout?" A rather similar involvement occurs in DIP psoriatic arthritis. These patients almost regularly feature grossly pitted nails. Rather than sclerosis, subchondral cysts, and cartilage thinning, which are typical of OA x-ray findings, psoriatic DIP arthritis includes centripetal erosions leading to "Pseudowidening" of the joint. Once again, look for psoriasis in acral sites and hidden areas.

Multicentric reticulohistiocytosis, mentioned in the previous pattern, also produces erosive changes at the DIPs.

OLIGOARTHRITIS

(Involvement of 2-3 joints)

- Psoriatic arthritis
- Reactive arthritis
- Lyme borreliosis, stage 3
- Gout
- Pseudogout
- Rheumatoid arthritis (RA)

Comments. Oligoarthritis should make one think automatically of psoriatic arthritis (actively look for the skin lesions) and postdysenteric or postvenereal reactive arthritis. The patient

may have, for instance, swelling in one knee, one ankle, and elbow, palmar and plantar lesions of reactive arthritis resemble pustular psoriasis. Shallow mucosal ulceration is virtually painless.

Lyme borreliosis, in the late stage, often features recurrent knee Oligoarthritis. Effusions may be huge. Inquire about exposure such as a history of a slowly enlarging rash during a summer beach vacation.

Gout and pseudogout are mentioned because both can resemble all joint conditions. Don't miss a crystal search. The same applies to RA. Interestingly, if in-patients with oligoarticular RA the examiner intently press the MTPs one by one, he or she almost regularly finds symmetric tenderness of which the patient was fully unaware.

SPONDYLITIS

- Ankilosing spondylitis(AS)
- Reactive arthritis
- Psoriatic arthritis
- Wipple,s disease
- Brucella infection
- Diffuse idiopathic skeletal hyperostsis(DISH)
- Osteomalacia

Comments: Characteristic of spondylitis is an inflammatory type low back pain associated with radiographic changes in the sacroiliac joints and often at higher levels in the spine. The prototypic disease is AS in which there is long-standing low back pain that is relieved by motion, limited lumbar spine motion, plus radiographic sacroiliitis. Similar symptoms are experienced in the reactive arthritides with axial involvement. The telltale sign is a preceding enteric or urogenital infection. Cases that clinically resemble reactive arthritis but lack an antecedent infection are known as undifferentiated spondyloarthropathies.

The unmistakable psoriatic plaques suggest psoriatic spondyloarthropathy. As mentioned, psoriasis may be hidden or absent in these patients. The presence of psoriasis in one or both parents should make one think of psoriatic spondylitis. Whipple's disease and brucellosis are believed to cause spondyloarthropathy by direct infection and perhaps as reactive phenomenon as well. Brucellosis is suspected when in the proper epidemiologic setting, patients develop a febrile illness with peripheral or axial arthritis. Forestier's disease is an ossifying rather than inflammatory condition. Bridging bone develops along the anterior longitudinal ligament of the spine, causing spinal stiffness. Severe cases feature ossification at tendon attachments causing enthesial pain. The end result in DISH may be quite similar to AS except that protracted morning stiffness is lacking. Patients with osteomalacia may feature back and pelvic pain as well as radiographic changes that resemble sacroiliitis. Diagnosis may be suspected from concurrent Hypophosphatemic proximal muscle weakness. Low phosphorus levels, low or normal calcium levels, increased alkaline phosphatase, the finding of pseudofractures on x-rays, and low 25(OH)D are diagnostic of vitamin D-deficient osteomalacia.

PROTRACTED DISTAL SYMMETRICAL POLYARTHRITIS

- Rheumatoid arthritis (RA)

- Systemic lupus erythematous (SLE)
- Polymyositis (antisynthetase syndrome)
- Certain drug reactions
- Psoriatic arthritis
- Gout
- Pseudogout
- Parvovirus B-19 infection(rare)
- Secondary syphilis(rare)
- Brucellosis(in endemic areas)

Comments: protracted distal symmetric polyarthritis in over 80% of cases represents RA. Because SLE may also present in this fashion, the appropriate serologic testing for both conditions should be requested.

Polymyositis will not be missed if proximal strength is checked. If the patient is taking procainamide, a beta-blockers, or quinidine, a drug reaction should be at least considered. These cases feature elevated antinuclear antibody titers, rheumatoid factor (Rf) test may be positive or negative, and antinative DNA and Sm. antibodies are negative. Preceding, concurrent, and familial psoriasis should be noted and examination should include areas in which psoriasis may be hidden.

Remember that both gout and pseudogout may present with a pseudo-RA pattern! Arthralgias are common in parvovirus infection, but cases with subacute or chronic arthritis are rare. An initial rash may suggest diagnosis. The condition resolves in a few days in most cases. Autoantibodies and parvovirus testing is not warranted in routine cases.

Secondary syphilis should be investigated if lymphadenopathy and a rash are present. Involvement of the palmar and plantar surfaces is frequent. Look also for oral or perigenital mucous plaques.

Brucellosis caused by *B. melitensis* occurs frequently in Latin America, the Middle East, and Mediterranean countries. Muscoloskeletal manifestations occur in over 50% of patients including a rheumatoid like peripheral arthritis or a spondylitic condition with unilateral SI involvement. Peripheral and axial involvement rarely coexists.

ARTHRITIS AND SUBCUTANEOUS NODULES

- Rheumatoid Arthritis
- Gout
- Pseudogout
- Sarcoidosis
- Light chain Amyloidosis(primary, multiple myeloma)
- Acute rheumatic fever(ARF)
- Hmochromatosis
- Whipple's disease
- Multicentric reticulohistiocytosis

Comments: In-patients with arthritis there is a tendency to consider subcutaneous nodules as proof of RA. While this is true in over 90% of cases, subcutaneous nodules may represent, among others, crystal deposits in chronic gout and pseudogout. If the patient remembers initial episodes of acute arthritis separated by periods of complete health, the likelihood of

crystal disease should be strongly considered, but there are RA cases which begin that way (Palindromic RA). If the patient has a negative RF, aspirate the nodule as discussed under gout!

In Sarcoidosis the RF test is usually negative and chest x-rays show adenopâthy and/or reticulonodular disease.

Light chain (LA) Amyloidosis should be suspected in an older person with arthritis, bilateral carpal tunnel syndrome, and subcutaneous nodules. 'A serum protein electrophoresis will reveal a monoclonal spike.

How about ARF? The condition is nowadays rare in the United States, and nodules, uncommon in pediatric cases, are even rarer in adults. But knowing the implications of a missed diagnosis, the condition must be mentioned.

Hemochromatosis and Whipple's disease are rare birds. Both feature a negative RF test, a red flag that should not be overlooked. Hemochromatosis is suspected from concurrent diabetes or cardiomyopathy or a family history that includes one or more of these findings. Whipple's cases often-present fever, lymphadenopathy, serositis, weight loss, CNS lesions, and diarrhea. Don't miss these diagnoses; the complications of Hemochromatosis are preventable and Whipple's disease is potentially curable (sometimes it can only be arrested). There is now a molecular test for it. Use it in the right setting!

The final condition listed, Multicentric reticulohistiocytosis, features small pearled periungual nodules as well as larger nodules elsewhere. This is a really rare condition that is usually diagnosed by rheumatologist and dermatologists.

ARTHRITIS AND MUSCLE WEAKNESS

- **Rheumatoid arthritis (RA)**
- **Ankilosing spondylitis (AS)**
- **Polymyositis**
- Dermatomyositis (DM)
- SLE, scleroderma, MCTD
- Sarcoidosis
- Whipple's disease

Comments: Rheumatoid arthritis patients become weak from several contributing factors. First, there is the so-called arthrogenic muscle inhibition in which joint inflammation weakens specific muscles, for example, the quadriceps in knee arthritis. Arthrogenic inhibition explains weak hands in finger and wrist arthritis, upper extremity weakness in elbow and shoulder arthritis, and lower extremity weakness, particularly hip and knee extension in hip and knee arthritis respectively. Thus, weakness caused by arthrogenic inhibition is focal. In addition, Corticosteroid use often results in steroid myopathy. Weakness in steroid myopathy is proximal and affects with predilection the iliopsoas muscles. Finally, a general decrease in activities results in generalized weakness. All of these mechanisms must be addressed in treatment planning. True myopathic weakness in an RA-like patient may represent Polymyositis of the variety associated with "mechanic's hands," interstitial pulmonary disease, and anti Jo-1 antibodies. It may also represent DM, a diagnosis that is hard to miss if the rash is properly identified.

SIE, scleroderma, and MCTD may feature proximal muscle weakness from associated myositis. These conditions are suggested by the presence of cutaneous sclerosis and Raynaud's in scleroderma/MCTD, and multisystem disease in SIE.
Various forms of arthritis occur in HIV infection, a condition that may cause myopathic weakness directly from HIV myositis or indirectly as a reaction to zidovudine.
Finally, the rare Whipple's disease may features arthritis and myositis. Wasting and lymphadenopathy in a patient with arthritis and myopathic weakness should bring into consideration both HIV infection and 'Whipple's disease.

ARTHRITIS, SUBACUTE, OR CHRONIC AND PERIFERAL NEUROPATHY

- Rheumatoid arthritis (RA) with vasculitis
- Polyarthritis nodosa (PAN)
- Wegner, s granolomatosis
- Leprosy

Comments: An unequivocal polyarthritis with synovial thickening and effusion suggests that a patient with arthritis and neuropathy indeed has RA as opposed to PAN, in which arthicular findings are comparatively minor. Although both conditions may cause similar neurologic findings, in large series PAN features predominantly mononeuritis multiplex whereas RA more often causes peripheral neuropathy.
Peripheral neuropathy and arthritis often occur in Wegener's granolomatosis, but ulcerated lesions in the nasal passages, sinuses, or mouth, pulmonary infiltrates, and often renal involvement suggest the diagnosis. A closely related condition, Churg-Strauss's syndrome, causes peripheral neuropathy and arthritis in a context of marked peripheral blood eosmophilia, pulmonary infiltrates, plus a recent or longstanding history of asthma.
Finally, Lepromatous leprosy may feature RA-like arthritis and peripheral neuropathy. Diagnosis is suggested by epidemiologic considerations and associated skin lesions.

PROXIMAL JOINT PAIN AND STIFFNESS

- Polimyalgia rheumatica (PMR)
- Osteomalacia
- Multiple myeloma (MM)
- Metastatic cancer
- Light chain (LA) Amyloidosis
- Ankilosing spondylitis (AS)
- Frozen shoulder

Comments: PMR is not to be missed because (1) some cases associate with giant cell arteritis, and (2) it is a readily treatable condition. Patients with PMR are older individuals who complain of severe proximal pain and stiffness involving shoulders, neck, hips, and often the lower back. Night rest is interrupted by pain, and morning stiffness and pain are severe and protracted, lasting several hours. The ESR is typically very high and there may be thrombocytosis.
Osteomalacia is another great mimicker. Bone pain from pseudofractures and fractures is typically proximal. Chest wall, shoulder regions, and hip regions are painful. Proximal muscle

weakness may also be present from hypophosphatemia. Consider osteomalacia in any older patient with these symptoms.
Multiple myeloma (MM) and disseminated Metastatic cancer also have a predilection for older people. Diffuse marrow expansion and widespread bone metastases often result in proximal pain. The ESR is very high, and confusion with PMR is possible. However, malignancy causes osseous tenderness, which is not a feature of PMR.
Amyloidosis of the LA type complicates both MM and benign monoclonal gammopathies. Amyloid infiltration of joints and muscles causes proximal pain and stiffness. Distinguishing features in Amyloidosis include macroglossia, firm synovial masses, and a profound bilateral carpal tunnel syndrome.
Ankilosing spondylitis, because it involves the lumbar and cervical spinal segments, plus the shoulders and hips, may also lead to confusion with PMR. However, in AS patients are younger, there is male predominance, and there is not only pain but also limitation of motion.
Frozen shoulder, an initially painful and then restrictive shoulder condition, occurs predominantly in middle-aged or older individuals. Bilateral cases of frozen shoulder may be clinically confused with PMR. Lack of lower body involvement, marked restriction of shoulder motion, and a normal ESR lead away from a diagnosis of PMR.

ARTHRITIS AND A RASH

- Chronic urticaria
- Vasculitic urticaria
- Systemic lupus erythematous (SLE)
- Dermatomyositis
- Polymyositis
- Psoriatic arthritis
- Reactive arthritis
- Chronic Sarcoidosis
- Sweet's syndrome
- Leprosy

Comments: in chronic urticaria, which associates with angioedema, there are minimal constitutional symptoms and individual urticarial wheals last less than 24 hours. In vasculitic urticaria the itchy lesions last longer and are followed by local hyperpigmentation. Obstructive bronchopulmonary disease, ocular inflammation, and nephritis can be present.
Systemic lupus erythematous features a variety of rashes; the best known is malar erythema and discoid lupus. The tip-off for the condition is its multisystemic involvement including arthritis, serositis, CNS disease, and renal disease in various combinations. Given consistent clinical findings, diagnosis of SLE is reached by (I) positive SLE-specific serology's such as double-stranded DNA or Sm antibodies, or (2) positive ANA plus the exclusion of mimicking conditions. The primary antiphospholipid syndrome does not cause arthritis, but patients with SLE may present a secondary antiphospholipid syndrome. At least half of these patients feature livedo reticularis, which is a macular netlike reddish blue rash most prominent around the knees and on the thighs. Livedo reticularis may also occur in disseminated intravascular coagulation, cholesterol atheroembolic disease, cryoglobulinemia, polyarthritis nodosa, and other conditions that cause vasospasm or occlusion in perforating arteries as they travel from the subcutaneous tissue to the dermis.

Dermatomyositis rash is erythematous and affects sun-exposed areas including face, neck, and upper chest depending on garments used. It may be quite itchy and photosensitive with prominent superficial edema. Heliotrope rash is a reddish purple periorbital discoloration of upper lids that also look edematous. Gottron's sign is a violaceous papular rash that erases the creases at the knuckles. One or more of these rashes in association with proximal muscle weakness is highly indicative of dermatomyositis, which may or not include arthritis.
In Polymyositis, a rash that has been aptly described as "mechanic's hand" is characterized by coarse, fissured skin in opposing surfaces of thumb, index, and middle finger. The area tends to accumulate dirt despite the patient's best efforts. Rheumatoid like arthritis may be prominent in these patients.
Psoriasis, a lesion that is well known to all primary care physicians, is the hallmark of psoriatic arthritis. Hidden psoriatic lesions should be intently looked for in suspected cases of psoriatic arthritis.
The rash in reactive arthritis includes palmar and plantar pseudopustules (keratoderma blennorrhagica), papular or hyperkeratotic glans lesions (balanitis circinata), and shallow painless erosions in the hard palate.
Chronic Sarcoidosis, in which arthritis and tenosynovitis may be prominent, features brownish violet infiltrated skin lesions in the form of papules or plaques. Another well-known but rare Sarcoidosis lesion is "lupus pernio" in which there is a diffuse red infiltration of nose, cheeks, and earlobes.
Sweet's syndrome, or acute febrile dermatosis, is characterized by skin lesions that appear abruptly and evolve in several days to several weeks. Lesions are bright red to purple, well demarcated, very painful, tender, and often multiple. The center may be depressed. Fever and leucocytosis are often present. Myalgias, Arthralgias, and arthritis are quite frequent. The arthritis is migratory and tends to affect knees, elbows, ankles, and fingers in descending frequency. Diagnosis of Sweet's syndrome is based on the skin biopsy that shows dense neutrophilic infiltrates in the upper and mid dermis.
Leprosy, a relatively rare condition in the United States where it is virtually confined to Asian or Latin American immigrants may present with a variety of lesions ranging from a leonine face in Lepromatous leprosy to a well-defined anesthetic hypopigmented muscle in tuberculoid leprosy. In the Lucio reaction, hyperacute vasculitis features high fever, neuritis, and skin ulcers. Myositis, rheumatoid like arthritis and enthesopathy are additional manifestations of Lepromatous leprosy. Interestingly, autoantibodies including rheumatoid factor and ANA may be present. The association of facial erythema, polyarthritis, and a positive ANA may suggest a diagnosis of SLE. Tip-off include the infiltrative appearance of the skin, peripheral neuropathy, and leucocytosis in patients with leprosy.

Arthritis and heart murmur

- Subacute bacterial endocarditis (SBE)
- Cardiac mixoma
- Ankilosing spondylitis
- Reactive arthritis
- Acute rheumatic fever
- Rheumatoid arthritis
- Systemic lupus erythematosous with Libman-Sack endocarditis
- Relapsing polycondritis

Comments: There are patients with valvular heart disease who on evaluation are found to have arthritis (the connection between the two may or not be realized), and there are arthritic patients who in the course of their condition develop a heart murmur. A heart murmur may of course be unrelated to the arthritis, such as murmurs due to valvular calcification.
Real associations between valvular disease and arthritis are relatively common. For instance, bacterial endocarditis causes peripheral arthritis and back pain in as many as 50% of cases. These patients may feature, late in the disease, a positive rheumatoid factor test as well as hypocomlementemia. Thus, febrile patients with arthritis and a heart murmur, particularly if back pain is also present, are suspected of having SHE. The same association holds true for cardiac mixoma, a notorious mimicker of systemic disease. Blood cultures and a transesophageal echocardiogram is basic to diagnosing both conditions.
Both AS and reactive arthritis cause, in about 5% of patients, dilatation of the aortic root leading to aortic regurgitation, sometimes retraction of the anterior mitral leaflet causing mitral regurgitation, as well as inflammatory changes in the fibrous portion of the interventricular septum leading to A-V conduction disturbances. Thus, in-patients being evaluated for aortic regurgitation, check any available pelvic film *for* possible sacroiliitis.
Acute rheumatic fever, as noted earlier, is uncommon nowadays, although pockets of disease pop up here and there even in the United States. Mitral regurgitation is often present at initial evaluation and may be reversible. Mitral stenosis develops late, often decades after the initial attack. The association of an additive arthritis and mitral regurgitation should therefore prompt determination of streptococcal antibodies. Rheumatoid arthritis rarely causes heart murmurs, but when it does it is likely that the patient is strongly seropositive and that rheumatoid nodules have formed in the valve leaflets.
In SLE heart murmurs may result from valve necrosis and rupture, Libman-Sack endocarditis (initially an inflammatory lesion), a secondary antiphospholipid syndrome resulting in thrombotic vegetations, or bacterial endocarditis. Lupus patients who are febrile and have a heart murmur should of course have the appropriate blood cultures.
Relapsing polycondritis is a rare condition that is made even rarer by lack of ascertainment. Diagnosis may be missed because appropriate questions may not be asked about nose or ear inflammation, and because the floppy ears that characterize late disease may be missing in the average case. Relapsing polycondritis may dilate the aortic root and cause aortic regurgitation.

HEMARTHROSIS

- Excessive anticoagulation
- Hemophilia
- Intraarthicular fracture
- Osteonecrosis
- Gout, pseudogout
- Pigmented villonodular synovitis (PVNS)
- Synovial hemangioma
- Synovial metastases

Comments: patients may bleed in to joints from excessive coumadin or heparin anticoagulation. If anticoagulants are not being used, a clotting abnormality may be present. Hemophiliacs tend to bleed recurrently, with minimal or no trauma, and usually in the same joint(s). In an adult or older person, new-onset coagulopathy may be due to factor V111

inhibitors such as those seen in cases of SLE or multiple myeloma, or fibrinolysis in prostatic and other cancers.

As mentioned, if there is a history of trauma (but also in unconscious or unreliable persons without such a history), the bloody SF should be placed in the refrigerator. If a fat layer forms an initial x-rays are negative, rule out an undisplaced intraarthicular fracture by bone scan, CT, or MRI (MRI is best) or by repeating the x-rays 2 weeks later. The problem with this approach is that during this period the affected part must be kept immobilized.

If osteonecrosis was responsible for the bleed (such as in patients on long-term corticosteroids and older patients who are prone to idiopathic osteonecrosis), a similar approach might be taken. As in suspected intra arthicular fractures, the author favors a quick diagnosis of osteonecrosis because with appropriate rest or other measures some of these lesions heal.

IT should be reemphasized that both gout and pseudogout may cause Hemarthrosis. Crystal search may be laborious in a slide flooded with erythrocytes.

Recurrent Hemarthrosis occurring in the same joint without trauma or coagulopathy may be caused by a benign intra arthicular lesion such as pigmented villonodular synovitis (PVNS) and synovial hemangioma. In PVNS the synovium feels very thickened. In synovial hemangioma a superficial lesion may be present and phlebolitis (calcific bodies within dilated veins) may appear on x-rays. MRI is quite helpful in diagnosis of PVNS, although it may not discriminate well the lesion from synovial hemosidrosis caused by repeated hemangiomas bleeds. Diagnosis of both lesions is Arthroscopic. Synovial metastases are quite rare. Bone lysis is often present on x-rays. In 50% of cases malignant cells are present on synovial fluid smears.

CHAPTER II

PHYSICAL EXAMINATION AND REGIONAL RHEUMATIC DISEASES

HAND AND WRIST PAIN

FUNCTION

The wrist and hand, each comprising many small joints, acts as a single functional unit. The hand undertakes a variety of important functions including:

1. *Grip and manipulation*: The two important basic grips are:
   - *Fine precision pinch* (1): This is achieved by opposing the pulp surfaces of the thumb and finger (usually the index finger), and requires rotation of the thumb (and to some extent finger) during opposition. 'Scissors' pinch is less precise opposition of the thumb against the side of the index finger.
   - *Power grip* (2): Achieved by curling the fingers tight into the palm, the power grip is optimized by extension and locking of the wrist (permitting full power from the finger flexors). The grip is maximal with 90-degree rotation and 45 degree ulnar deviation ('hammering' position). The hook grip is a modification of the power configuration.

A range of variants can be generated from these basic grips, giving the hand great flexibility for manipulation. Grip is enhanced by complimentary use of both hands, and impaired function in one hand may reduce overall function considerably.

2. ***Proprioception***: Hands are the principal points of contact for touch sensitivity with the environment.

3. *Communication:* Hands undertake a range of non-verbal signals and are important in social contact (e.g. handshakes, caresses).

4. *Locomotion*, e.g. during crawling, swimming.

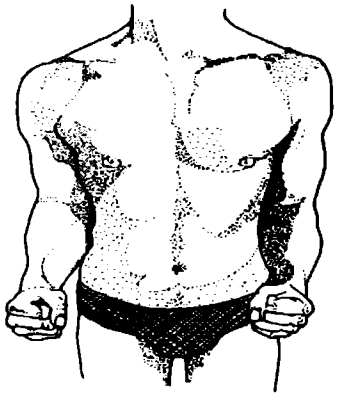
BASIC ANATOMY

The *inferior radioulnar joint* (3) is delineated distally by the triangular disc or 'ligament' (a key stabilizer to the wrist). The capsule and synoviom extend proximally between the radius and the ulnar as the sacciform recess. Together with the superior radioulnar joint it permits supination/pronation.

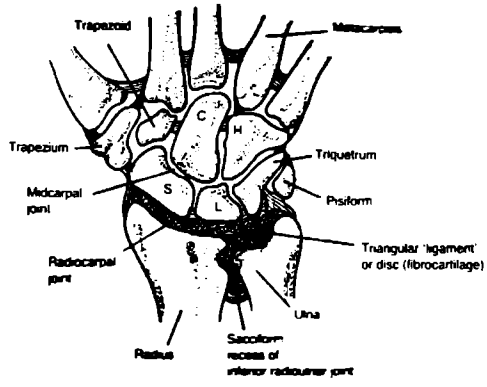
The *radiocarpal joint*, with its separate synovial cavity, permits flexion, extension and lateral movement between the radius (and triangular ligament) and the proximal row of the

carpus (scaphoid, lunate, triquetrum). The *midcarpal joint* has a separate synovial space (often communicating with carpometacarpal cavities) and connects the proximal and distal

1 Fine precision pinch.



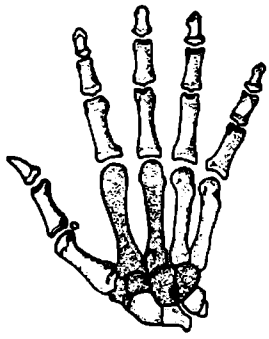
2 Power grip.



3 Bones and joints of the wrist (S = scaphoid, L = lunate, C = capitate, H = hamate)

(1,2,3)

rows (trapezium, trapezoid, capitate, hamate): only minor movement (flexion, extension, and some rotation) occurs here. The second and third *carpometacarpal joints* (CMCJs) permit little if any movement; the second and third metacarpals and distal carpal row thus form a fixed L-shaped unit (4) around which, in functional terms, the rest of the hand is built. The first CMCJ is exceptionally mobile: the metacarpal sits astride the trapezium, facing the ulnar border. The fourth and fifth CMCJs are less mobile than the first but move together on the hamate, permitting formation of a 'hollow palm'

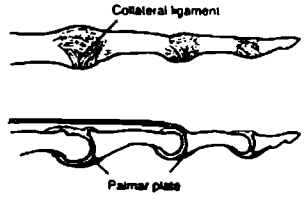


4 The L-shaped immobile segment.

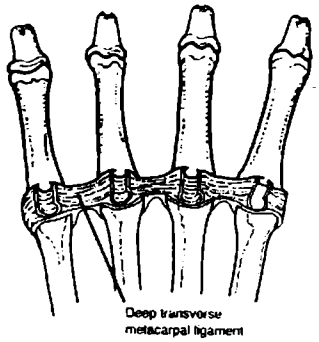
(4)

The *metacarpophlangeal joints* (MCPJs: 5) are modified hinge joints. Their position is marked on the palmar surface by the distal palmar crease. Each proximal phalanx base has a cartilaginous Volar extension (the palmar ligament or plate). The deep transverse metacarpal ligament joins the second to fifth Volar plates (6). Each MCPJ has a radial and ulnar lateral ligament eccentrically placed across the joint, tightening only in flexion (7).

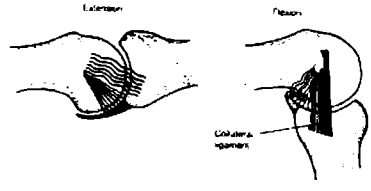
The *proximal* and *distal interpalangeal joints* (PIPJs, DIPJs) are true hinge joints. They also posses palmar plates and fibrous tunnels occur on the palmar aspects of the phalanges. Each interpalangeal joint (IPJ) has radial and ulnar collateral ligaments centrally placed across the joint (tight in both flexion and extension). The synoviom of each MCPJ and IPJ extends more proximally than distally (5) due to the arrangement of tendon slips and other structures, which cause progressive tightening of space distally.



5 Metacarpophalangeal and interphalangeal joints.



6 Volar plate arrangement.

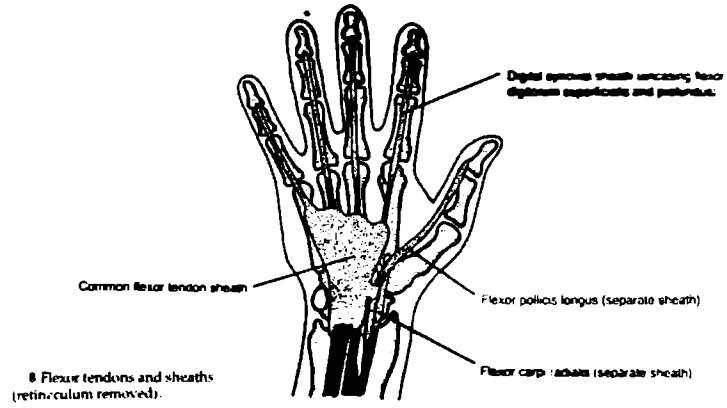


7 Tightening of MCPJ collateral ligaments in flexion

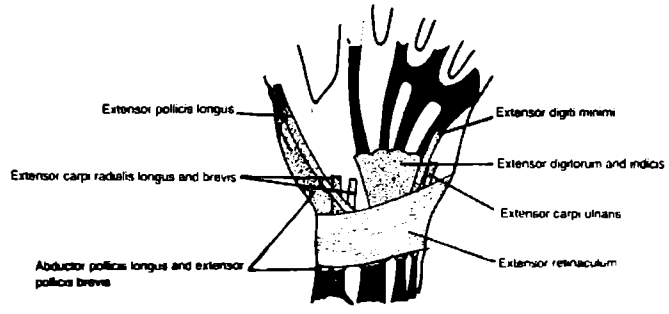
(5,6,7)

The hand is primarily designed for flexion (gripping). The long flexor tendons (8) run in the common flexor tendon sheath (flexor pollicis longus often has a separate sheath): all are encased by the flexor retinaculum. The palmar fascia (an extension of palmaris longus) attaches to the flexor retinaculum and partly divides into four attaching to deep transverse metacarpal ligament and phalanges. The median nerve passes tight alongside the flexor

tendons beneath the retinaculum in the carpal tunnel (it may thus be compressed by tenosynovitis). The extensor tendons are also long structures enclosed by the extensor retinaculum (9). Extensor policies brevis and abductor policies longus are encased in a separate fibrous canal at the radial styloid region where inflammation may give rise to stenosing tenosynovitis (*de Quervain's tenosynovitis*).



8 Flexor tendons and sheaths (retinaculum removed).



9 Extensor tendons and sheaths.

(8,9)

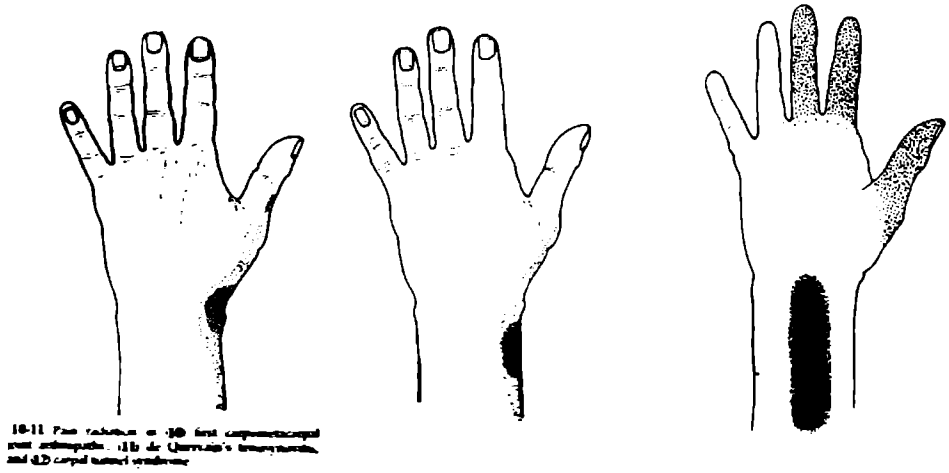
Wrist and hand joints are commonly involved in inflammatory conditions (e.g. rheumatoid, seronegative spondyloarthropathy). Generalized osteoarthritis predominantly involves DIPJs and PIPjs, the first CMCJ and the scapho-trapezoid joint (other carpal joints are spared). Isolated involvement of radiocarpal and midcarpal joints is common in crystal-associated arthropathy (pyrophosphate arthropathy and gout).

Symptoms

Pain from any of the small joints in the wrist and hand is usually well localized and the patient easily pinpoints its source. However, three common conditions may cause radiation of pain on the radial side of the hand (10-12):

- *First CMCJ arthropathy* (*usually osteoarthritis*): This is the one joint that may cause wide radiation (distally up the thumb, proximally up the distal forearm), though pain is maximal over the joint itself.

- *De Quervain's tenosynovitis*: Pain is maximal around the radial styloid but often radiates into the thumb and proximally up the forearm.
- *Carpal tunnel syndrome*: The key symptoms suggesting peripheral nerve entrapment are nocturnal or early morning exacerbation of symptoms (symptoms may be confined to this time). In carpal tunnel syndrome, median nerve compression may cause (1)- paraesthesia and dysaesthesia distally to the thumb, index and middle fingers, often with clumsiness, and (2) painful aching proximally up the forearm, occasionally to the elbow.



(10,11,12)

Problems (particularly synovitis) affecting hand joints are often very apparent to the patient, due to early interference with activities of daily living (dressing, etc.): even mild tenosynovitis or IPJ synovitis may cause tightness of rings.

Pain and sensory disturbance may radiate into the hand from above, particularly the elbow (arthritis, epicondylitis), shoulder (arthropathy, rotator cuff), and cervical spine (root entrapment of C6, 7, 8). In these situations symptoms are often less well defined and accompanied by more proximal symptoms.

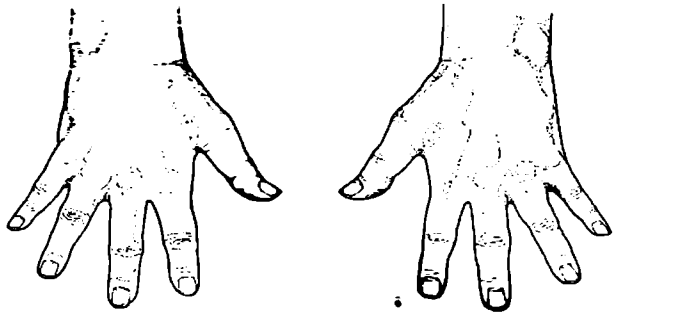
Inspection at rest

- Inspect and compare the dorsal and palmar surfaces of both hands, then inspect from
- the side with hands outstretched.

Inspection of the extensor surface.

Inspect the back of the hands rested on the patient's lap or on a flat surface (13). Look for:

Skin and nail changes



Inspection of the extensor surface

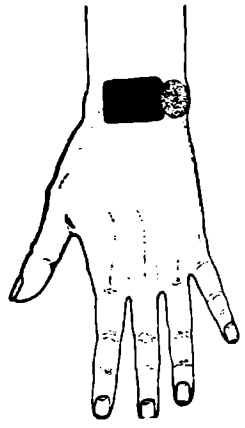
Inspect the back of the hands rested on the patient's lap or on a flat surface (13). Look for:

(13)

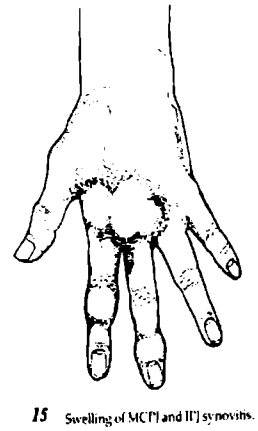
These include erythema, psoriasis, vitiligo, mixed hyper/hypopigmentation ('salt and pepper' appearance of scleroderma), skin tightness with loss of flexures (sclerodactyly: typical of scleroderma and overlap connective tissue syndromes); and current or past evidence of trauma. Violaceous/silvery raised lesions (Gottron's papules) occur over extensor surfaces in dermatomyositis. Lupus rashes often affect skin between joints, vasculitic rashes often affect lateral more than dorsal aspects of fingers. The nails require inspection for clubbing, thimble pitting, splinter hemorrhages subungal hyperkeratosis, dystrophy and abnormalities of nailfold capillaries.

Swelling

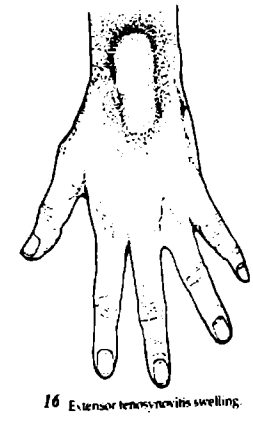
All joint swellings are most prominent on the dorsal surfaces. Radiocarpal synovitis (14) produces rectangular swelling symmetrically placed each side of the joint line; inferior radioulnar synovitis causes a domed swelling that rounds off the distal ulna prominence; intercarpal and CMCJ synovitis usually produce modest swelling central over their joint lines. However, both MCPJ and IPJ synovitis cause swelling more proximal than distal to the joint line (15), MCPJ synovitis producing swelling between metacarpal heads, and IPJ swelling producing posterolateral bulging between extensor tendon above and the lateral collateral ligament on each side. With moderate gross IPJ swelling, stretching of the skin makes the overlying wrinkles less distinct. Extensor tenosynovitis also produces swelling over the carpus: it may differ from radiocarpal synovitis in being asymmetrically spread across the joint line, extending more distally over the metacarpals and having an irregular distal contour (16).



14 Position and shape of swelling due to radiocarpal (black) and anterior radioulnar (stippled) synovitis.



15 Swelling of MCPJ and IPJ synovitis.

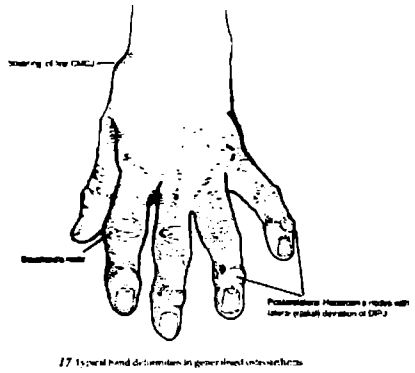


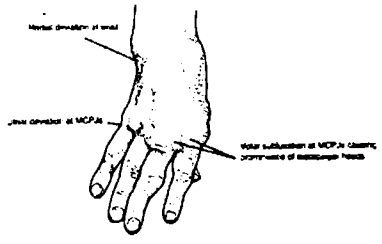
16 Extensor tenosynovitis swelling.

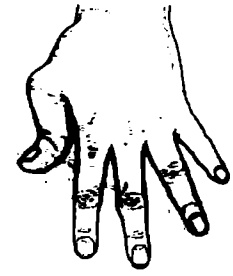
(14,15,16)

Deformity

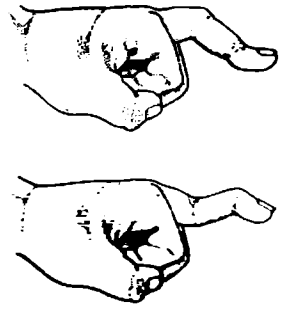
A variety of deformities may occur, all best seen from the dorsal aspect. Apart from synovitis, undue prominence of the ulnar styloid may result from subluxation caused by weakening and rupture of the distal radioulnar ligament (usually rheumatoid). Conversely, the ulnar prominence may not be apparent, due to erosive disease or previous surgery. Ostephytosis of the first CMCJ may cause squaring of the hand (17). Ulnar deviation at MCPJs (18) usually accompanies and follows radial deviation at the wrist (usually rheumatoid). Volar subluxation of phalanges at MCPJs leads to prominence of metacarpal heads and a step-down deformity. Lateral deviation (radial or ulnar) at IPJs (17), and firm posterolateral swellings of DIPs (Heberden's nodes) and PIPJs (Buchard's nodes) are characteristic of osteoarthritis. Combined deformities include 'Swan neck' (PIPJ hyperextension, DIPJ hyperflexion: 19), 'Boutonniere' ('buttonhole': PIPJ hyperflexion, DIPJ hyperextension- the PIPJ pushing through the extensor tendon like a button through a buttonhole-20), and 'Z' deformity of the thumb (MCPJ hyperflexion, IPJ hyperextension: 21). Inability to place the fingers flat (fixed- flexion) can result from flexor tendon or joint problems.







Combined deformities *19* swan-neck; *20* boutonniere; *21* Z deformity of thumb.



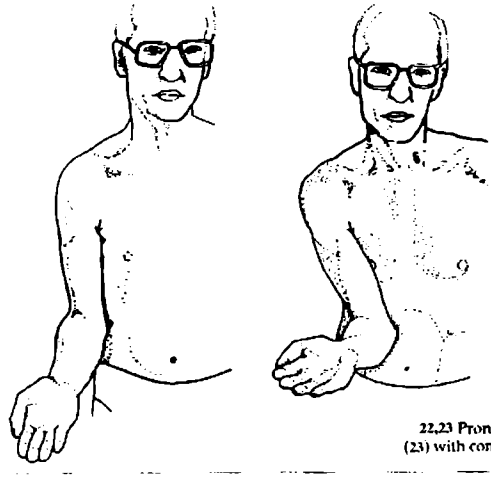
(17,18,19,20,21)

Wasting

This is often difficult to observe. Apart from wasting of dorsal interosseous muscles, marked 'guttering' between extensor tendons may result from Volar subluxation of the carpus, which automatically makes the extensor tendons and gaps between them more prominent.

Inspection of the palmar surface

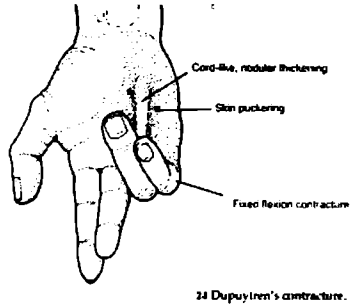
Ask the patient to pronate the hands. Difficulty or pain during pronation reflects problems with the superior and/or inferior radioulnar joints. Supination/pronation involves the radius moving over the stationary ulna: if the radioulnar joints are compromised the patient may use a trick maneuver (22, 23), bringing the elbow across the abdomen to increase supination (by partly rotating the ulna). On the palmar surface look for:



22,23 Pronation with flexed elbow: (22) normal; (23) with compromised radioulnar joints.

Skin changes

Palmar erythema is common in rheumatoid disease. Dupuytren's contracture is recognized as puckering of the skin with thickening of the palmar fascia: in established cases there may be fixed flexion contracture, predominantly affecting the ring and little fingers (24). Small vessel infarcts often occur as black spots, particularly on finger pulps.

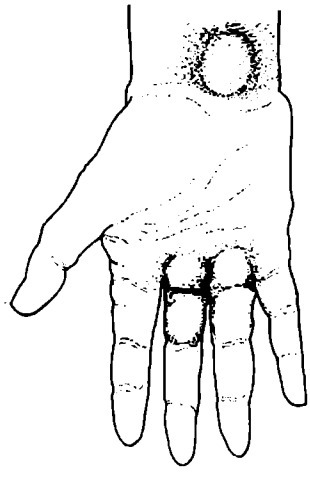


24 Dupuytren's contracture.

(24)

Swelling

Swelling on the Volar aspect most commonly represent flexor tenosynovitis, often most obvious proximal to the wrist crease, between the distal palmar crease and finger base, and occasionally between the skin creases of the fingers (25). As a result of the thick flexor apparatus joint swelling is rarely apparent on the palmar surface.



(25) 25 Flexor tenosynovitis swelling.

Wasting

Unlike the dorsal surface the palms are an excellent site to observe muscle wasting. Irrespective of age, both thenar and hypothenar eminencies should be convex. Localized outer thenar wasting usually reflects median nerve compression: it often also accompanies first CMCJ arthritis.

Lateral inspection

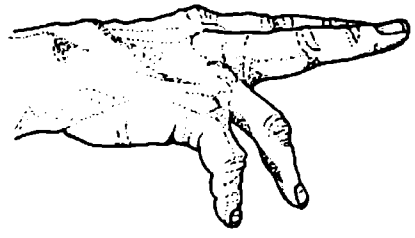
Ask the patient to outstretch the hands and inspect from the side. The main features to note are:

Ability to fully extend fingers

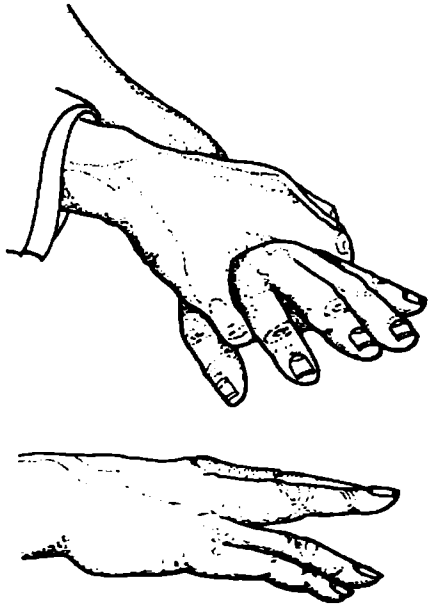
The patient may have almost no active extension of one or more fingers (commonly the little finger and the ring finger: 26). The examiner, however, may be able to extend the finger passively, only to see it drop down when the finger is released. This combination reflects extensor tendon rupture (in inflammatory disease this usually occurs close to the ulnar styloid). Some patients have partial active, but full passive, extension: this reflects rupture of the extensor tendon slips, permitting the extensor tendon to drop to the ulnar side of the MCPJ. Sometimes the slipped extensor tendon can be palpated and pushed onto its usual position above the MCPJ: if the examiner holds it there, the patient may be able to fully extend the finger only for it to return to the original situation when the examiner lets go (27, 28).

Volar subluxation of the carpus

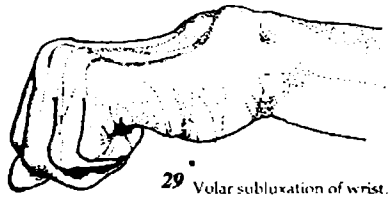
This produces a 'dinner fork' deformity (29: perhaps the most characteristic hand deformity of rheumatoid).



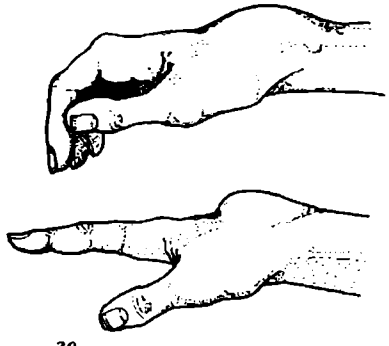
26 Complete extensor tendon rupture (ring finger and little finger).



Extensor tendon slip. In *27* the examiner can correct the slip, permitting full finger extension. When the examiner lets go *28* the fingers drop again.



29 Volar subluxation of wrist.



30 The 'tuck' sign of extensor tenosynovitis.

(27,28,29,30)

Swelling over the dorsum of wrist

This may have been noted earlier, reflecting synovitis (radiocarpal, midcarpal) or extensor tenosynovitis. To differentiate tendon sheath from joint swelling, inspect the swelling with the MCPJs flexed; then ask the patient to fully extend the fingers (30). Tenosynovial swelling moves proximally with the tendons, its distal boundary becoming more definite as the tenosynoviom 'tucks' in (the 'tuck' or 'shelf' sign). Joint swelling remains unaltered by extensor tendon movement.

Volar subluxation of the MCPJs

This may be more obvious from the lateral view (see 18).

Inspection during usage

Ask the patient to clench the fist in a power grip (see 2), observing the ability to curl the fingers tightly in to the palm. Asses power by asking the patient to grip examiner's finger(s) as hard as possible. Next, observe the fine precision pinch (see 1) by asking the patient to oppose the tip of the thumb to the tip of each finger in turn: to test whether this movement is functional ask the patient to pinch examiner's finger (looking for weakness, thumb instability). Such screening, together with the history, gives a good indication of the patient's likely functional problems.

Palpation

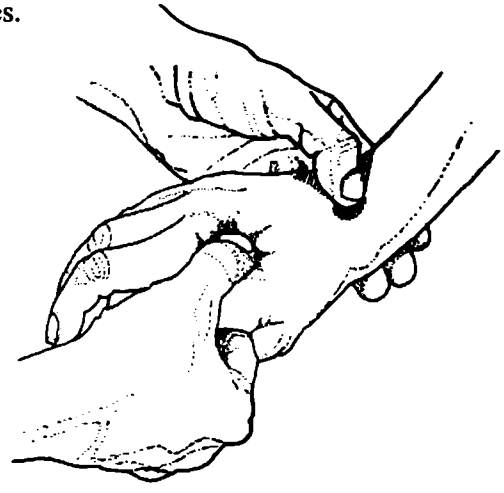
This is best done by facing the patient at a slight distance, and supporting the relaxed hand from below with the fingers of one hand. Assess the following in turn.

Increased warmth

This is felt by sweeping the back of the hand down over the forearm, the wrist, and the back of the hand and the fingers. Normally, skin temperature decreases towards periphery: marked coolness may be noted with Raynaud's. Increased warmth, particularly over the wrist and the MCPs, is easily felt if present.

Radiocarpal joint

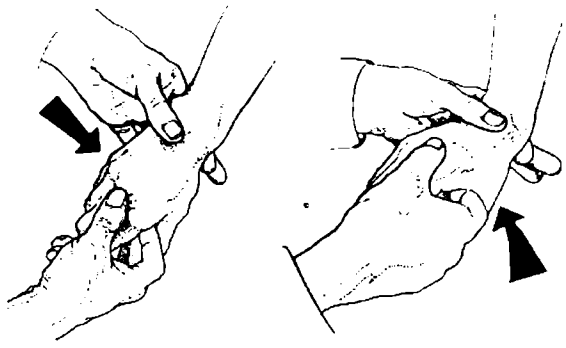
Identify the joint line by palpating with the left thumb while the right hand uses the second/third metacarpals as a lever to passively flex and extend the joint (31). The triangular space between radius, scaphoid and lunate is usually easily felt, representing the center of the joint line. Press all along the dorsal joint line for tenderness. If there is soft-tissue swelling, palpate to delineate its boundaries.



31 Palpation for radiocarpal joint line.

(31)

(radiocarpal synovitis symmetrically spreads across the joint line). Then feel across the top and bottom of the joint for crepitus while assessing the range of extension and flexion passively (32, 33). Note any restriction and presence of stress pain. Lateral radial and ulnar movement at the joint can also be assessed.



32,33 Palpation for crepitus while assessing radiocarpal flexion (32) and extension (33)

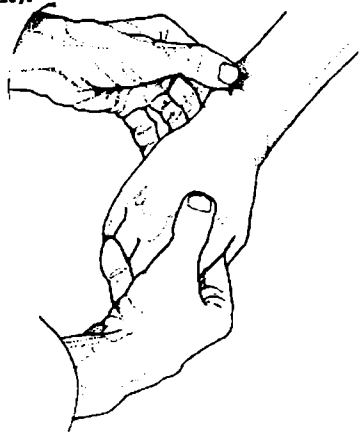
(32,33)

Inferior radioulnar joint

Move the left thumb to the distal ulnar prominence and palpate to insure the styloid region is intact and has not been whittled away by erosive disease and to detect any soft-tissue swelling due to synovitis (if moderate- to-marked, a balloon sign may be detected.

Pull the carpus over the radial side and press the ulnar styloid firmly in a volar direction (34) to:

- Elicit any pain from the joint
- Feel for crepitus.
- Detect any increased movement (the 'piano key' sign, reflecting weakness or rupture of the distal radioulnar ligament).



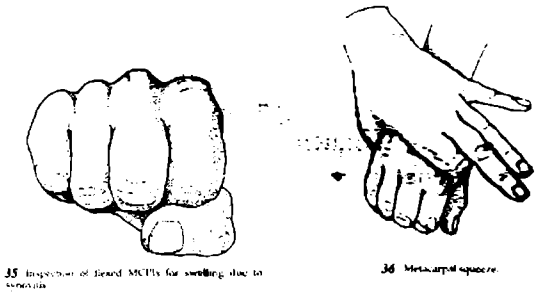
34 Palpation for 'piano key' sign, crepitus and tenderness of the inferior radioulnar joint.

(34)

MCPJs

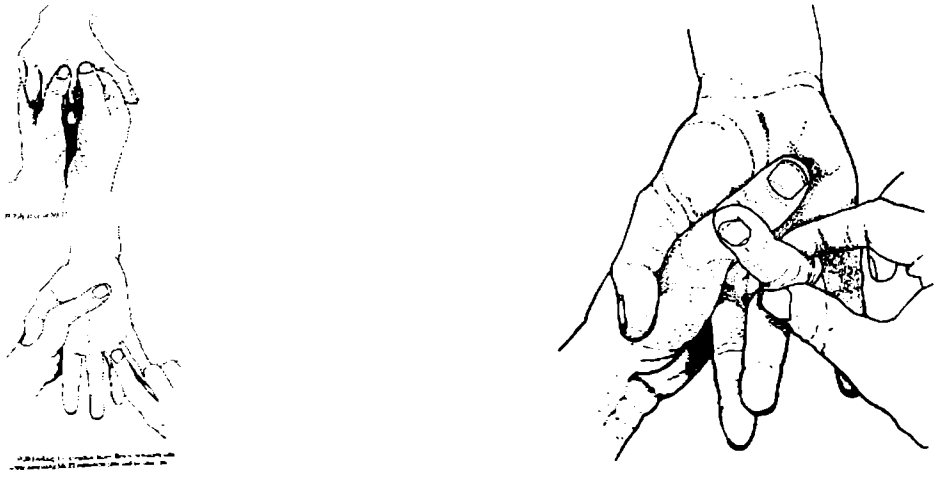
Inspect the joints flexed (35) to look for filling in of the gutters between the metacarpal heads (representing synovial swelling). Tenderness of all the MCPJs can be elicited by squeezing with one hand across all the metacarpals ('metacarpal squeeze, 36): tenderness reflects inflammation of one or more MCPJs. For individual MCPJs, continue to support the patient's hand from underneath with the fingers of both hands and use the thumbs to detect

posterolateral joint line either side of the extensor tendon. The joint line is surprisingly distal and is best found by moving the thumbs proximally along the proximal phalanx to find its expanded base- between this and the metacarpal head is the valley of the MCP joint line (37).



(35,36)

To confirm, keep palpating while slightly flexing and extending the proximal phalanx: this additionally allows detection of any crepitus and palpation for volar subluxation. Having confirmed the position of the joint line, press firmly with both thumbs for joint-line tenderness, then palpate around each side of the metacarpal head to determine the presence and extent of any soft tissue swelling. Examine each of the second- fourth MCPJs in this way.
To assess MCPJs movement, gently supinate the patient's hand. Place your left thumb proximally in the palm to feel for crepitus of flexor tenosynovitis while your other hand holds the proximal phalanx and moves the MCPJ from full extension through to maximal flexion (38, 39). Repeat this with each of the second-fifth MCPJs.

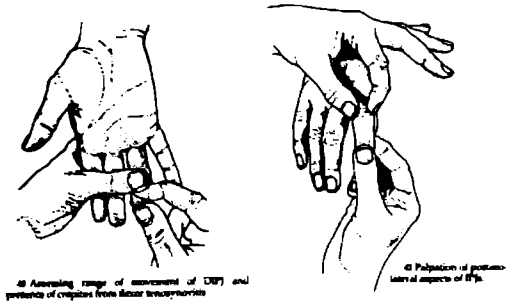


(37,38,39)

IPJs

Having just concurrently assessed MCPJ movement while feeling for tenosynovitis, continue in similar fashion assessing the range of movement of the second-fifth PIPjs and DIPJs again feeling for crepitus over the flexor tendon sheath. For each PIPJ, place the left thumb over the flexor tendon as it crosses the proximal phalanx and with the right hand move the middle phalanx from extension through to maximal flexion (40). For each DIPJ, place the left thumb

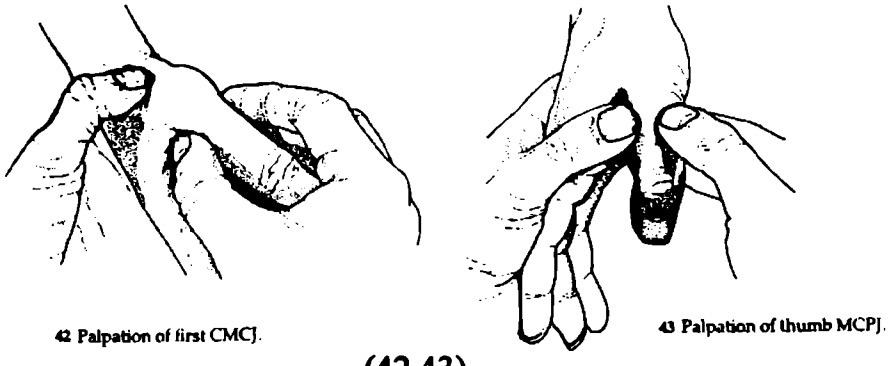
over the flexor tendon as it crosses the middle phalanx (to detect crepitus), while the right hand uses the distal phalanx to move the DIPJ from extension through to full flexion.
Now passively pronate the patient's hand again to palpate the IPJs (PIPjs and DIPJs are examined identically). Place the thumb and index finger of the left hand on the two-posterolateral aspects of each IPJ (between the extensor tendon and the collateral ligaments on each side; 41). Passively flex and extend the IPJs with the other hand to confirm correct positioning over the joint line and to feel for any crepitus. Then squeeze on the posterolateral aspect for joint-line/capsular tenderness and palpate proximally for soft-tissue swelling. If swelling at this site extends proximally much more than distally, and becomes tenser as the finger is flexed, it is capsular/synovial in nature.



(40,41)

The thumb

Identify the first CMCJ by palpating with one hand while passively moving the thumb metacarpal with the other (42): feel for crepitus as the range of movement is assessed. Press firmly for joint-line/capsular tenderness and palpate for any capsular swelling. Next, place the left thumb and finger over the posterolateral aspects of the thumb MCPJ, while the right hand passively moves the joint, assessing the range of movement and feeling for crepitus. Having identified the joint lines, press firmly for tenderness (43) and palpate for capsular swelling (this extend proximally more than distally, and tenses on flexion of the MCPJ). The thumb IPJ is assessed similarly to the other IPJs.



42 Palpation of first CMCJ.

43 Palpation of thumb MCPJ.

(42,43)

Additional tests

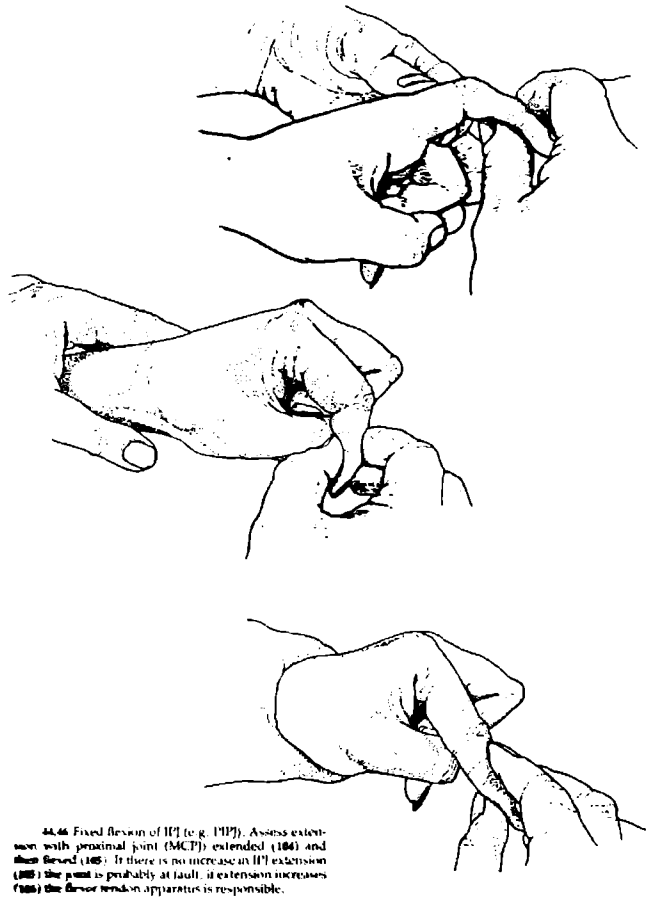
Stability of small joints

Stability of IPJs is tested by holding the phalanx,s each side of the joint and moving one laterally while the other is held in position. Normally there is little lateral movement,

irrespective of whether the IPJs is flexed or extended. Stability of MCPJs is tested by forced lateral movement while the MCPJs is fully flexed (MCPJ collateral ligaments tighten only in flexion, in extension, they are lax and permit marked lateral movement).

Fixed flexion of IPJ

To determine whether this results from problems with the flexor tendon apparatus or with the capsule/joint itself, assess the degree of flexion with the proximal joint in full extension (44). Then flex the proximal joint, relaxing the flexor tendons (45), and again assess the range of movement in the joint of interest. If there is now more movement than before (46) it suggest that the flexor tendon apparatus be primarily at fault. If, however, the range of movement is equally reduced with the proximal joint flexed or extended, it is more likely that restriction is due to capsular joint damage.



44-46 Fixed flexion of IPJ (e.g. PIPJ). Assess extension with proximal joint (MCPJ) extended (184) and then flexed (185). If there is no increase in IPJ extension (185) the joint is probably at fault, if extension increases (186) the flexor tendon apparatus is responsible.

(44,45,46)

De Quervain's tenosynovitis

In this condition a localized area of tenderness may be detected in a line along the lateral border of the distal radius. Increased temperature and linear soft-tissue swelling may accompany this. A useful stress test is to ask the patient to grasp the thumb in the palm while

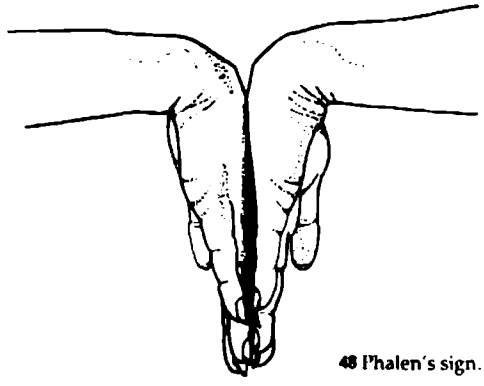
the examiner cautiously performs passively lateral flexion to the ulnar side (47). This maneuver can be uncomfortable in normal individuals but in De Quervain's tenosynovitis it causes marked pain (Finklestein's test).

47 Finkelstein's test.

(47)

Carpal tunnel syndrome

The median nerve gives off a sensory branch (supplying skin on the radial side of the palm) above the wrist before it passes through the carpal tunnel. Compression in the tunnel therefore causes numbness/dysaesthesia and altered sensation only in the thumb, index and middle fingers. The patient's symptoms may be reproduced (1) by percussion over the anterior wrist distal to the proximal skin crease (Tinel's sign), or (2) by asking the patient to sustain forced flexion of the wrist for at least one minute (Phalen's test; 48).



48 Phalen's sign.

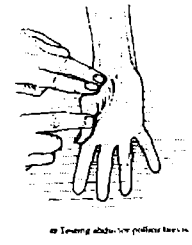
(48)

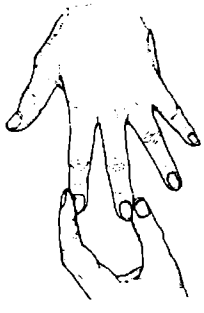
The median supplies the lateral two lumbricals, opponens pollicies, abductor pollicies brevis and flexor pollicies brevis ('LOAF'). To determine early weakness, test abductor pollicies brevis (always median nerve supply) by asking the patient to raise the thumb vertically from a flat supplanted hand against resistance, feeling the muscle as it contract (49). Asking the patient to oppose the thumb and index finger as the examiner tries to pull through the loop with an index finger tests later diminution of thumb opposition power. Wasting of abductor brevis and opponens may cause conspicuous hollowing of the outer thenar eminence.

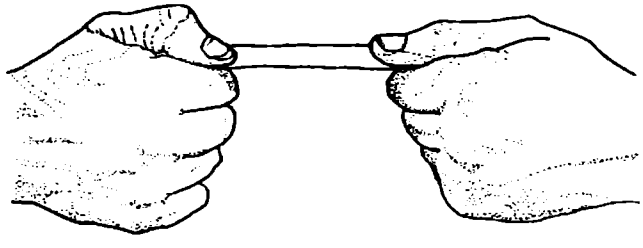
Ulnar nerve lesion

Compression is usually at the elbow but may also occur, (± combined carpal tunnel syndrome) at the wrist as the nerve passes through Guyon's canal (formed by the pisohamate

ligament bridging the pisiform and hamate). The ulnar gives off two sensory branches above the wrist (supplying the palmar and dorsal surfaces of the hand): therefore, compression in Guyon's canal causes altered sensation only in the little and ring fingers (± small muscle changes).
For early ulnar nerve lesions look for weakness (± wasting) of the first dorsal interosseous: with the hand flat, ask the patient:
To abduct the index against resistance, comparing one side with the other. Later weakness of all dorsal interossei and abductor digiti minimi (ulnar nerve; C8, T1) and all palmar interossei (ulnar nerve; C8, T1) leads to weak abduction and adduction of the second-fifth fingers. To test these movements the hand must be flat since the long extensors and flexors acts to some extent as abductors and adductors (50). Apart from resisted active movement, adduction can be tested, by trying to remove a strip of paper held between the patient's adducted fingers. Adductor policies brevis is also supplied by the ulnar nerve and may be tested by asking the patient to grasp a piece of paper between the adducted thumb and index finger: as the examiner attempts to pull the paper away the terminal phalanx of the thumb will flex due to weakness of the adductor and unopposed pull of the flexor policies longus (Froment's sign: 51,52).







51,52 Froment's sign: (51) positive (thumb flexes); (52) normal (thumb adducts).

(49,50,51,52)

Radial nerve lesion

This causes weak wrist dorsiflexion if mild, wrist drop if severe, with wasting of the forearm muscles. Sensory loss on the dorsum of the hand is minimal.

Summary of examination of the hand

(1) Inspection at rest
- (a) Dorsal surface
  - Skin, nail changes
    - Swelling (joint synovitis, tenosynovitis)
  - Deformity
  - Wasting
  - Attitude
- (b) Palmar surface
  - Skin changes (erythema, Dupuytren's)
  - Swelling (tenosynovitis)
  - Wasting
- (c) From side, fingers outstretched
  - Tendon rupture, slip
  - Deformity (Volar subluxation of the wrist and MCPJs)
  - Wrist swelling (tuck sign)

(2) Inspection during usage
- (a) Power grip
- (b) Fine precision pinch

(3) Palpation
- (a) Increased warmth
- (b) Each joint in turn (swelling, joint-line tenderness, crepitus, movement)
  - Radiocarpal joint
  - Inferior radioulnar joint
  - Second-fifth MCPJs (+ metacarpal squeeze, flexor tendon sheath crepitus,)
  - Second-fifth IPJ (+ flexor tendon sheath crepitus)
  - Thumb joints: first CMCJ, MCPJ, IPJ

Suggested Reading: Michael Doherty. John doherty. Clinical examination in rheumatology, 33-51, 1992
HAND AND WRIST PAIN

Dupuytren's Contracture

Summary of Diagnosis

- Diagnosis is based on the identification of nodular or string like palmar fibrosis.
- A "Dupuytren's diathesis" is suggested by the association of "knuckle pads,"peyronie's disease, and plantar Fibrotic nodules.
- Diabetes should be ruled out when two or more of the following overlap: Trigger finger, carpal tunnel syndrome, lateral epicondylitis, and frozen shoulder.
- Atypical inflammatory cases suggest an underlying neoplasm (Medsger's syndrome).

Dupuytren's contracture

Key Points

- Its cause is unknown.
- Middle-aged and elderly individuals are affected.
- There is fibrosis of the palmar fascia. Other fibrotic processes are often present.
- Progression is unpredictable.
- Systemic and local therapies (drugs, injections) are ineffective or of-unproven value.
- Functional limitation or cosmetic concern should trigger consultation with a hand surgeon.
- Palmar fasciectomy has excellent results.

Ganglia

Summary of Diagnosis

- Diagnosis is based on the characteristics of the lesion on physical examination: smooth surface, tense, typical location around the wrist are characteristic.
- Deep ganglia require ultrasound investigation: homogeneous, smooth- walled lesions represent ganglia.
- MRI is indicated when malignancy is suggested clinically or by heterogeneous findings on ultrasound examination.
- A clear, highly viscous fluid is characteristic of ganglia.

Ganglia

Key Points

- Benign cystic lesions with a mucoid content that are most frequently found in hand and wrist.
- Disfiguration and compressive neuropathies are important consequences of ganglia.
- Conservative treatment includes aspiration of the jell-like contents and Corticosteroid infiltration.
- Success rate of conservative treatment is about 50%.
- Surgical treatment includes removal of the cyst, the feeding duct, and its capsular attachment.
- The success rate of surgical treatment is about 90%.

Trigger Finger (digital stenosing tenosynovitis)

Summary of Diagnosis

- Pain, irregular motion, or snapping at the distal palm caused by motion of one or a few digits.
- Pediatric cases and late-adult cases often present with a digit incarcerated in flexion.
- A nodule is usually felt at the A1 pulley.
- No evidence of inflammation should be present.
- Metabolic causes must be ruled out when multiple digits are involved.

Trigger finger (digital stenosing tenosynovitis)

Key Points

- Idiopathic cases usually involve one digit.
- Multiple finger involvement should raise the possibility of missed systemic synovitis (RA, psoriatic, etc.), diabetes, hypothyroidism, or some unusual metabolic or infiltrative condition.
- One or more Corticosteroid infiltration succeed in over 90% of idiopathic cases.
- Diabetic stenosing tenosynovitis fails to respond to Corticosteroid infiltration in 50% of insulin-dependent cases and about 25% of non-insulin-dependent cases.

De quervain's tenosynovitis

Summary of Diagnosis

- Female predominance.
- Pain in the radial wrist
- A thickened EPB/APL tendon sheath.
- A positive Finkelstein's test.

De quervain's tenosynovitis

Key Points

- Fibrotic stenosis at the APL/EPB tendon sheath.
- The "bracelet syndrome," carpal tunnel syndrome, radiculopathy, inflammatory and infiltrative tenosynovitides, and carpometacarpal and radiocarpal osteoarthritis must be ruled out.
- Most cases respond to Corticosteroid infiltration.

Differential Diagnosis of radial wrist pain

	Location	Paresthesias	Tinel sign	Torque test	finkel-man	X-rays
Dequervain Tenosynovitis	*Anterior* border of snuffbox	*No*	*Negative*	*Negative*	*Positive*	*Positive* (STS)
Bracelet Syndrome "SRN"	outer distal forearm wrist, hand	Yes	Positive	Negative	Negative	Negative
Carpal tunnel Syndrome	Radial 3-digits	Yes	Positive	Negative	Negative	Variable
First CMC Osteoarthrosis	Base of-thumb	No	Negative	Positive	Negative	Positive
Radiocarpal OA	radial-wrist	No	Negative	Negative	Negative	Positive

Acute digital tenosynovitis

Summary of Diagnosis

- Finger held in semiflexion.
- Volar redness, swelling, and tenderness.
- A preceding puncture wound in septic tenosynovitis; underlying scleroderma or mixed connective tissue disease in calcific tenosynovitis.
- Proximal extension depends on the digit
- X-rays: amorphous calcium deposits are present in calcific tenosynovitis.

Acute digital tenosynovitis

Key Points

- The condition may be septic or caused by basic calcium crystals (calcific tendinitis).
- Any break of the skin in calcific tendinitis should make the case suspicious of coexistent supporative infection.
- Early diagnosis and treatment is essential.
- A hand surgeon should be immediately notified of the case.
- Suppurative digital tenosynovitis is treated with systemic antibiotics and surgical drainage.
- Calcific tendinitis is treated with a splint plus NSAID or IM ACTH.

Proliferative tenosynovitis

Summary of Diagnosis

- With exception of RA and other systemic synovitides, diagnosis can only be guessed at clinically.
- The overall yield of x-ray studies is low.
- Tuberculosis should be considered in immunosuppressed patients, particularly if they have emigrated from endemic areas.
- Diagnosis in single sheath tenosynovitis requires surgery (tenosynovectomy) with appropriate tissue handling including routine histology, gram, acid–fast, and PAS stain, and bacterial, mycobacterial, and fungal cultures.

PATIENT 1: A 60-year-old man presented with a rapidly enlarging lump in the valor aspect of the left index finger developing during past 3 weeks. The patient felt otherwise well .On examination the mass was fleshy and interfered minimally
with finger motion. Rather than a proliferative tenosynovitis it was felt that the patient probably had a soft tissue sarcoma. A chest x-ray showed a large mass in the left lung field. Excisional biopsy revealed a monophasic synovial sarcoma. A needle biopsy of the lung mass obtained, revealing identical tissue.

Proliferative tenosynovitis

Key Points

- It may involve one or multiple tendon sheaths.
- Single sheath tenosynovitis is most often caused by chronic infection.
- Diagnosis in single sheath tenosynovitis requires tenosynovectomy and appropriate histologic and microbiologic studies.

- Complications of Proliferative tenosynovitis include tendon rupture, compressive neuropathies and in some cases, destruction of adjacent bones and joints.
- Treatment depends on the etiology. Corticosteroid infiltration are quite useful in residual rheumatoid tenosynovitis. Tenosynovectomy is curative in certain localized processes. Antibiotic therapy may be required.

Carpal tunnel syndrome: patient evaluation

HISTORY	LABORATORY (if suggested clinically)
Occupational factors?	TSH
Pregnancy?	ESR
Diabetes?	RF
Hypothyroidism?	ANA
Acromegaly?	Serum protein electrophoresis
Arthritis?	

RECURRENT OR NONRESOLVING CARPAL TUNNEL SYNDROME AFTER SURGICAL RELEASE

Incomplete section of flexor retinaculum
Carpal tunnel mass (ganglia, hypertrophic scar, etc.)
Synovitis
Diabetes
Incomplete diagnosis (double-crush lesions)
Erroneous diagnosis (neuropathy, particularly in diabetes)

Carpal tunnel syndrome

Causes of carpal tunnel syndrome

Idiopathic
Pregnancy
Occupational (repetitive wrist motions)
Diabetes
Myxedema
Acromegaly
Chronic synovitis (RA, polymyalgia rheumatica, psoriatic arthritis, SLE, eosinophilic fasciitis)
Crystal-induced synovitis (gout, pseudogout
Median nerve enlargement (leprosy)
Soft tissue infection
Hematoma (hemophilia)
Fractures (especially Colles)
Amyloidosis (LA, hemodialysis related)
Tumoral calcinosis
Tumors (ganglia, Pigmented villonodular synovitis, synovial sarcoma, other)

Carpal tunnel syndrome

Summary of Diagnosis

- Pain and paresthesias in median nerve territory.
- Nocturnal symptoms.
- Hand numbness and clumsiness.
- Positive provocative tests: Phalen's test, Tinel's test, or McMurthry's sign.
- EMG and nerve conduction studies if provocative tests are negative and in the preoperative evaluation of patients.
- Assessment, by history and physical examination, of possible underlying etiologies.
- CBC, ESR, TSH, serum protein electrophoresis in the older age group.

Carpal tunnel syndrome

Key Points

- As important as diagnosing CTS is searching for its etiology.
- CTS due to pregnancy, hypothyroidism, and synovitis responds well to a splint plus treatment of the underlying condition.
- Idiopathic and diabetic CTS may transiently respond to conservative therapy, but surgical release is eventually required in most cases.
- A wrist splint in the neutral position is the mainstay of conservative therapy.
- Corticosteroid infiltrations are a helpful adjunct in rheumatoid artheritis and other synovitides.
- Surgical release, whether open or endoscopic, is indicated in failures of conservative therapy, when there is evidence of significant neuropathy, and in acute CTS.
- The chosen procedure should be the one the surgeon is most experienced with.
- Following surgery, resolution of pain and paresthesia occurs within a few weeks; improvement in strength takes several months to 2 years.

Suggested Reading

Juan J. Canoao/ Rheumatology in primary care

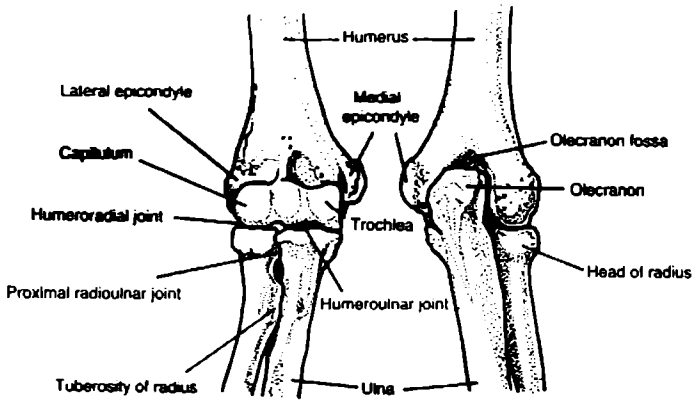
ELBOW PAIN

The primary role of the elbow is to permit accurate spatial positioning of the hand. The elbow anchors the strong flexors and extensors of the wrist and hand, and once the shoulder has grossly directed the arm, elbow movements permit fine adjustment to limb height and length. In addition forearm rotation (at elbow and wrist) helps place the hand in the most effective functional position.

Heavy demands on forearm muscles and poor soft-tissue protection make the elbow particularly prone to enthesopathy and bursitis. Although not commonly involved in inflammatory arthropathies (e.g. rheumatoid). The elbow is an uncommon target site for arthritis other than hemophilia and syringomyelia-associated Charcot arthropathy. Primary osteoarthritis is distinctly unusual.

Functional anatomy

The elbow is a compound joint comprising three articulations: the humeroulnar and humeroradial joints (permitting flexion/extension) and proximal radioulnar joint (which with the humeroradial and inferior radioulnar joints, permit rotation: 53, 54).

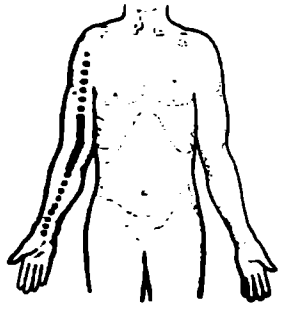


53,54 The elbow joint: (53) anterior view; (54) posterior view.

(53,54)

Humeroulnar joint (trochlea) joint forms a uniaxial hinge between the trochlea of the humerus and the ulna trochlea notch. When the elbow is fully flexed (about 145 degree) the longitudinal axes of the upper arm and forearm are parallel; however, due to the shape of the trochlea, as the arm extends in the anatomical position (palms forward) the upper arm and forearm form a valgus 'carrying angle' at the elbow (55). This is wider in females (10-15 degree) than in males (5degree) and may be increased (cubitus valgus) as a developmental abnormality (e.g. part of Turner's syndrome).

The humeroradial joint is a modified uniaxial hinge (allowing rotation and flexion/extension), corresponding to a ball and socket between the capitulum of the humerus and the concave fovea of the radial head: during supination/pronation the radial head revolves on the capitulum.

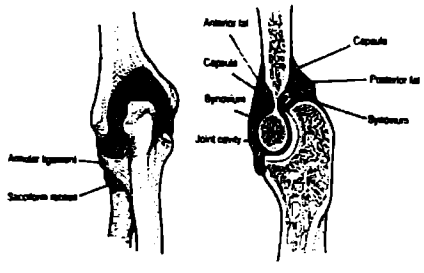


55 Extension of the elbow, showing 'carrying angle'

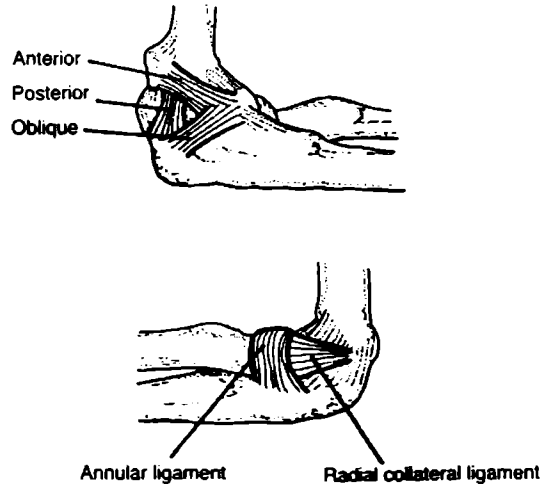
(55)

The superior radioulnar joint comprises the pivot between proximal rim of the radial head and the ulnar radial notch, together with the cartilage-lined annular ligament that encircles the radial head. The strong interosseous membrane of the forearm prevents parallel displacement of the ulna and the radius and transmits longitudinal stresses from one bone to the other.

The three joints share a common capsule (56, 57). On the radius the capsule extends as the sacciform recess beneath the annular ligament. Large intracapsular fat pads lie in the three fossae of the humerus, buttressing against extremes of movement. The shape of the trochlear joint, the annular ligament, and the cord-like radial and fan-shaped ulnar afford stability collateral ligaments (58, 59). The latter, together with flexor carpi ulnaries, form the cubital tunnel through which the ulnar nerve passes.



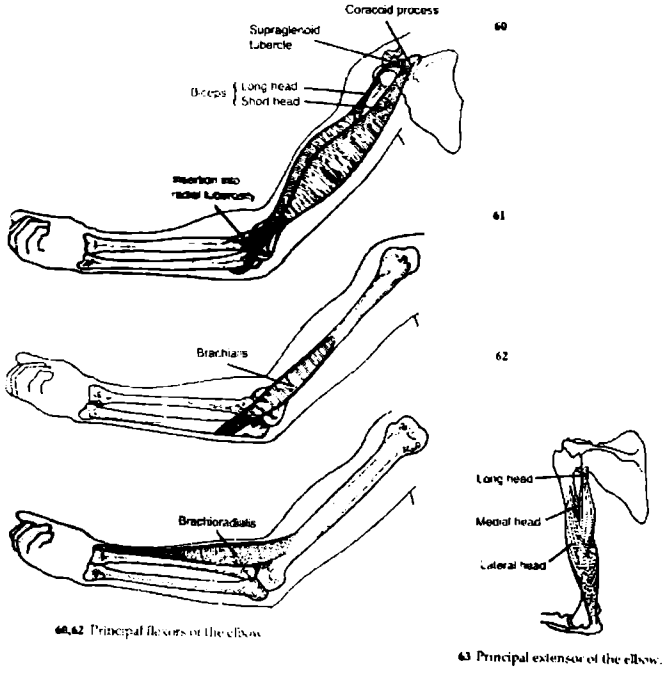
56,57 Synovial outline of the elbow: (56) posterior view; (57) section through the joint



58,59 Ligaments around the elbow: (58) the three portions of the fan-shaped medial ligament; (59) the lateral ligament.

(56,57,58,59)

The axis of flexion/extension runs through the two epicondyle; muscles in front of this axis act as flexors, those behind as extensors. Many of the muscles act on several joints-their action at the elbow varies according to the attitude of neighboring joints.
Principal elbow flexors (60-62) are the biceps (inserting into the radial tuberosity thus supinating as well as flexing: 60), the brachilis (a short pure flexor: 61), and the brachioradialis (a flexor with the forearm in neutral rotation: 62). The principal elbow extensor is the triceps, joining the scapula (long head) and humerus (medial, lateral heads) to the olecranon (63). Pronation is principally via the pronator teres (the anterior interosseous nerve passes between its two heads) and the pronator quadratus. Although the biceps is the strongest supinator in flexion, the supinator muscle acts in any flexion/ extension position (in 30% of people the posterior interosseous nerve passes through the fibrous arcade of Frohse between the two heads of the supinator and may rarely become compressed 'radial tunnel syndrome' causing weakness of the forearm extensors but no sensory loss).
Normal active flexion is about 145 degree from the fully extended position: passive flexion often achieves another 10-15 degree. The elbow hyperextend 10 degree in many- normal women (more in hypermobility syndrome). Muscular individuals may lack 10- degree at each end of the range.



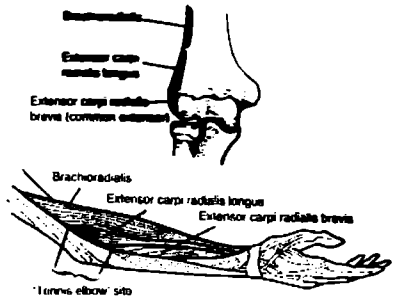
60,62 Principal flexors of the elbow.

63 Principal extensor of the elbow.

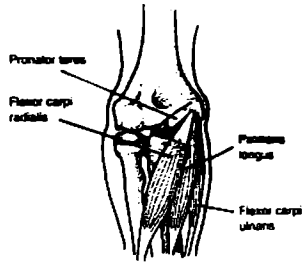
(60,61,62,63)

The bones and ligaments around the elbow anchor the forearm muscles the origins of extensors carpi radialis brevis and longus ('fist clenchers') at the lateral epicondyle (with the brachioradialis origin just above) are the usual site of pain in 'lateral epicondylitis' (64, 65). Both muscles are weak elbow flexors but principally extend the wrist, optimizing the action

of the flexors in the power grip. The common tendon insertion at the medial epicondyle (for pronator teres, flexor carpi radialis, palmaris longus, and flexor carpi ulnaris) is, similarly, the site of pain in 'medial epicondylitis' (66).



64,65 Tendon insertions around the lateral epicondyle.



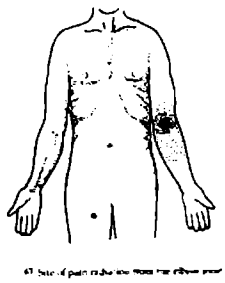
66 Tendon insertions around the medial epicondyle.

(64,65,66)

Several bursa, none communicating with the joint, occur around the elbow, the largest being the superficial olecranon bursa overlying the olecranon prominence.

Symptoms

Pain from the three elbow compartments is usually felt maximally at the elbow, close to its origin: severe arthropathy may cause radiation of pain down the forearm and, to a lesser extent, proximally to the upper arm (67). Pain of lateral epicondylitis ('tennis elbow') is usually maximal close to the epicondyle, radiating down the outer aspect of the forearm towards the wrist (68): it is particularly marked during power grip with the wrist extended.



(67)

Medial epicondylitis ('golfer's elbow') causes pain maximum around the medial epicondyle, radiating down the flexor aspect of the forearm towards the wrist (69). Pain from olecranon bursitis is well localized, usually showing no clear relationship to passive or resisted elbow movement: it may be provoked by leaning the elbow on a table, or on flexion at the elbow when tight clothing is worn.

Four dermatomes cover sensation around the elbow (70). Pain referred from above is usually ill defined at the elbow, with the site of maximum intensity elsewhere: it may originate from glenohumeral or rotator cuff lesions or from root entrapment (C5 or C6; less commonly, T1 or T2). Pain may refer up towards the elbow from de Quervain's tenosynovitis, carpal tunnel syndrome or, rarely, severe wrist arthropathy.

Examination

Inspect from in front and from behind with the patient's arm hanging by the side; then inspect during active flexion, extension, and supination/ pronation; then palpate.

Inspection at rest:

From behind (71) the most prominent feature is the olecranon process; para-olecranon grooves separate this from epicondyle, the medial epicondyle being more prominent than the lateral (the three bony prominences form a straight line with the elbow extended). *From in front* the triangular cubital fossa (72) forms a hallow bounded superiorly by the biceps and its tendon, medially by the pronator teres and the common flexors, laterally by the brachioradialis, and the floor comprising the brachialis muscle and tendon (+joint capsule and supinator). The fossae contains the brachial artery and veins, the median and musculocutaneous nerves, and superficially the median cubital vein (joining the medially placed basilic to the cephalic vein).

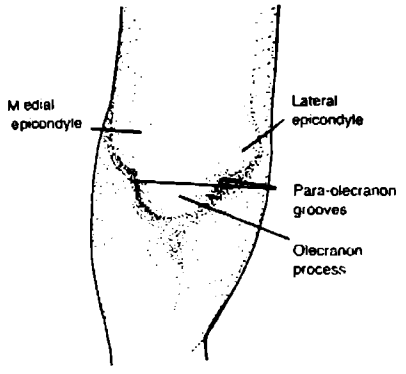
With the patients arms extended by the side, examine from infront and then from behind for the following.

Skin changes

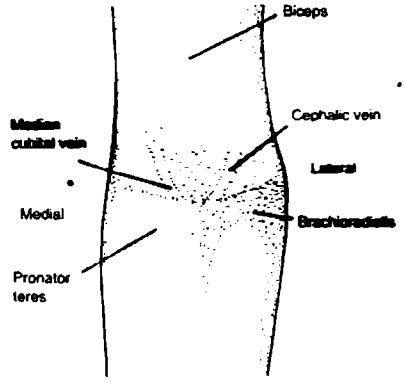
For example, erythema (confined in bursitis or over whole joint) and scars: the extensor aspect is a common site for psoriasis, nodules, and pressure sores (73).

Swelling

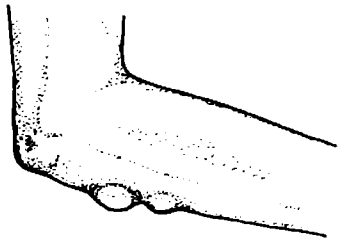
Synovial swelling is most apparent over the radial head anteriorly, and over the para-olecranon grooves posteriorly (medial> lateral): if marked, the whole elbow origin appear swollen. Olecranon bursitis causes localized smooth swelling around the olecranon prominence (74): nodules within it may produce a lumpy contour.



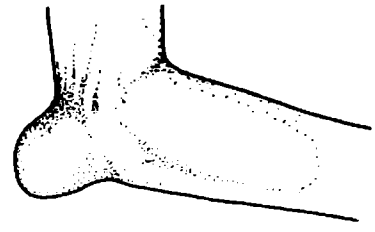
71 Surface landmarks of the elbow – posterior view.



72 Surface landmarks of the elbow – anterior view.



73 Nodules around the elbow.



74 Olecranon bursitis.

(71,72,73,74)

Deformity

In particular, cubitus valgus or varus, and fixed extension (from in front); and posterior subluxation of the olecranon on the humerus (from behind).

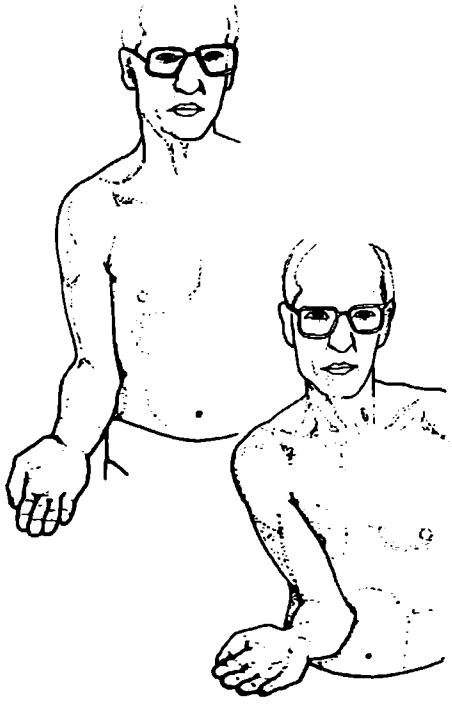
Attitude

A capsular pattern of restriction usually affects flexion more than extension, with supination/pronation affected last: in the presence of synovitis or effusion the patient is therefore most comfortable with the elbow positioned in flexion (about 45-70 degree).

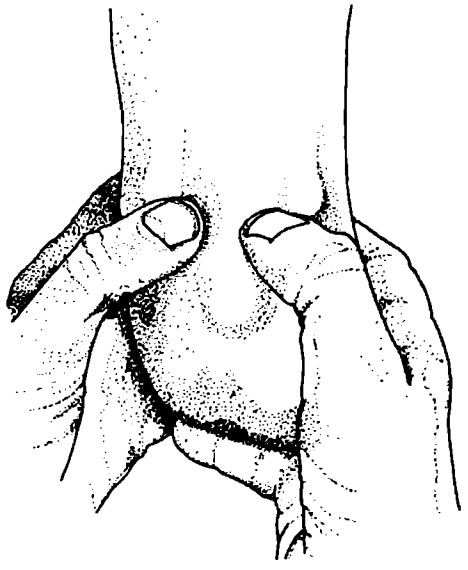
Inspection during movement

Ask the patient to bend up the elbow, assessing active flexion and extension for restriction and presence of stress pain. Then ask the patient to supinate and pronate the hands with the elbows tucked into the side at 90-degree flexion (75). If proximal or distal radioulnar (or humeroradial) joints are compromised, these movements may be painful and/or reduced: the

patient often undertakes a trick maneuver, adducting the elbow across the abdomen to rotate the ulnar and thus increase supination (76).



75,76 Supination with flexed elbow: (75) normal; (76) trick manoeuvre (limited supination).



77 Palpation of the elbow (posterior approach).

(75,76,77)

Palpation

1-from behind

Stand behind the patient with their shoulder extended and elbow pointing backwards in mild flexion. Feel for:

Increased warmth

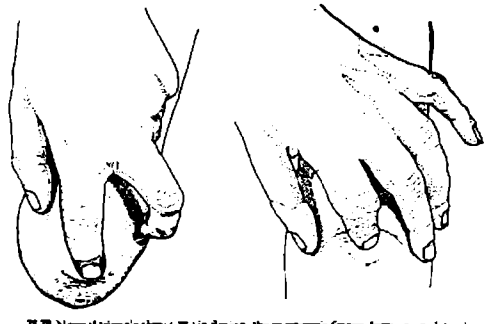
Pass the back of the hand over the distal upper arm, elbow, and forearm to detect increased warmth over the para-olecranon grooves and olecranon bursa.

Swelling, tenderness

Synovitis produces palpable soft-tissue swelling in the para-olecranon grooves: firm palpation at these sits may produce capsular tenderness (77). Feel for swelling of olecranon bursitis with the elbow extended: as the arm goes into flexion the bursa becomes more tense and prominent. A balloon sign will confirm a moderate-to-large fluid collection. Palpate for nodules along the extensor forearm border.

Deformity

The landmarks of the medial and lateral epicondyle are easily felt. Place a thumb and two fingers of one hand over the olecranon, medial epicondyle, and lateral epicondyle. In extension the three fingers form a straight line, in flexion they form an equilateral triangle (78, 79). Loss of such symmetry on flexion implies loss of height at the elbow due to cartilage and bone attrition ('triangle sign').



78,79 Normal triangle sign (78) in flexion, the examiner's fingers form an equilateral triangle; (79) in extension, the examiner's fingers form a straight line.

(78,79)

Crepitus

Place a finger in each para-olecranon groove to feel for crepitus from the humeroulnar and humeroradial joints while the patient flexes or extends.

Periarthicular structures

On the medial side feel for the ulnar nerve below the epicondyle for thickening and disproportionate tenderness: this is the most common site for ulnar nerve entrapment. The medial supracondylar lymph nodes may be palpable if enlarged.

2- from in front

Increased warmth

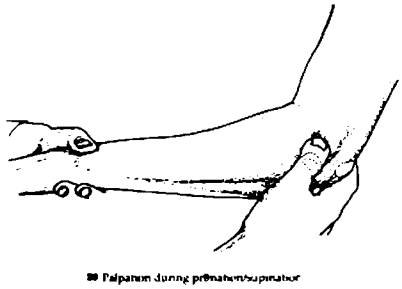
Again use the back of the hand to feel for increased warmth over the radial head region.

Swelling

Palpate over the radial head for the soft-tissue swelling of synovitis. Occasionally, palpate anterior synovial extensions may almost fill the cubital fossae (predisposing to partial radial nerve palsy from pressure on the posterior interosseous nerve).

Proximal radioulnar joint (tenderness, laxity, crepitus, passive movement)

Pressure over radial head may produce capsular/joint line tenderness and if there is associated joint damage and annular ligament laxity, excessive movement of the radial head with crepitus. Keeping the thumb over the radial head region, passively supinate and pronate with the other hand (the thumb is placed over the ulnar styloid region, 80) to detect crepitus (both joints), assess range of passive movement, and inquire concerning pain.



(80)

Passive movement humeroulnar joint

Assess the range of passive flexion and extension (looking for restriction- stress pain) and compare with active movements: similar restriction suggests synovitis; a greater passive range favors a neuromuscular rather than joint cause.

Epicondylar tenderness, resisted active movement

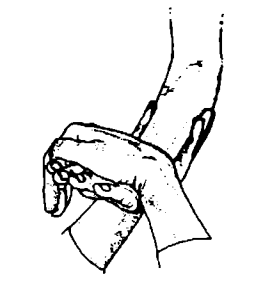
For tennis elbow, palpate for tenderness over the common extensor origin at the lateral epicondyle: in some cases tenderness is more distal, occurring over the radial head region. Confirmation is by resisted active wrist extension, which reproduces the pain (81).

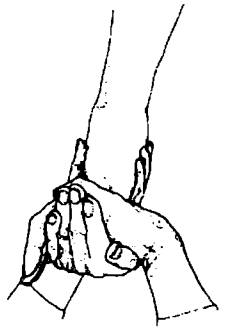
For golfer's elbow, palpate for tenderness over the medial epicondyle at the insertion of the wrist flexor/ pronator group (pronator teres, flexor carpi radialis, palmaris longus, and flexor carpi ulnaris). Resisted active wrist flexion with the hand supinated reproduces the pain (82).

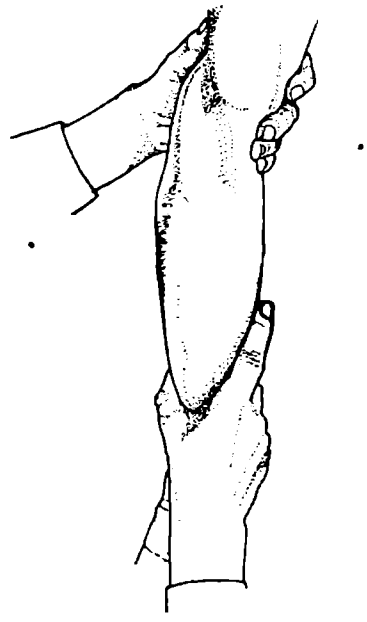
Additional tests

Collateral ligament stability

This may be tested by flexing the patient's elbow to about 20-30 degree and then holding the elbow in one hand while applying a progressive varus force (testing the lateral ligament) followed by a valgus force (medial ligament) on the distal forearm (83), noting any pain or increased lateral movement.







83 Testing collateral ligament stability.

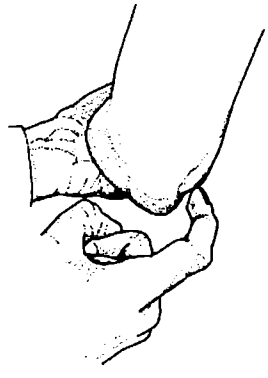
(81,82,83)

Tests for nerve entrapment at elbow:

The ulnar is affected more commonly than median or radial nerves. Helpful tests include:

Tinel's sign

Light percussion over the ulnar nerve as it travels through the para-olecranon groove produces tingling in an ulnar distribution in the forearm/ hand distal to the point of compression (84).



84 Tinel's sign.

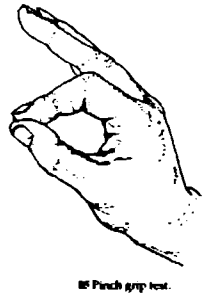
(84)

Elbow flexion test

The patient holds the elbow in full flexion for 5 min. Tingling in an ulnar distribution again suggests a cubital tunnel syndrome.

Pinch grip test

The patient attempts to oppose the tips of the index finger and thumb. If the normal tip-to-tip pinch is replaced by a pulp-to-pulp pinch (85), reflecting impairment of index finger and thumb flexors, entrapment of the anterior interosseous nerve, as it passes between the two heads of the pronator teres, is suggested ('anterior interosseous nerve syndrome'). If the median nerve is compressed just prior to the anterior interosseous division, the flexor carpi radialis, palmaris longus and flexor digitorum muscles are also weak ('pronator teres syndrome') In both cases there is sensory impairment in a median nerve distribution. Rarely the median nerve is compressed as it passes (± the brachial artery) beneath the ligament of Struthers, an anomalous band in 1% of people that runs from a spur on the humerus to the medial epicondyle; in this case ('humerus supracondylar process syndrome') the pronator teres is also involved (± vascular, as well as neurological, symptoms).



(85)

SUMMARY OF ELBOW EXAMINATION

(1) Inspection at rest
- Skin changes
- Swelling (synovitis, bursitis, nodules)
- Deformity (valgus, varus, posterior subluxation)
- Attitude

(2) Inspection during movement
- Flexion/extension
- Supination/pronation

(3) Palpation from behind
- Warmth
- Paraolecranon grooves
  - Swelling
  - Tenderness
  - Crepitus
  - Deformity (triangle sign)
  - Palpable ulnar nerve, enlarged nodes

Olecranon region (bursa, nodules)
(4) Palpation from in front
Warmth
Swelling
Radial head (proximal radioulnar joint)
Tenderness
Laxity
Crepitus
Passive movements
Supination/pronation
Flexion/extension
Peri-Epicondylar tenderness
Resisted active wrist extension (tennis elbow)
Resisted active wrist flexion (golfer's elbow)

Suggested Reading

Michael Doherty, John Doherty. Clinical examination in Rheumatology, 52-62. 1992

ELBOW PAIN

CAUSES OF LATERAL ELBOW PAIN

Lateral epicondylitis

Compressive neuropathy of the deep branch of the radial nerve
Enthesitis
Radiohumeral arthritis
Fibromyalgia
C5 or C6 radiculopathy

Tennis elbow

Summary of Diagnosis

- Tennis elbow is diagnosed clinically based on the presence of lateral elbow pain, tenderness just distal to the lateral epicondyle, and pain on resisted wrist dorsiflexion.
- X-rays should be obtained to rule out calcific tendinitis, exostosis, osteoarthritis at the radiocapitellar joint, and malignancy. A traction spur is present in 20% of cases.
- Infrared thermography, useful as a research tool, is unnecessary in clinical practice.

Treatment

- Avoid overuse
- Hold weights in supination only
- Compressive band
- NSAID
- Isotonic and isometric exercises fore the entire extremity
- Deep massage and manipulation?
- Corticosteroid infiltration?

Key Points

- Lateral elbow pain that is reproduced by resisted wrist dorsiflexion.
- An enthesopathy caused by repetitive traction injury.
- The condition is self-limited in mild cases.
- Treatment is empirical. If tendon overuse can be stopped, symptoms cease. Counterforce bracing in the upper forearm discourages full use of the muscles and helps relieve pain. Capsaicin and NSAID may afford relief. Corticosteroid infiltration may be used after failure of conservative therapy.
- Treatment failures may represent diagnostic errors. Have a rheumatologist or orthopedic surgeon review the case.
- Surgery in refractory bona fide cases of refractory tennis elbow has a success rate of near 90%.

PATIENT 2. A 30-year-old man was referred for evaluation of a protracted elbow. Symptoms had been present for a year and had been progressive despite several Corticosteroid infiltration and formal physical therapy. Unusual features of his condition included severe pain and about 20 degrees of elbow flexore contracture that was attributed to chronicity and lack of use of the extremity. Flexion was full, but extension had a soft tissue endpoint at 20 degrees flexion consistent with impingement-type limitation. Some soft tissue bulging was noted posterolaterlly between the olecranon process and the lateral epicondyle left tennis. Because a soft tissue mass was strongly suspected, a bilateral CT scan was requested. However due to cost factors only the left side was examined and findings were considered normal. Given the clinical findings, a CT scan of the right elbow was once again requested, and on comparison with the left side a previously unidentified soft tissue lump was noted at the site of the bulge. The patient had a clear cell sarcoma.

Radial tunnel syndrome

Summary of Diagnosis

- Diagnosis is clinical based on the presence of deep aching pain in the posterolateral muscles of the forearm, tenderness at Frohse's arcade, and pain upon resisted supination of the passively dorsiflexed hand.
- EMGs and nerve conduction studies are normal.

Compressive neuropathy of the deep branch of the radial nerve

Summary of Diagnosis

- Diagnosis is clinical based on paresis of extension in digits 2 through 5, a positive tenodesis effect, and radial deviation of the wrist in dorsiflexion.
- EMGs show denervation of the paretic muscles.
- MRI gives the highest diagnostic yield in nonreumatoid cases. Ultrasound may be used as a screening procedure.

Radial tunnel syndrome

Key Points

- Radial tunnel syndrome, a pain syndrome involving the lateral elbow and forearm, is often confused with tennis elbow. Both lesions may coexist.
- Occupational or recreational factors are similar in both conditions.
- Their differentiation is based on the quality and distribution of pain, location of maximal tenderness, and triggering maneuvers.
- Conservative treatment is as in tennis elbow.
- Surgical treatment of chronic cases is often successful.

True compressive neuropathy

Key Points

- In a true compressive neuropathy of the radial nerve, neurologic findings prevail, namely, neurotic pain and paresis.
- The space occupying lesion may be obvious, such as elbow synovitis in RA, or it may be a primary expansive lesion (ganglia, sarcoma , etc.).
- Prompt orthopedic or surgical oncology consultation should be obtained if a focal lesion is shown.

Golfer's elbow (medial epicondylitis)

Summary of Diagnosis

- Golfer's elbow is diagnosed clinically based on a history of medial elbow pain plus the results of resisted wrist flexion and forearm pronation.
- Differential diagnosis includes calcific tendinitis, bone and soft tissue tumors, ulnar neuropathy, and radiating pain (T1).
- X-rays findings in golfer's elbow are normal. The study is useful to exclude alternative diagnosis.

Key Points

- Elbow pain at the medial epicondyle.
- Increased symptoms upon resisted wrist flexion and resisted forearm pronation.
- Association with repetitive manual activities.
- Treatment includes partial rest, activity modification, a forearm compression band, and exercises for the entire extremity, general conditioning, capsaicin, and NSAIDs.
- Corticosteroid infiltration is reserved for resistant cases.
- Surgical medial release is a last-resort treatment.

Cubital tunnel syndrome

Summary of Diagnosis

- Diagnosis is clinical based on symptoms, physical findings, and a positive result of provocative tests (flexion, compression, Tinel).
- Elbow x-rays including a cubital tunnel view should be routinely obtained.
- Electrodiagnosis is useful in selected cases.

Key Points

- Is a common cause of ulnar neuropathy.
- It should be distinguished from radiculopathy, thoracic outlet compression, and nerve entrapment at the wrist (Guyon's canal).
- A positive provocative test indicates nerve entrapment at the cubital tunnel.
- Persistent symptoms (dysaesthesia unrelieved by elbow extension) are an indication for surgery.
- Arcuate ligament section is effective in early neuropathy (sensory symptoms only). Late cases require anterior transposition of the ulnar nerve.

Olecranon bursitis

Summary of Diagnosis

- Diagnosis is usually self-evident from the characteristic saccular distension of the bursa.
- Exceptions to this rule are cases with extensive edema and cases that mimic acute arthritis.
- In bursitic edema, residual bursal distension at the elbow tip allows a correct diagnosis to be made. In pseudoarthritis cases, normal passive extension suggests bursitis.
- Etiology may be suggested by clinical findings, but overlapping features result in a high probability of error. Bursal fluid analysis including gross appearance, WBC count and differential, crystal search by polarizing microscopy, and culture is essential for diagnosis.

Key Points

- Is characterized clinically by a cystic swelling at the elbow tip.
- Etiology of bursitis may be suspected clinically, but aspiration is essential to diagnose bacterial infection and gout.
- Aspiration should be made from the lateral side to avoid chronic leaks and damage to the ulnar nerve.
- Gross appearance of bursal fluid may be deceiving. Hemobursa is nonspecific. Milky fluids may contain urate or cholesterol crystals. "Clear" fluids, although usually traumatic, may also occur in septic bursitis and gout.
- Upon similar disease stimuli subcutaneous bursae respond differently than joints. This includes lower WBC counts, a higher percentage of mononuclear cells, and a poorer mucin clot test.

PATIENT 3. A 50-year-old man was transferred to the author's hospital in shock and with a white lung. Three days before he had been seen at a walk-in clinic for evaluation of a swollen, slightly tender olecranon bursa. He was afebrile and felt well. Clear fluid was aspirated and sent for analysis and culture. In the same procedure, because the case appeared to be aseptic, 20 mg of methylprednisolone acetate was injected in the bursa. A day later there was local pain; the patient became febrile, extremely weak and dyspneic and upon evaluation at a local hospital toxic shock was diagnosed. Retrospectively, the original bursal fluid contained 2000 WBC/mm3 with 95% polys and gram stain was negative; however, S. aureus grew on culture. Upon transfer the bursa was inflamed and its top was crusted. Attempted bursal reaspiration

failed due to flocculent debris, and open drainage was undertaken. A picture of his feet shows the classic desquamation of toxic shock.

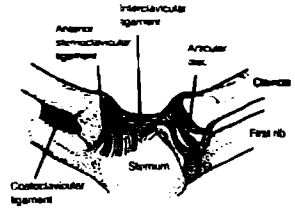
Suggested Reading

Rheumatology in primary care/ Juan J. Canoso

SHOULDER PAIN

The shoulder girdle comprises three joints (sternoclavicular, acromioclvicular, and glenohumeral) and one articulation (scpulothoracic).

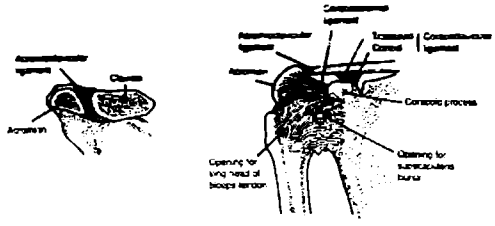
The *sternoclavicular joint* (SCJ) is a saddle-shaped synovial joint that connects the medial clavicle, manubrium sternum, and the cartilage of the first rib (86). It is divided into two cavities by a fibrocartilage disc. The capsule is strengthened by the sternoclavicular (anterior and posterior) and interclavicular ligaments; the costoclavicular ligament binds the undersurface of the clavicle to the first rib.



86 The sternoclavicular joint.

(86)

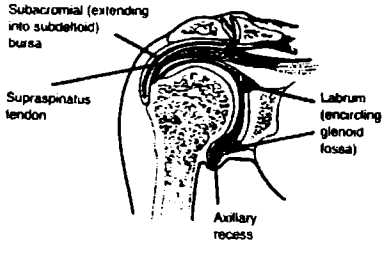
The acromioclvicular joint (ACJ) is a plan synovial joint angled to allow the clavicle to slide over the acromion (87, 88); a disc is variably present. Stability is due to the coracoclavicular ligament (comprising lateral 'trapezoid' and medial 'conoid' portions) and the acromioclvicular ligament. The joint has sensory branches of the suprascapular and long thoracic nerves.



87,88 The acromioclavicular joint. Ligaments and glenohumeral capsule.

(87,88)

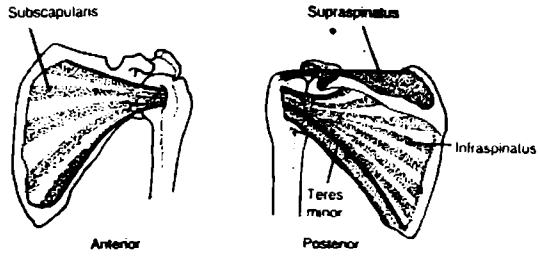
The glenohumeral joint is a multiaxial spheroidal synovial joint (89). Its range of movement, greater than that of any other joint, is permitted at the expense of stability.



89 The glenohumeral joint (in section).

(89)

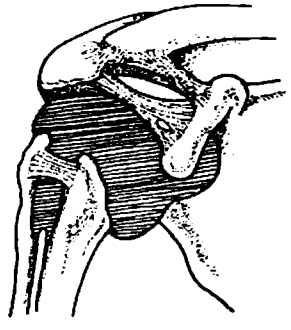
The glenoid fossa, though widened by the fibrocartilaginous labrum, is shallow the capsule is lax and thin, and there is no strong traversing ligaments. Stability primarily depends on the muscles and conjoining tendons of the rotator cuff (90, 91). Supraspinatus, infraspinatus and teres minor arise posteriorly on the scapula and insert into greater tuberosity; subscapularis arises anteriorly on the scapula and inserts into the lesser tuberosity. The deltoid and the rotator cuff form a mechanical couple: the rotator stabilize the humerus and cause the head to 'drop down' into the lower wider part of the glenoid cavity, converting the deltoids upward pull into a powerful abducting force.



90,91 Rotator cuff muscles

(90,91)

The joint is protected superiorly by an 'arch' formed by the coracoid process, the acromion, and the coracoacromial ligament. The lax capsule has a deep inferior fold and two openings (see 88); one allows the long head of biceps tendon to inter the bicipital groove (taking an extension of the synoviom with it as its sheath: 92), the other permits an outpouching of the synoviom to act as a bursa for the subscapularis.



92 Synovial limits, and extension around the biceps tendon (long head) in the bicipital groove.

(92)

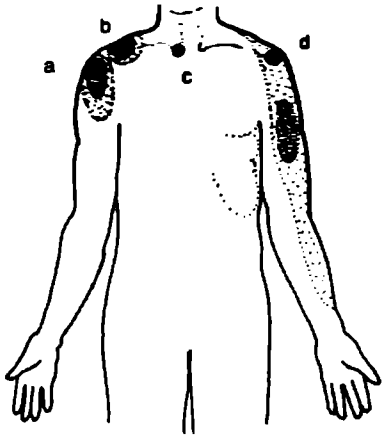
A large subacromial bursa permit smooth movement between the rotator cuff and undersurface of the acromion; it extends laterally into the subdeltoid bursa. The subacromial bursa communicates with the joint cavity in some normal individuals: since the Supraspinatus tendon forms the floor of the bursa and the roof of the capsule, any tear of the tendon/cuff is likely to lead to communication between the two. The subcoracoid bursa, between the coracoid and the capsule, may be separate or communicate with the subacromial bursa. The

suprascapular nerve supplies the superior and posterior parts of the joint and capsule and most of the rotator cuff; the axillary nerve supplies the anterior aspect of the joint and capsule.
The SCJ is commonly affected by major arthropathies (e.g. rheumatoid, osteoarthritis, seronegative spondyloarthropathy), and is an occasional site for sepsis, particularly in the immunocompromised. Although it is common to find abnormal signs at the SCJ, it is an uncommon site for symptoms. The ACJ is also often involved in major arthropathies (particularly osteoarthritis); unlike the SCJ, it is a common source of symptoms. The glenohumeral joint may be involved in inflammatory arthropathy-
(rheumatoid, seronegative spondyloarthropathies): Though an uncommon site for 'primary' osteoarthritis it is often involved in the subset of pyrophosphate arthropathy in the elderly. Rotator cuff injuries are exceedingly common, and the cuff and bursa can be directly involved in inflammatory conditions. Because of its close relationships, subacromial bursitis can result from pathology relating to the rotator cuff, ACJ, or the glenohumeral joint.
The scpulothoracic articulation is of little rheumathological interest. It is a common site of painless, reproducible grating, or clunking; such noises are, in general, abolished by moving the scapula laterally and merely reflect movement over uneven surfaces.

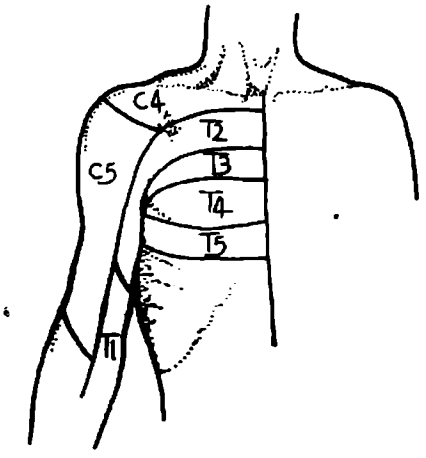
Symptoms

SCJ pain is usually well localized, with little radiation (93). The ACJ derives from C4 and produces pain close to its origin, with some radiation to the shoulder tip but no significant radiation to the arm. All glenohumeral joint structures, including the rotator cuff and the subacromial bursa, develop from the C5 sclerotome and produce pain maximal at the outer aspect of the upper arm close to the deltoid insertion; if severe, pain may radiate down the radial aspect of the forearm to the elbow (rarely to the wrist) and upward to the shoulder (rarely to the neck). such pain will be worsened by shoulder movements. Tendinitis of the long head of biceps also produces upper arm pain C5/C6.

Pain over the superior aspect of the shoulder and the C4/C5 distribution (94) may also be referred from the neck: such pain may involve the whole length of the arm-



93 Pain patterns around the shoulder: (a) bicipital tendinitis; (b) acromioclavicular joint; (c) sternoclavicular joint; (d) glenohumeral joint/rotator cuff/subacromial bursitis.



94 Dermatomes around the shoulder.

(93,94)

(± The hand), be worsened by neck movements (and only the extremes of shoulder movements), and be accompanied by sensory or motor impairment in the limb.

Radiation of pain into the arm associated with numbness, or paraesthesia, suggests compressive neuropathy (e.g. thoracic outlet syndrome, suprascapular or axillary nerve entrapment). Lesions involving or close the diaphragm may cause pain referred to the shoulder tip region, unrelated to shoulder movement. Myocardial pain may produce variable aching and pain down the arm.

Differentiation between glenohumeral-joint and rotator cuff disease is often suggested by the history (table 8). A typical rotator-cuff patient has often performed unaccustomed exercise with the involved arm (e.g. decorating the ceiling): the patient usually notices nothing at the time, but the following day complains of upper arm aching as a single regional pain syndrome. Pain is nonprogressive and is usually noted only during one or two movements of the arm (e.g. reaching up to a shelf); it is generally more an inconvenience than a major problem, and uncommonly interferes with sleep. Conversely, though acute monoarthritis of the glenohumeral joint can occur, arthritis at this site is usually part of an oligo- or polyarthritis that produce multiple regional pain problems. Pain is often insidious in unset without an obvious provoking event: it is variable but usually progressive, often keeps the patient awake, affects several (eventually all) shoulder movements, and tends to interfere greatly with simple activities of daily living. Acute subacromial or subdeltoid bursitis is often characterized by its speed of unset, the patient being unable to abduct the arm within just a few days.

Table 8. Comparison between typical symptoms arising from rotator cuff lesions and glenohumeral arthritis

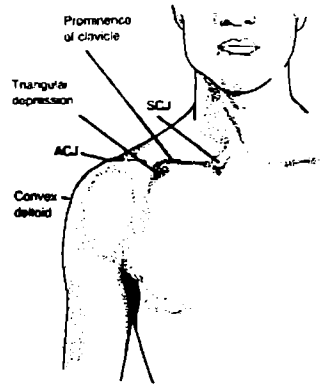
	Rotator cuff	Glenohumeral arthritis
Pain: onset	Acute	Insidious
Progression	Non-progressive	Variable, progressive
Movements affected	Few	Many
Provoking factor	Often apparent	Absent
Localized problem	usually	Uncommonly
Severity	+ +	+ + + +

Inspection at rest

Get the patient standing or seated on a chair or the side of the couch/bed, to permit inspection from in front and from behind.

Inspection from in front

Look at the ACJ region where the jugular notch above the manubrium is clearly evident (95). Look for erythema and swelling overlying the medial end of the clavicle: fluid from the joint will appear as a smooth rounded swelling; irregular swelling is more likely to be osteophyte. If the SCJ is subluxed the medial end of the clavicle comes anteriorly, medially and inferiorly across the sternum, appearing more prominent than usual (comparison between the two sides is helpful if unilateral). Look along the clavicle for irregularity and bony swelling (e.g. from an old fracture, Pajet's, or a primary or secondary tumor).

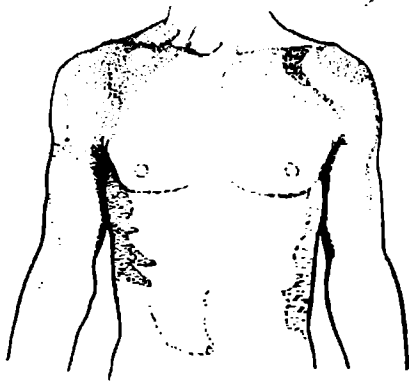


95 Normal surface landmarks (anterior view).

(95)

The site of ACJ may be apparent due to prominence of the lateral end of the clavicle, but in many subjects this is a flat joint with no surface landmark. Inspection over the approximate area proximal to the shoulder tip, however, may show erythema or swelling (fluid at this site is rare and swelling usually reflects osteophyte).

Inspect muscle bulk. All individuals, whatever their age, should have a convex, full deltoid. Large glenohumeral effusions are uncommon but if present may push anteromedially and present as a swelling which fills the usual triangular depression bordered superiorly by the lateral end of the clavicle, laterally by the medial curve of the deltoid, and inferiorly by pectoralis (96). A large subdeltoid bursa may cause undue prominence of the deltoid contour. The typical attitude of a patient with glenohumeral arthropathy, is for the shoulder to be held in internal rotation and adduction, with the hand folded across the abdomen as if in a sling; this is the most confortable position for minimizing intra-arthicular hypertension (conversely, when the examiner comes to palpation, the opposite movements – external rotation and abduction – are the earliest and most severely involved, in terms of pain and restriction).



96 Swelling due to right glenohumeral joint effusion.

(96)

Inspection from behind

Now go behind the patient and again inspect for muscle bulk on the two sides, looking particularly at the Supraspinatus and the infraspinatus muscles, but also comparing the trapezius and rumboid muscle bulk. Glenohumeral lesions commonly cause generalized wasting of shoulder girdle muscles. Isolated wasting, e.g. of Supraspinatus muscle, suggests a local Periarthicular lesion. Occasionally, a congenitally underdeveloped, elevated scapula may be noted (sprengl's deformity).

Inspection during movement

All individual glenohumeral movements can be examined in turn, comparing one side with the other; but to quickly screen for joint and rotator cuff problems ask the patient to perform two composite active movements (97, 98):

Place the hands round behind the neck (testing abduction, external rotation, and flexion of the glenohumeral joint and the Supraspinatus, infraspinatus, and teres minor muscles).

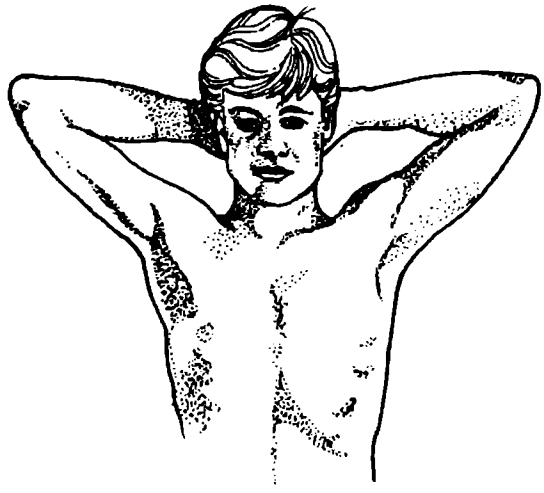
Tack the hands down and round behind the back (testing internal rotation, abduction and extension of the glenohumeral joint and principally subscapularis muscle).

If the patient is able to perform both movements without any problem the glenohumeral and rotator cuff apparatus are probably normal.

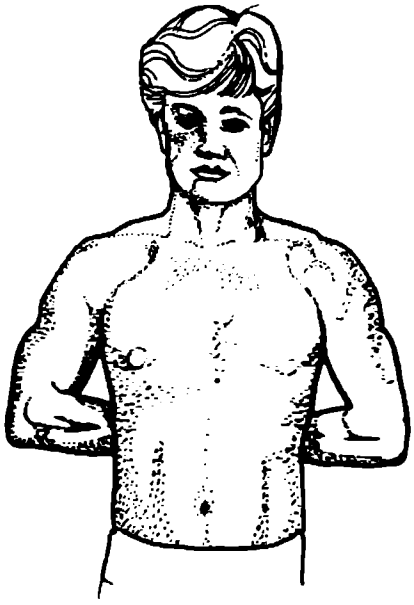
However, if the patient has pain or difficulty with these actions, look for a painful arc

For this, ask the patient to raise the arm slowly through 180 degree towards the [illegible] then slowly to lower it again. This is a composite movement (99). the [illegible]

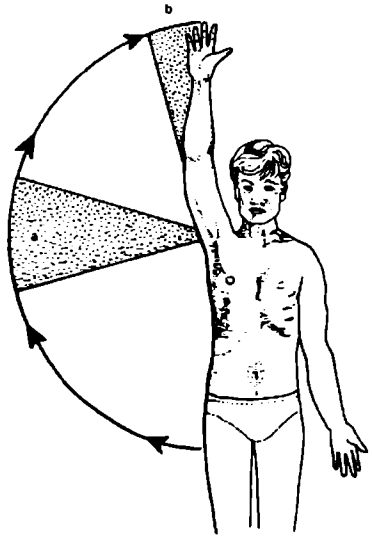
representing glenohumeral abduction, the next 70 degree being principally scapula rotation, and the final 20degree being further glenohumeral movement. During the latter-half of this composite movement the SCJ and ACJ are also moving, and many people also tend to rotate their arm. Two principal patterns of painful arc may be observed: painful middle arc and superior painful arc.



97 Composite movement testing: 'hands behind the head'.



98 Composite movement testing: 'hands behind the back'.



99 Painful arc patterns: (a) middle arc (supraspinatus/ subacromial bursitis); (b) superior arc (ACJ).

(97,98,99)

1. Painful middle arc

The patient experiences pain when the hands get within the central 30-degree or so of the painful arc (i.e. around the end of initial glenohumeral abduction). In this situation the greater tuberosity of the humerus rises relative to the acromion and can squeeze intervening

structures (Supraspinatus tendon and subacromial bursa). As the arm is further elevated the greater tuberosity drops relative to the acromion and relieves the pressure. This arc is characteristic of a Supraspinatus lesion or subacromial bursitis. Supination of the hand may decrease impingement of the humerus on the acromion and reduce or eliminate the pain in the middle arc.

2. Superior painful arc.

Pain occurs at the top 20-30 degree of the arc. This is when there is maximal stress on the ACJ, suggesting a lesion of that joint.

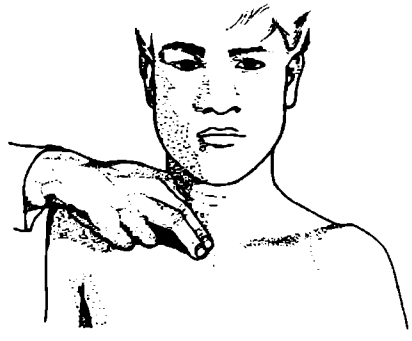
Occasionally, patients with Supraspinatus lesions have no painful arc on moving their arm upwards, but on slowly lowering the arm they experience a painful catching sensation in the middle-arc range, which causes them to quickly drop the arm to their side. The patient should therefor be asked to take the arm slowly up and down.

Palpation

Palpation is best conducted from behind. With the patient seated, the standing examiner is in ideal position. A typical procedure is as follows:

To palpate the SCJ, identify the manubrial notch, then take the fingers laterally to the medial end of the clavicle. Having found the approximate position ask the patient to move the joint by shrugging the shoulders upwards (100). This will allow the examiner to:

- Confirm the position of the joint.
- Feel for crepitus.
- Feel for subluxation (shrugging exaggerates subluxation and the examiner may feel the medial end of the clavicle push his fingers anteriorly, inferiorly, and medially across the front of the manubrium).



100 Palpation of SCJ as patient shrugs shoulders.

(100)

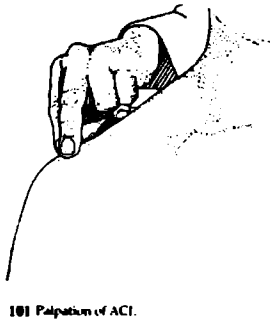
Having found SCJ, press firmly for joint tenderness. Palpate for swelling to determine whether it is soft or bony (bony osteophyte is the most common cause of swelling). If the swelling is soft, look for a balloon sign by placing two fingers of one hand at opposite limits of the swelling and pressing with the other hand over its center. Pass the back of the hand over the joint, and with the same sweep continues over the clavicle and the ACJ to determine increased temperature at each site. Palpate along the clavicle for any obvious localized tenderness and then come to the ACJ.

The site of ACJ may be visible if the distal end of the clavicle is prominent. However, if unsure, feel the outermost bony contour at the shoulder tip (i.e. the coracoid as it slopes posteriorly) and palpate approximately two fingers breadth in from that site (this approximates to the joint line in adults). With the fingers in the approximate joint position ask the patient to move the joint by shrugging the shoulder or abducting the arm. This allows the examiner to:

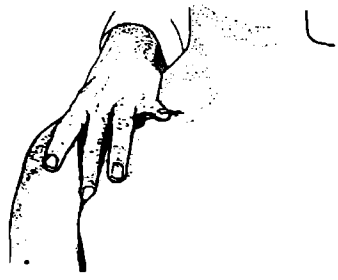
- Localize the correct joint site
- Feel for crepitus.

Having found the joint line, press on it for tenderness (101) and palpate for soft or bony swelling (the latter, resulting from osteophyte, is again most common). If the swelling is soft, look for a balloon sign (rarely present). If ACJ appears to be the main problem, forced adduction of the arm across the front of the patient's chest stresses the joint and may reproduce the pain (this movement is not painful in glenohumeral disease).

The glenohumeral joint is well protected from the examiner's hands by the partially encircling rotator cuff, the acromion superiorly, and the overlying deltoid. The anterior part of the joint is the most accessible for palpation. Start by palpating the anterior triangular region just inferior to the clavicle at the medial border of the deltoid: if there is any soft-tissue fullness (suggesting glenohumeral effusion) press firmly and then release to see if fluid refills the swelling. Move the palpating finger laterally from this position to identify the forward-pointing coracoid process. Taking the finger further laterally (between the coracoid process medially and the humeral head laterally: (102) push firmly upwards and backwards to elicit any anterior joint-line/capsular tenderness (often marked in glenohumeral arthritis and 'capsulitis).



101 Palpation of ACJ.

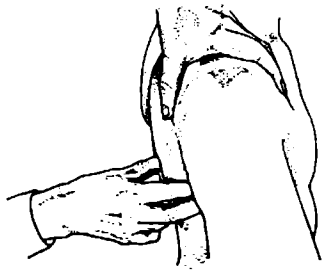


102 Palpation for anterior glenohumeral joint-line tenderness

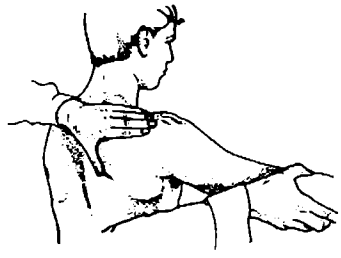
(101,102)

Keep the fingers over the anterior joint line to feel for crepitus while the glenohumeral joint is now moved. Since abduction and external rotation are the movements affected earliest and maximally by the glenohumeral disease they are good screening movements to test. First, locate the blade of the scapula and place the thumb and finger either side of its lower limit so that any scapula movement can be identified. With one hand palpating for crepitus over the anterior joint line and the other holding onto the scapula (103), ask the patient to slowly take the arm out in abduction and assess the range or movement (normally 80-90degree). If there is glenohumeral restriction the examiner will feel the scapular move early before the arm has got to 90 degree: the patient often perform a trick mancuver, with hunching of the shoulder up

towards the ear. If active abduction is painful, determine whether this is in a stress-pain pattern (progressive pain towards the limit of restricted abduction). If pain or restriction of active abduction is present rest one hand on top of the spine of the scapula (to assess scapula movement) and passively abduct the arm with the other hand to determine the extent of passive glenohumeral abduction (104). In glenohumeral/capsular disease, active and passive findings will be similar for both pain and degree of restriction. If, however, passive movement is far greater and less painful than active movement, a muscle/tendon (or nerve) lesion is more likely.



103 Position of hands while testing active abduction at the glenohumeral joint.



104 Passive abduction at the glenohumeral joint.

(103,104)

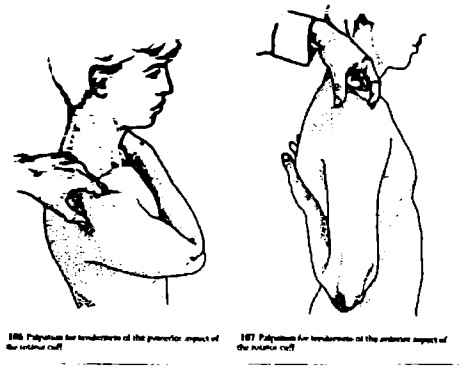
While still behind the patient, palpate the anterior part of the humeral head, while passively internally and externally rotating the arm. The stationary palpating finger should feel greater (lateral) and lesser (medial) tuberosities passing to and fro underneath. Having identified the two tuberosities, palpate firmly ups and down in a line between them over the biceps tendon (105): this may produce the patient's pain if bicipital tendinitis is present.

105 Palpating for tenderness over the biceps tendon.

(105)

Tenderness of the rotator cuff and subacromial bursa can also be sought while the examiner stands behind the patient. The cuff is protected beneath the acromion while the patient arm is by the side. If the patient places the hand onto opposite shoulder, however, this produces posterior rotation of the humeral head and will partly bring the rotator cuff from under

protective cover of the acromion. Palpation just below the posterior aspect of the acromion may then elicit tenderness of the posterior part of the rotator cuff (106). Similarly if the patient places the hand round behind the back this causes anterior movement of the humeral head and brings the anterior part of the cuff from beneath the acromion. Palpation just in front of the acromion will then elicit tenderness of the anterior part of the cuff (107). Palpation below the lateral part of the acromion may occasionally produce tenderness in subacromial/subdeltoid bursitis. Palpation directly over the Supraspinatus and infraspinatus may produce tenderness in lesions of these muscles.

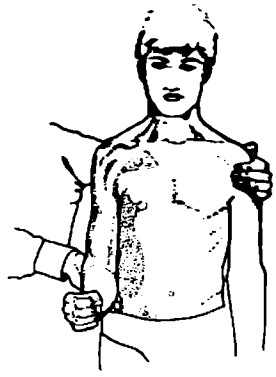


(106,107)

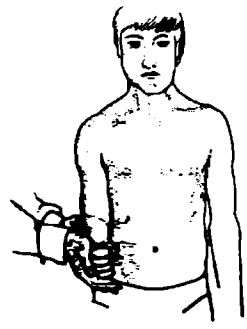
2Resisted active movements

Resisted active movements are used to detect rotator cuff lesion. Sit alongside the patient and place the patient's elbow at their side with the elbow at 90 degree and the hand pointing forwards with clenched fist and thumb upwards. The following movements are then tested:

- ***Resisted active abduction. Abduction is a strong movement and the examiner should*** place his arm around the patient and hold onto the other shoulder while the patient is asked to push their elbow outwards against the examiner's hand (108). Supraspinatus initiates abduction and with lesions of the muscle or tendon this attempted movement will reproduce pain in the upper arm (away from the examiner's restraining hand). If the patient had a painful middle arc and resisted active abduction reproduces pain then a Supraspinatus lesion is the likely cause of pain. If, however, they have a painful middle arc and resisted active movement is pain-free then the likely problem is subacromial bursitis (resisted active abduction does nothing to the subacromial bursa).
- *Resisted active external rotation.* Steady the patient's elbow at their side with one hand (to prevent any abduction) and ask the patient to push the hand outwards against the examiner's other restraining hand (109). Pain experienced in the upper arm suggests infraspinatus/teres minor lesion.



108 Resisted active abduction.



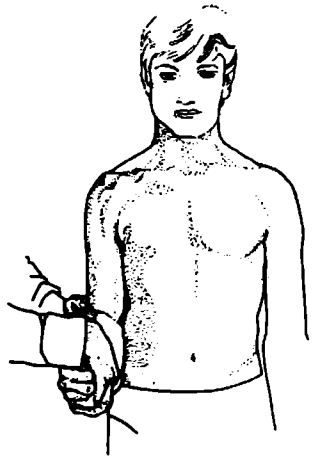
109 Resisted active external rotation.

(108,109)

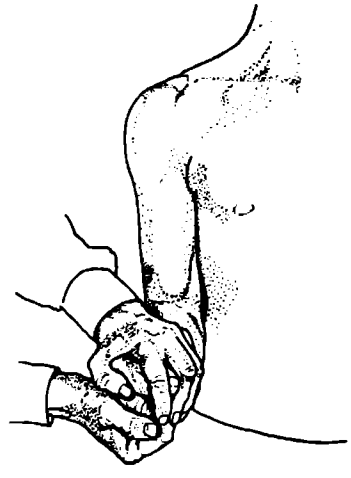
- *Resisted internal rotation*. Again steady the patient's elbow at their side and ask them to push the hand inwards against the examiner's restraining other hand (110). Pain experienced in the upper arm suggests a subscapularis muscle tendon lesion.

Weakness any of these movements may result from pain, partial or complete tears of the cuff, or from neurological abnormality (if power is restored following injection of a local anaesthetic into the subacromial space, pain inhibition rather than rupture is implied).

Bicipital tendinitis is also tested by resisted active movement (111). With the patient's arm in the same position as for rotator cuff testing, hold onto the patient's clenched fist with both hands and ask the patient to turn the wrist outwards in supination. This is a very strong movement and the examiner can observe the biceps tensing up: upper arm pain will be reproduced with bicipital tendinitis, if the patient has ruptured the tendon of the long head of the biceps, the muscle bulge produced by this maneuver will be predominantly in the distal part of the upper arm, producing a larger, more localized, swelling than usual.



110 Resisted active internal rotation.



111 Resisted supination for bicipital tendinitis.

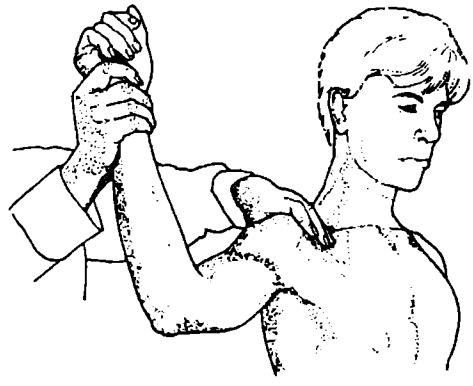
(110,111)

Tests for glenohumeral instability

These are particularly important in the younger patient with a history suggestive of subluxation/dislocation (sometimes as a part of generalized hypermobility). Stability should be tested in anterior, posterior, and inferior directions.

With the patient standing, and the arm hanging relaxed by their side, fix the scapula and shoulder girdle from behind with one hand while gripping the top of the humerus with the other (112): move the humeral head backwards and forwards in the glenoid fossa, noting the degree of movement and any palpable clicks which may signify labral pathology. Next, position the patient with their shoulder abducted to 90 degree and the elbow flexed (113). Gently passively extend and externally rotate the glenohumeral joint: a positive test for anterior instability is when the patient experiences apprehension as an external rotation force is applied.

112



113 'Apprehension' test.

(112,113)

A more controlled method involves the patient lying supine, with the shoulder at the edge of the couch. Support the arm with one hand while fixing the scapula/girdle firmly with the other: move the humeral head anteriorly, posteriorly, and inferiorly using direct pressure. Pain may be reproduced at the limits of movement. If there is significant inferior subluxation a distinct gap may be felt between the humerus and the acromion. As in the apprehension test, position the arm in abduction and external rotation until the patient experiences discomfort: backward pressure on the upper humerus (i.e.anterior glenohumeral support) may now relieve the patient's pain and permit further passive external rotation. if the stabilizing force to the upper arm is then removed, the patient may again experience pain if anterior instability is present. Flexion of the arm in this position, with gentle axial pressure, can be used to demonstrate posterior subluxation.

SUMMARY OF SHOULDER EXAMIATION

(1) **Inspection** at rest

From in front (skin changes, swelling, wasting, attitude)

From behind (wasting, Sprengel's deformity)

(2) Inspection **during** movement

'Hands behind head' 'hands behind back'

Painful arc

(3) Palpation
SCJ (crepitus, **subluxation**, **tenderness**, swelling, warmth)
Glenohumeral joint
Effusion
Anterior joint line/capsular tenderness
Active abduction (restriction, pain, crepitus)
Passive abduction (restriction, **pain**)
Periarthicular tenderness (biceps **tendon**, **rotator cuff**, muscles, bursa)
(4)Resisted active movements (weakness, **pain**)
Abduction (Supraspinatus)
External rotation (infraspinatus, teres minor)
Internal rotation (subscapularis)
Wrist supination (biceps)

Suggested Reading: Clinical examination in Rheumatology. Michael Doherty. John Doherty

SHOULDER PAIN
Pain Location and causes of shoulder pain

SUPERIOR (C4)
- Cervical
- acromioclvicular
- Sternoclavicular
- Diaphragmatic

SUPEROLATERAL
- Rotator cuff tendinitis
- Frozen shoulder
- Synovitis
- Suprascapular nerve entrapment

AXILLARY
- Apical tumor
- Postoperative
- Herpes zoster

Rotator Cuff Tendinitis
Causes of rotator cuff tendinitis
- Impingement
- Calcium crystals-induced inflammation
- Senile tendon degeneration
- Shoulder instability
- Enthesopathy (inflammation at tendon attachment sites)

Impingement

Causes of rotator cuff impingement

Structural	*functional*
J shaped acromion	*Rotator cuff weakness*
Subacromial spurs	*Joint hyperlaxity*
AC joint spurs	
Tumoral calcinosis - at Supraspinatus	

Impingement

Summary of Diagnosis

- History of trauma, sports, and overhead activities.
- Instability maneuvers.
- Impingement maneuvers.
- X-rays.
- MRI if operative treatment is considered.

Key Points

- Is a prominent cause of shoulder pain and tendon rupture.
- Impingement may be structural or functional.
- Diagnosis is clinical.
- Keys in diagnosis are a mid-range painful arc of motion and positive impingement maneuvers.
- Most cases respond to conservative treatment.
- Treatment in functional impingement is with muscle strengthening exercises. Arthroscopic inspection may be important in refractory cases to rule out labral tears and other internal derangement's of the shoulder.
- Surgical unroofing is highly successful in refractory case of structural impingement.

Calcific tendinitis

Summary of Diagnosis

- Radiographic demonstration of amorphous calcium deposit in the vicinity of the rotator cuff tendon.
- Tangential views may be required.
- In-patients with very acute symptoms, especially if a calcium deposit is not shown, the possibility of a missed posterior shoulder luxation should be considered. Identification of this lesion requires an axillary view.

Key Points

- Most frequent in young adults.
- Acute shoulder pain with an inability to move the arm.
- No history of trauma.
- An amorphous calcium deposit is seen radiographically in relation with the rotator cuff.
- Rapid improvement with NSAID, intrabursal Corticosteroid, or IM ACTH.
- Impingement by residual deposits may occur.

Shoulder pain in old age

Summary of Diagnosis

- Usually multifactorial.
- The predominant lesion is rotator cuff tendon fraying and tears plus subacromial impingement from Ac and acromial spurs.
- A painful stiff shoulder may occur.
- Cervical spondylosis frequently coexists.
- Unattended, shoulder pain is a frequent cause of disability.
- Accurate diagnosis is essential for successful treatment.

Frozen shoulder

Summary of Diagnosis

- Fibrotic retraction of the anterior and inferior capsular structures, particularly involving the coracohumeral ligament.
- Diagnosis is clinical.
- Shoulder x-rays are normal or show patchy osteopenia (abnormal findings on x-rays or elevated ESR should put a diagnosis of frozen shoulder in question).
- Diabetes and thyroid should be investigated, particularly in bilateral cases.

Key Points

- Painful restriction of the shoulder pain.
- Unilateral (a bilateral onsets suggests diabetes).
- Shoulder x-rays are normal or show (late) osteopenia.
- ESR is normal.
- Clinical stages: stage 1 with night pain and progressive restriction; stage 11 with restriction and pain on motion; stage 111 of painless resolving restriction.
- Treatment includes Corticosteroid (intraarthicular or orally), analgesics, and Codman's exercises.
- Refractory cases should be evaluated for arthroscopic release.

Suprascapular nerve entrapment

Summary of Diagnosis

- History of direct trauma or heavy sports.
- Pain in the posterior or lateral shoulder.
- Weakness of abduction and external rotation.
- Absence of relief with subacromial xylocaine infiltration
- Positive Electrodiagnosis: delayed conduction, fibrillation potentials.
- MRI should be performed to rule out a compressive lesion.

Key Points

- The entrapment may be at the suprascapular notch or at the spinoglenoid notch.
- Physical findings resemble those of subacromial impingement with rotator cuff tear.
- Diagnosis is by exclusion.

- Delayed conduction and fibrillation potentials in the supra-and infraspinatus muscles are diagnostic.
- Some cases improve spontaneously.
- Surgical release is highly effective in pain relief. Muscle recovery may be delayed and incomplete.

acromioclvicular pain

Summary of Diagnosis

- Suspect by location of pain on top of shoulder.
- Pain in terminal arc of elevation and with full shoulder adduction.
- Pain on direct compression of the joint.
- Acromial view is best to visualize the joint and distal end of clavicle.
- Aspiration may provide an essential sample for diagnosis (pseudogout, septic arthritis).

Key Points

- There are multiple causes of AC joint arthritis.
- AC osteoarthritis can, through inferior spurs, cause subacromial impingement with damage to the rotator cuff.
- Osteolysis of the distal clavicle affects predominantly weight lifters and can cause secondary AC joint osteoarthritis.
- Resection of the distal end of the clavicle is successful in most forms of severely damaged AC joint.

Sternoclavicular pain

Summary of Diagnosis

- Local pain.
- Local tenderness.
- X-rays (limited yield).
- CT scan: very useful in the diagnosis of septic arthritis and its complications, abscesses, and osteomyelitis.
- Aspiration.

Key Points

- Sternoclavicular infiltration is technically difficult.
- Sternoclavicular pain in a debilitated individual may be the sole indication of septic arthritis.
- Septic sternoclavicular arthritis is often complicated by clavicle and sternum osteomyelitis and by abscess formation.
- CT should be obtained early in-patients with suspected sternoclavicular septic arthritis.

Thoracic outlet syndrome

Provocative maneuvers to use in patients with suspected thoracic outlet syndrome

- Downward traction of the arm for one minute.

- Adson's maneuver: ask the patient to turn the hand to one side, hyperextend the neck and inhale deeply while checking pulse on that side
- Wright's maneuver: Ask the patient to elevate and externally rotate the arm and hold a deep breath. If test is positive with ulnar symptoms, repeat with elbow fully extended to rule out ulnar tunnel syndrome.
- Shoulder brace position (military brace position).
- Ask patient to elevate the arms and repeatedly open and close the hands. Maneuver is positive if fatigue and cramps occur within 30 seconds.

Summary of diagnosis

- Atypical shoulder pain
- Neurologic or, less frequently, vascular symptoms in the upper extremity
- A drooped shoulder
- A positive downward traction maneuver
- Relief with upward pulling of the arm
- Examine the supraclavicular fossa for pathology
- Consider reflex sympathetic dystrophy
- A positive Adson's or other maneuver for arterial compression (low specificity)
- Obtain cervical spine films (cervical rib and foraminal pathology) and chest x-rays for apical tumors
- Neurophysiologic tests are almost always normal but are important to detect mimicking or associated conditions (such as a carpal tunnel syndrome)
- A thoracic outlet CT should obtained preoperatively in cases that are refractory to conservative therapy

Thoracic outlet syndrome

Key Points

- The condition should be suspected in-patient with upper extremity neurologic symptoms or with atypical shoulder, upper back, or neck pain.
- Diagnosis is clinical and is based on symptom reproduction by maneuvers that stretch the brachial plexus.
- A drooped shoulder position accounts for most case of thoracic outlet syndrome. A head forward position is likely to add to the symptoms. In the remaining cases neuropathic weakness or an anatomic variant (cervical rib, hooked transverse process, and fibrous bands) is involved.
- Concurrent compressive neuropathy, most prominently carpal tunnel syndrome, must be excluded.
- Initial treatment includes postural advice and exercises. Improvement with an optimal program approaches 90%. A failure to improve is an indication for supporting bracing. Surgery should be used as a last-resort treatment except in-patients with motor loss or ischemia; these patients should be promptly decompressed.

Anterior chest wall pain

Summary of Diagnosis

- Rule out cardiac ischemia by history and ancillary studies if required.
- Rule out fibromyalgia by history and examination of tender points.
- Consider malignancy, which gives focal or diffuse sternal tenderness.
- Consider spondyloarthropathy.
- Consider SAPHO
- Consider pectoralis major insertion strain. Consider xiphodynia in-patients with epigastric pain and tenderness at the xiphoid.
- Consider the slipping-rib syndrome in-patients with upper quadrant pain and localized tenderness at the costal margin.
- ESR elevated in spondyloarthropathy, SAPHO, septic arthritis, osteomyelitis., and malignancy.
- X-ray findings in spondyloarthropathy and SAPHO.
- Bone scan useful to locate osteomalacia-related and other fractures and also to locate neoplastic deposits, osteomyelitis, and osteitis. CT and MRI best to identify SCJ septic arthritis and sternal, clavicular, or costal osteomyelitis/osteitis.

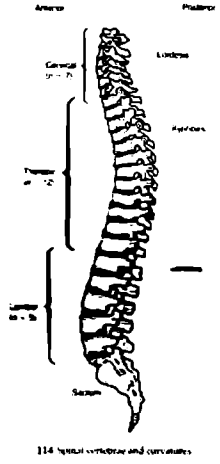
Key Points

- Conditions to keep in mind include cardiac ischemia, excessive respiratory effort, fibromyalgia, spondyloarthropathy, SAPHO syndrome, osteomyelitis, and an underlying chest malignancy.
- Diagnosis requires detailed palpation of the anterior chest wall particularly the SCJs, the manobriosternal joint (a ridge at the level of the second costal cartilage, the costo-chondro-sternal joints, the xiphoid, and the costal margin.
- Most cases are without swelling and are caused by increased accessory respiratory muscle work or fibromyalgia.
- Cases with swelling are either inflammatory (spondyloarthropathy, SAPHO) or due to misshapen costal cartilage.
- An increased ESR speaks an inflammatory lesion such as chondritis or osteitis, microbial SCJ arthritis, or osteomyelitis.
- Presence of sacroiliitis suggests spondyloarthropathy, SAPHO, and rarely brucellosis.

Suggested Reading: Rheumatology in primary care/ Juan J. Canoso

SPINE AND SACROILIAC JOINTS

The unique structure of the vertebral column serves to protect the spinal cord, accompanying vessels, and viscera, and to allow controlled movement of the back, neck and neck. The normal balanced spinal curves (cervical and lumbar lordosis, thoracic and sacral kyphosis - 114) help maintain an upright posture with minimal muscular effort and, together with the resilience of the intervertebral discs, facilitate impact loading through the spine.



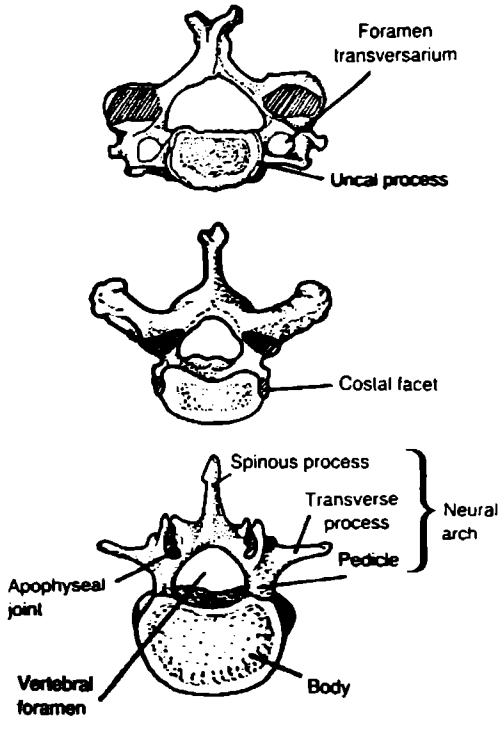
(114)

The vertebrae from C3 to L5 have a common pattern of an anterior vertebral body and a posterior neural arch (115-117). The arch comprises three processes, two lateral (transverse) and one posterior (spinous) that are primarily adapted for muscle attachment, and a synovial facet or apophyseal joints above and below, for articulation with adjacent arches. The bodies and their separating discs are the main weight-bearing portions, body size increasing from C2 to the first sacral segment, then decreasing down to the coccyx as body weight transmits to the pelvis. The sliding apophyseal joints help stabilize the vertebrae, particularly limiting anterior displacement: varying alignment of their arthicular surfaces largely determines the extent and type of movement at different regions. Differences between levels are superimposed on this basic pattern:

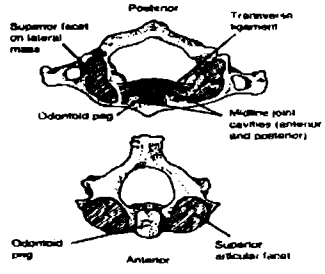
- Cervical vertebrae have foramina in the transverse processes for vertebral arteries, and superolateral ridges (uncal processes) for the joints of Luscka (these increase lateral stability while facilitating free motion between vertebrae).
- Thoracic vertebrae have long transverse processes directed posteriorly; arthicular facets for ribs occur on their tips and on the posterior corners of the bodies.
- Lumbar vertebra, have facet joints orientated in the saggital plane.

The atlas (C1) and the axis (C2) are unique (118, 119), the axis having the odontoid peg, and the atlas being a ring that receives the odontoid peg anteriorly, held in place by the transverse ligament. No disc exists between the atlas and axis (or occiput and atlas), the axis articulating with the atlas through midline and paired lateral synovial joints. The midline joint has two synovial cavities that extend between the peg and anterior arch of the atlas and between the

peg and transverse ligament: synovitis here may damage the peg and ligament, leading to C1/C2 instability and cord damage.



115-117 Vertebral configuration at different levels: (115) cervical; (116) thoracic; (117) lumbar.



118,119 Atlas (118) and axis (119) viewed from above.

(115,116,117,118,119)

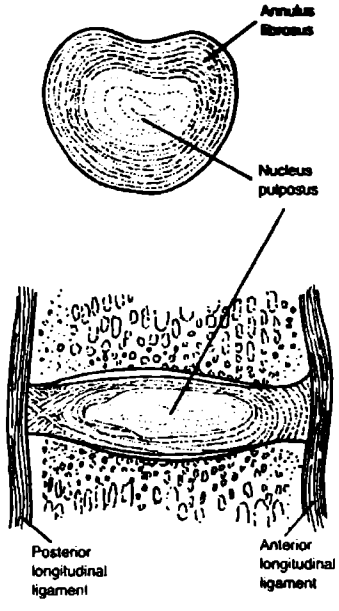
Intervertebral discs (IVDs) are complex symphyses comprising about 25% of the total high of the spine. Each consists of two zones (120, 121):

- The outermost fibrocartilaginous annulus fibrosus, with concentrically arranged fibers (kept under tension by the nucleus), firm attachment to the vertebral bodies, and enervation to the outer lyre. The interwoven concentric fiber arrangement affords great tensile strength while facilitating torsional movements.
- The central mucoid nucleus pulposus, containing high proportion of water, permitting shape (but not volume) change in response to compressive force. Loss of spinal high with age largely results from decrease in water content of the nucleus (from about 90% in youth to 65% in old age: associated decrease in turgor slackens tension in the annulus, predisposing to tears).

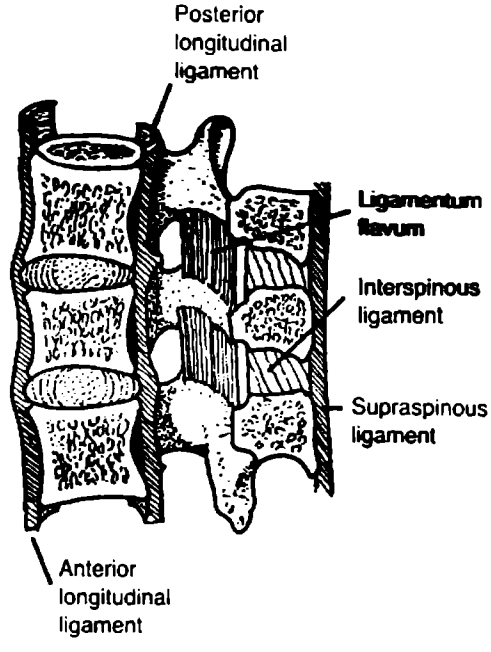
IVD height (and movement between vertebrae) is greatest in the cervical and lumbar regions, and their anteroposterior asymmetry largely determines the normal spinal curves. Pusture has a profound effect on intra-disc pressure, particularly at the lumbosacral junction where

forward flexion associates with the highest increase. The lumbosacral junction is the point of transition between movable and immovable parts of the spine: the spine can act as a lever on the pelvis at this point (this, together with the marked angulation between L4, L5, and S1, makes it a common site for spondylolisthesis). Particularly vulnerable to mechanical stress, the lumbosacral region is also a common site for congenital anomalies of the vertebrae and abnormalities of the IVDs.

Numerous strong ligaments stabilize the spine (122). The posterior and anterior longitudinal ligaments run the length of the spine attaching to discs (particularly the posterior ligament) and vertebral bodies (especially the stronger anterior ligament): they act to restrict flexion and extension and protect the discs. There are also ligaments between adjacent vertebral arches (ligamenta flava), transverse processes (intertransvese ligaments) and spinous processes (interspinous and supraspinous ligaments).



120,121 Intervertebral disc structure: (120) disc viewed from above, showing concentric fibre arrangement; (121) saggital section.

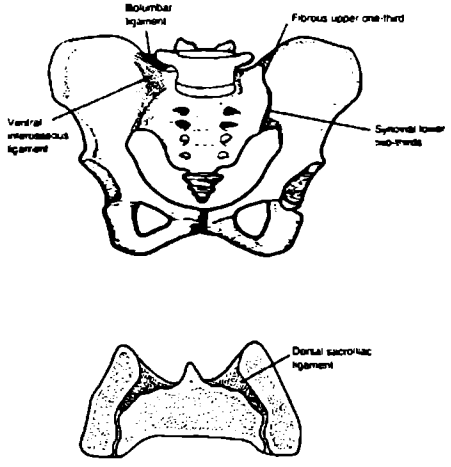


122 Principal ligaments attached to vertebrae.

(120,121,122)

The large superficial muscles of the back (trapezius, latissimus dorsi) largely cover the deeper layers of intrinsic muscles within the lumbodorsal fascia. The numerous deep longitudinal muscles join adjacent segments and span several segments. The longest and strongest extensors are the erector spinae (sacrospinalis), which runs either side of spinous processes, from the sacrum to the skull; they are most developed in the lumbar region. The supra and infra hyoid muscles assist in neck flexion; the pectoralis minor and major assist thoracic flexion; and most lumbar flexion is by the paired rectus abdominae, assisted by the muscles attached to the anterior vertebrae (quadratus lumborum, iliopsoas). Lateral flexion and

rotation is achieved by the oblique abdominal muscles. The neck has a complex system of musculature that enables fine movements to be accurately controlled.
Movement of occipital condyles on the atlas produces discrete nodding (about 30- degree), and the atlanto-axial joint permits discrete rotation (about 30 degree) of the head. Below the craniocervical junction, movements involve distortion of IVDs and sliding movements of the facet joints. Flexion and extension is mainly at the low cervical and low lumbar spine, lateral flexion is greatest in the neck, and rotation greatest in the lower thoracic spine.
The variable sacroiliac joints (SIJs) lie between the wedge-shaped sacrum and the medial aspect of each ilium (123, 124). Fibrocartilage covers the iliac side and thicker hyaline cartilage the sacral side. The lower portion of each SIJ is aligned in an anteroposterior plane, but the upper portion is oblique, with the ilia extending beyond the lateral aspect of the sacrum posteriorly. Viewed from infront, the upper one-third (superior posterior) is a fibrous joint (syndesmosis), while the lower two-thirds (anterior, inferior) is synovial The bones are bound by ventral and dorsal interosseous, sacrotuberous, sacrospinous and iliolumbar ligaments and, except in pregnancy (and childhood), there is no movement.

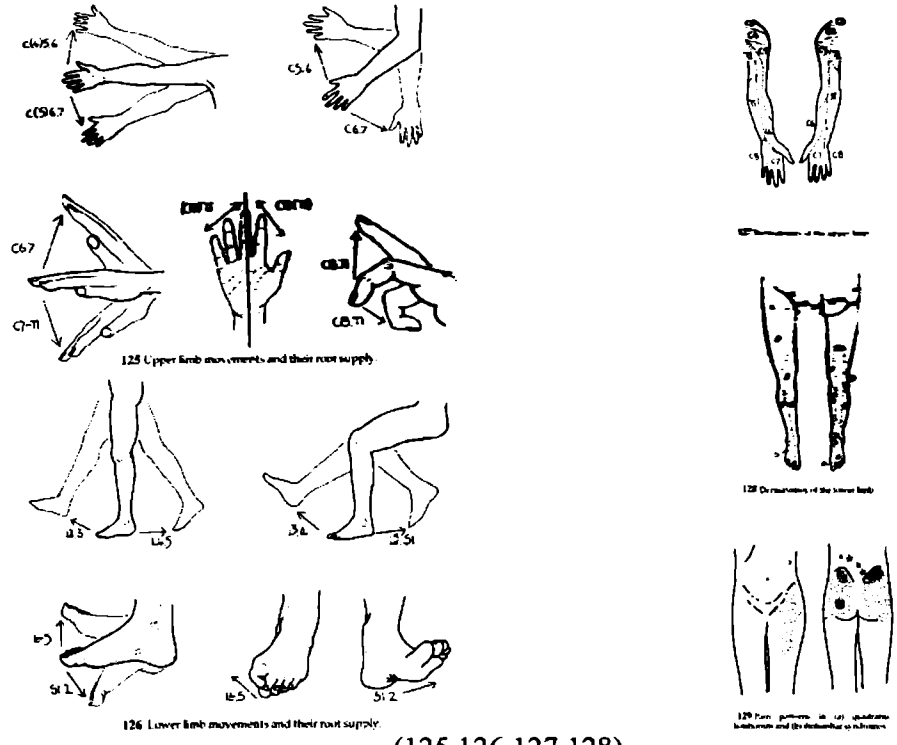


123,124 The sacroiliac joints viewed from in front (123) and in transverse section (124).

(123,124)

Basic neurological aspects require consideration in respect of the spine. The narrow, rigid confines of the spinal canal and emerging foramina may cause problems relating to root or, less commonly, cord compression; it is also vulnerable to damage by atlanto-axial subluxation. The lumbar cord ends opposite the L1/2 disc space, so lower lesions cause root syndromes only. Nerve roots are most vulnerable as they merge from their dural sheaths, just after leaving the exit foramina; they lie in the immediate path of a prolapsing lateral disc. In the lumbar spine such prolapse compresses the lower emerging root. Cervical roots C1-C7 emerge over the top of their respective vertebra, but the C8 root emerge below C7 and above T1 (giving eight cervical but only seven vertebrae). Below T1 all roots emerge below their respective vertebrae. Movements and their root supplies are shown in (125 and 126); dermatomes are shown in (127 and 128)

Upright posture is still relatively recent evolutionary development. As a result of high mechanical stresses through the spine (amplified by poor posture and muscle tone, and obesity), sprain injuries of ligaments, enthesis, and muscles are particularly common. Apophyseal joint osteoarthritis and degenerative disc disease are also common, especially at the mobile, stressed lower cervical and lower lumbar regions: root syndromes, caused by pressure from extruded disc material or bony encroachment (particularly apophyseal joint osteophyte) also predominate here. Less commonly, spinal joints and enthesis are target sites for inflammatory disease (especially seronegative spondyloarthropathy), and bone involvement by malignancy or sepsis may also occur.



(125,126,127,128)

Pain and pain syndromes

Enervation of apophyseal joints, outer annulus, longitudinal and short ligaments, and spinal dura is ultimately shared by the same spinal nerve, with considerable overlap between segments. Pain from spinal locomotor structures is thus poorly defined, with radiation over a wide area, which includes, potentially, the head, thorax, abdomen, and the upper or lower limb). The differential diagnosis of spinal syndromes may therefore be wide and embrace other systems.

'Mechanical' spinal pain

The heterogeneous group is by far the most common problem encountered. The pain is predominantly axial (unilateral, bilateral, or central) but may radiate into proximal or even distal limbs in an ill-defined, non-dermatomal pattern. It may be made worse by movement (usually in one plane) or prolonged standing or lifting, and is usually relieved by rest. O-

examination there may be painful restriction of movement in one direction more than another; a localized site of maximal tenderness (reproducing the pain); 'referred' tenderness and spasm of paraspinal muscles; and absence of neurological signs. In many cases, anatomical localization is difficult and no specific title can be given; in some cases, however, localized tenderness may permit labeling in anatomical term's (129).

Nerve root entrapment

Symptoms may be three-fold:

- Axial or unilateral proximal girdle pain from unilateral compression and tension of the dura mater.
- Root pain along part or all of the dermatomes (variable between individuals) from pressure on dural sheath.
- Weakness, paraesthesia and numbness due to nerve root (parenchymal) compression.

Root pain may occur on its Owen or be superimposed or follow mechanical back pain. It is characteristically sharp and shooting, and made worse by movement and increased intrathecal pressure (coughing, sneezing, and straining at stool). Examination may reveal neurological signs (altered sensation, reduced power, reduced reflex) consistent with single root involvement.

Lumbar canal stenosis

This causes symptoms typical of low 'mechanical' back pain, expect that:

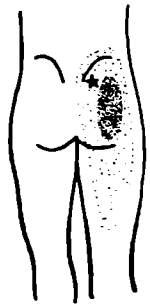
- Paraesthesia (nondermatomal) occurs in one or both legs
- Although symptoms worsen on activity, they may be absent or improve when the lumbar spine flexes forward, increasing the diameter of the canal (walking up steep inclines is better than down, cycling may cause no problems). The patient may adopt 'simian' posture for this reason (hips, knees, lumbar spine slightly flexed).
- Neurological signs (decreased sensation, reflexes) may be present, though these may occur only after exercise.

Inflammatory neck or back pain

This is characterized by diffuse axial pain and stiffness, worsened by rest, but improved by continuing exercise (initial movement may worsen it). Early morning and inactivity stiffness may be marked. Examination shows diffuse, symmetrical tenderness and muscle spasm, and restriction of movement in several or all directions. It may accompany sacroiliac symptoms or signs.

Sacroiliac pain

Characterized by diffuse, ill-defined pain in the ipsilateral buttock, radiating down the back of the leg (130). sacroiliac pain is worsened by stressing the joint, e.g. by running, or by standing on one leg.



130 SIJ pain.

(130)

'Bony pain'

Neck or back pain that is constant, severe, progressive, and present at night is very suggestive of malignancy or infection.

Referred pain

This may be from proximal locomotor structures (especially glenohumeral, hip joints), the major viscera, retropritoneal structures, the urogenital system, or aorta. Associated features in the history and general examination should suggest the correct diagnosis: the pain shows no clear relationship to spinal movement; and examination of the spine is predominantly normal (referred tenderness can occur, but the patient's pain will not be reproduced by pressure on spinal structures).

Examination of the spine

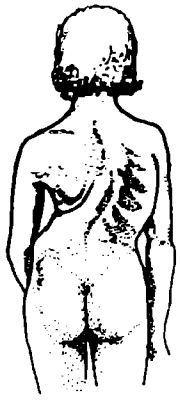
The patient should be wearing only loose underwear. Inspect the standing patient from in front, from the side and from behind; inspect during walking: examine movements; then undertake palpation and appropriate neurological testing with the patient on a couch.

Inspection of the standing patient

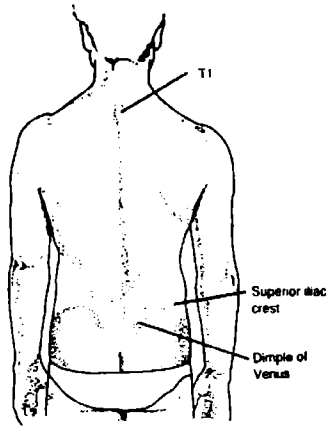
Upper cervical and lumbar spinous processes normally lie deep within the spinal muscles: spinous processes of C7 ('vertebra prominens') and T1 (even more prominent) are most readily identified (131), and the thoracic spines are usually well seen, particularly on bending forward. After T1 the spine of each thoracic vertebra overlies the body of the vertebra below. The 'dimples of Venus' overlie the posterior iliac spines: a line between them overlies the S2 spinous process. The iliac crests may be visible (always palpable) and a line between these overlies the L4/5 interspace. The tip of the coccyx lies in the upper part of the natal cleft. Look particularly for the following symptoms.

- *Loss of normal curves (cervical and lumbar lordosis, thoracic kyphosis)*
- Forward or lateral angulation of the head is common, resulting from diminished lower cervical lordosis and compensatory extension at the craniocervical junction: this may cause prominence of posterior muscles and horizontal skin folds below the occiput. Lateral angulation with rotation may accompany sternomastoid contracture, and several congenital abnormalities result in a short neck. If the thoracic spine shows excess forward angulation, note whether it is a smooth kyphosis (due to multisegment vertebral disc disease) or sharply angulated (localized vertebral damage).
- *Scoliosis*

Its site is represented by the apex of the curve (thoracic, thoracolumbar, or lumbar), its laterality by the side of the convexity (132). Scoliosis may be compensated (T1 centered over sacrum) or uncompensated (a perpendicular from T1 lying outside the sacrum). Postural scoliosis (no intrinsic bony abnormality in spine or ribs) resolves as the patient flexes forwards; by contrast, structural scoliosis persists or is accentuated by flexion. With thoracic scoliosis, rotation of vertebra may produce a hump or 'gibbous' of the ribs on the convex side. Pelvic tilt (iliac crests at different heights +- asymmetry of gluteal folds) may accompany scoliosis or relate to leg shortening, or to hip or other lower limb arthropathy. 'Sciatic'scoliosis resulting from spinal pain is postural and usually mild.



132 Scoliosis (right thoracic).



131 Normal surface landmarks (posterior view).

(132,133)

- ***Reduced chest movement***
- Usually resulting from intrathoracic disease, reduced chest movement may also occur with arthropathy. If expansion appears reduced, measure from full expiration to full inspiration at the nipple line, with the patient's arms on or behind their head (normally 4 cm or more in the adult male).
- *Paraspinal muscle spasm*
- The muscles look as though they are bulging out alongside the spinous processes. Spasm may be unilateral or bilateral and may be associate with spasm of ipsilateral buttock muscles (especially with sciatic pain due to disc prolapse).
- *Skin changes*
- Moles, vascular malformations, and hairy tufts may indicate the site of underlying congenital abnormalities of vertebral bodies. Note any scars or nodules (most common over the bony prominences).

Inspection of the walking patient

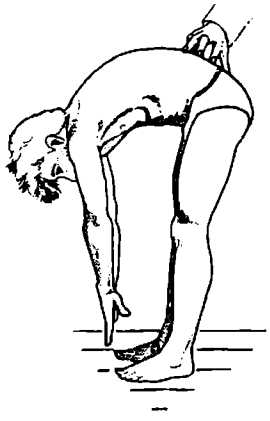
With low-back problems the pelvis may not rotate fully with the advancing leg, remaining mainly aligned with the thorax. This gives a shortened step, jerkiness of movement, and considerable caution and awkwardness during turning. SIJ pain may be worsened by weight bearing and is particularly provoked by standing on one (the ipsilateral) leg.

Inspection during movement

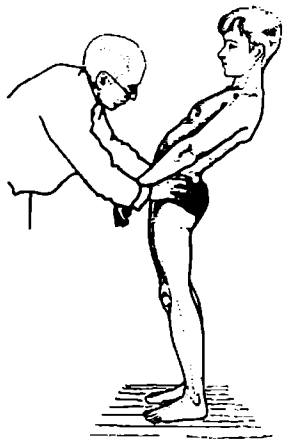
As far as possible it is best to isolate movements at different segments, look for any asymmetry, restriction, or pain on movement.

- *Lumbar movements*

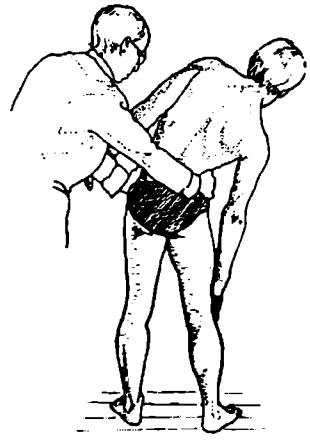
With the patient still standing, the examiner places the fingers of one hand over consecutive lumbar spinous processes and asks the patient to bend forward to touch the toes (this also involves hip flexion). The lumbar lordosis should be replaced by a smooth curve, the degree of movement indicated by separation of the examiner's fingers (133). If present, observe any change in scoliosis. Then, stabilizing the pelvis firmly with both hands, ask the patient to bend backward (extension; 134), and then slide their hand down each leg (lateral flexion-lumbar and thoracic segments; 135).



133 Thoracolumbar flexion.



134 Extension.

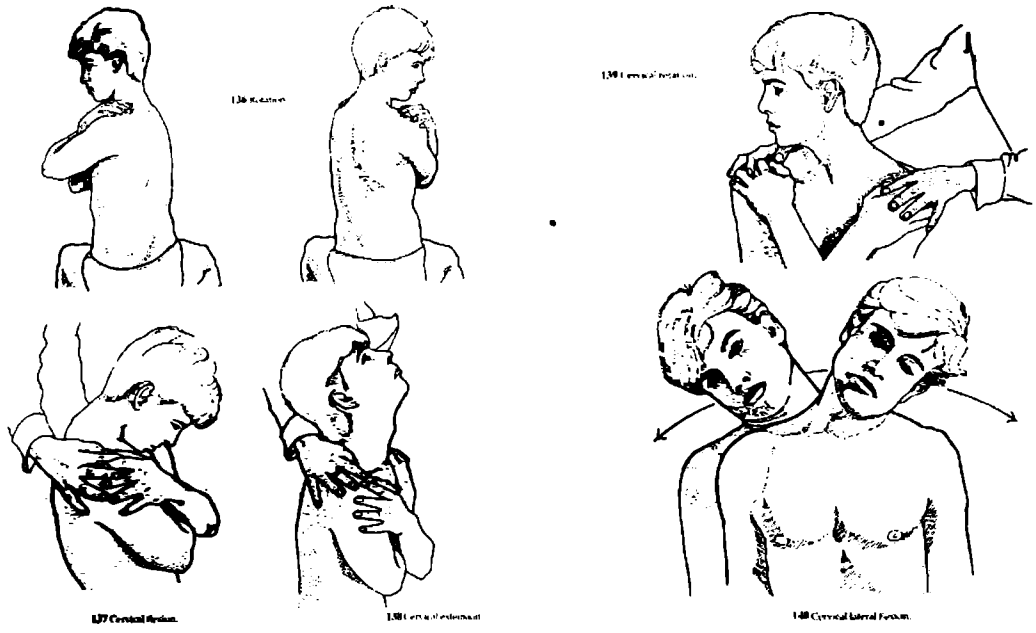


135 Lateral flexion.

(133,134,135)

- *Thoracholumbar rotation and cervical movements*

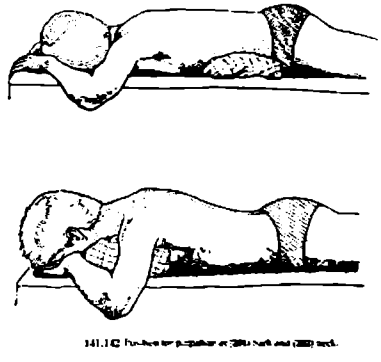
For these, fix shoulder girdles to the trunk by getting the patient to clasp their arms across their chest, and fix the pelvis by firmly holding each iliac crest (with the patient's feet apart) or, preferably, by seating the patient astride a chair. Ask the patient to turn round as far as possible to each side (rotation-mainly thoracic, 136). Then, holding the patient's fixed shoulders, ask the patient to put the chin on their chest (flexion, 137), look up in the air (extension, 138), look round to each side (rotation, 139), and then put each ear over onto the shoulder (lateral flexion, 140). During lateral flexion, pain felt on the side to which the head moves suggests facet joint disease, whereas pain on the opposite side is more likely muscle spasm.



(136,137,138,139,140)

Palpation

Lay the patient face down on the couch, relaxed with arms folded underneath. For palpation of the neck, place a pillow under the upper chest; for the thoracic and lumbar spine moves the pillow under the abdomen, this helps to relax the muscles, supports the spine in flexion, and aids separation of the spinous processes (141, 142). Palpate in turn the following areas.

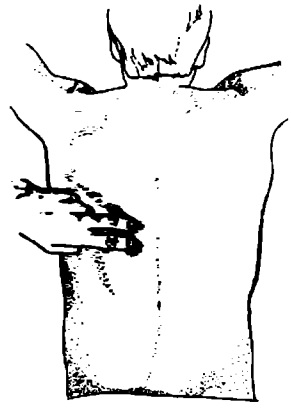


(141,142)

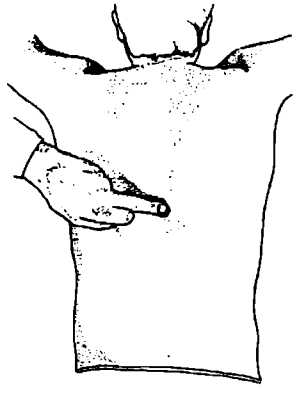
- ***Skin*** *and subcutaneous tissues:* Palpate in a line down each side of the trunk. Use a 'skin rolling' technique (143), to look for areas of hypersthesia. This is a useful but poor localizing sign suggesting possible pathology in the nearby region of the spine (analogous to generalized abdominal tenderness with appendicitis).
- *The* ***paraspinal*** *muscles.* Feel for increased tone and tenderness on one or both sides, (144). This again is poor localizing sign.

- *The interspinous ligaments:* Apply firm pressure over each in turn (145). Tenderness with reproduction of the patient's pain suggests local ligamentous or disc disease. Other abnormalities to note during palpation include defects in the spinous processes (spina bifida occulta), or a 'step'deformity of spondylolisthesis (usually L4/5) or retrolisthesis (more common in the cervical spine).
- *The facet joints.* Applying firm, jarring pressure with each thumb just lateral to the spinous process (146) may cause pain relating to local facet joint, disc, or ligament lesions.
- *Mid-trapezius:* This is palpated for hyperalgesia of fibromyalgia syndrome.
- *The medial iliac crest:* this is a common site of tenderness, with reproduction of the patient's pain ('iliolumbar' or 'iliac crest syndrome').

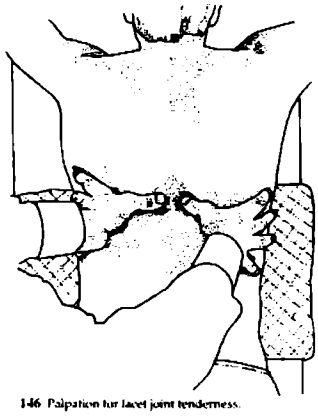
143 'Skin rolling' for hyperaesthesia.



144 Palpation of paraspinal muscles.



145 Palpation of interspinous processes.



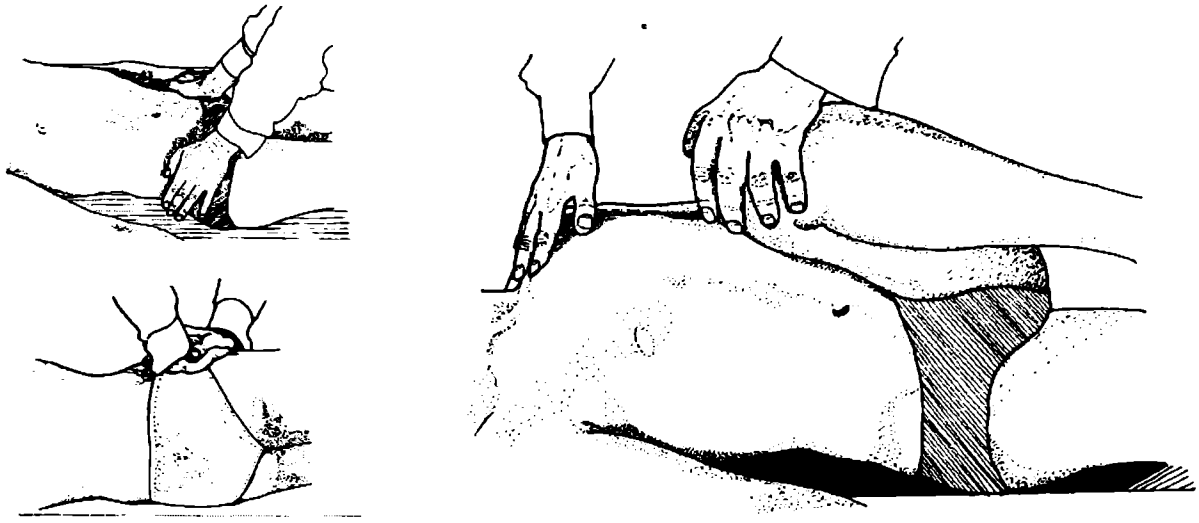
146 Palpation for facet joint tenderness.

(143,144,145,146)

Examination of the Sij

The SIj, which is inaccessible to palpation, is difficult to assess clinically. Only florid inflammation or damage to the fibrous portion is likely to result in local tenderness posteriorly (most such tenderness is probably ligamentous). Tests designed to stress the SIjs and produce pain in the buttock are non-specific and include:

- *Distraction tests.* Firm downward pressure over both sides of the pelvis with the patient lying supine (147) or over the pelvis with the patient lying on one side (148).
- *'Knee-to-shoulder test* (149). With the patient lying flat, flex and adduct one hip and push the flexed knee towards the opposite shoulder, stressing the ipsilateral SIj. This test is helpful only if the hip and lumbar spine are normal.



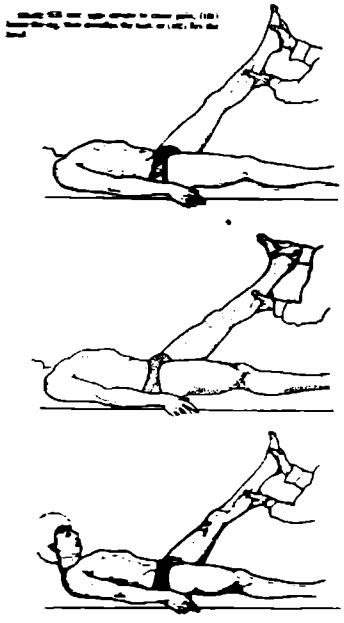
149 Knee-to-shoulder test.

(147,148,149)

Neurological aspects

Provocation tests for root lesions: Numerous named tests exist, all using maneuvers to distracts roots or increase intrathecal pressure and thus reproduce the patient's symptoms.

- *Straight leg raising (SLR, Lasegue's test).* The most common test used. With the patient lying flat on the back and completely relaxed, slowly raise the straightened leg on the affected side by 70 degree, maintaining full extension at the knee, until the patient complains of pain or tightness down the leg (150-152). Note the angle of elevation, then drop the leg back slightly to eliminate the pain. Now ask the patient to flex the neck by putting the chin on the chest, or passively dorsiflex the raised foot. Reproduction of pain by either action indicates stretching of the dura (a central prolapse often causing back>leg pain, a lateral prolapse the reverse); SLR pain not reproduced by these actions suggests hamstring pain (mainly posterior thigh) or lumbar or sacroiliac pain (felt more in the back than the leg).

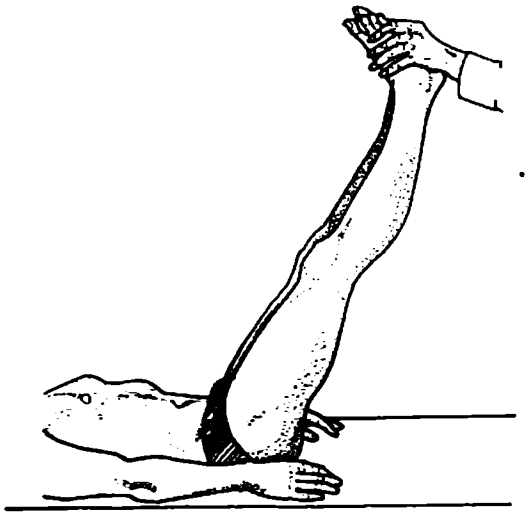


(150,151,152)

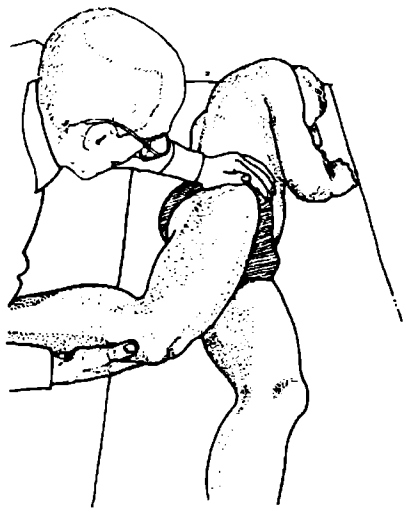
During elevation of the leg, from 0 to 40-degree, there is no traction on the roots but 'slack' in the sciatic arborization is taken up; between 40 and 70-degree there is tension applied to the roots (mainly L5, S1, and S2); above 70 degree, no further root deformation occurs, and any pain after this elevation is probably arthicular. Compare both legs for any difference. Reproduction of pain in the affected side by elevation of the opposite leg (*cross- over sign* or 'well leg raise test') indicates thecal compression by an often-large lesion medial to the nerve root (disc or tumor). If both legs are raised together (bilateral SLR), little distortion of nerve roots occurs; pain appearing before 70 degree probably arises from SIjs, pain beyond 70 degree from the lumbar spine (153).

- *Femoral nerve stretch test*

This produces traction on the L2, L3, and L4 nerve roots. Lay the patient on the unaffected side with the affected hip and knee slightly flexed the back straight and the head flexed. Gently extend the hip and increase knee flexion; pain down the anterior thigh indicates a positive test (154). As with SLR a contralateral positive test may also occur.



153 Bilateral SLR.



154 Femoral nerve stretch test.

(153,154)

Neurological examination for root abnormality:

The principle abnormalities of sensation, power, and reflexes accompanying individual root lesions are summarized in table 9 and 10.

Examination for cord signs:

A spastic gait, lower limb ataxia, increased reflexes, and extensor plantar responses (i.e. upper motor neuron signs) indicate pressure or damage to the cord, the level being determined principally by the division between normal and abnormal reflexes and the level of any accompanying lower neuron signs.

Table. Principal cervical root syndromes (affected dermatomes, myotoms, and reflexes)

Root	Sensation	Weakness	Reflex
C5	Lateral upper arm	Shoulder abduction	Biceps
C6	Lateral forearm	Elbow flexion wrist extension	Brachioradialis
C7	Middle finger	Elbow extension, wrist flexion	Triceps
C8	Medial forearm	Thumb extension, unlar deviation of wrist	-
T1	Medial elbow region	Hand intrinsics, finger abdduction	-

Table. Principal lumbar root syndromes (affected dermatomes, myotomes, and reflexes)

Root	*Sensation*	*Weakness*	*Reflex*
L4	Anterior leg, medial foot	Ankle dorsiflexion (tibialis anterior)	Knee jerk
L5	Lateral leg/thigh, web of hallux	Extension of great toe (ext. hallucis longus)	-
S1	Posterior leg, lateral foot	Eversion of foot (peroneals)	Ankle jerk

Assessment of power, plantar responses, and sensation may be difficult in patients with polyarthritis and joint deformity, muscle wasting, and entrapment or peripheral neuropathy. Upper cervical cord damage due to C1/2 instability in rheumatoid arthritis is a particular problem: helpful signs in this situation may include positive pectoralis jerks (155; suggesting a lesion above C4), a normal jaw jerk (implying a lesion below the brainstem), and diminished/absent corneal reflex (the sensory part of the fifth cranial nerve center extends into the upper cervical cord).

(155)

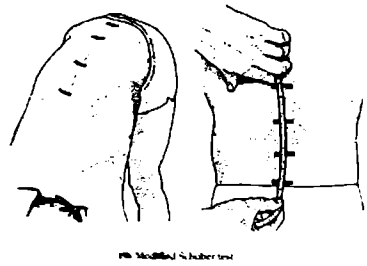
Examination of other systems

Pain in the spine may be referred, and a thorough assessment of other systems (particularly the lower bowel and genital tract) may be required in individual cases.

Additional tests/procedures

Measurements of spinal movements

In addition to simply assessing distraction of fingers placed on spinous processes, thoracholumbar flexion can be estimated by the modified Schober test (156). Get the patient as flexed as possible (standing or sitting) and, starting from an upper sacral spinous prominence, mark out three 10cm segments up the spine. Then remeasure the distances between the marks with the patient erect: the lowest segment should shorten by at least 50%, the middle by 40%, the upper by 30%

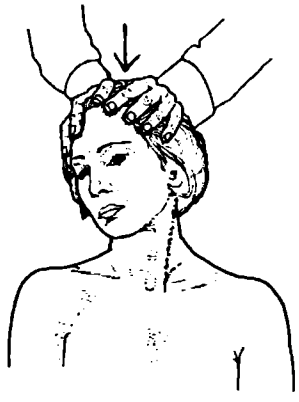


(156)

(Shortening is greater in tall subjects). An alternative is to measure the C7-T12 and T12- S1 distances erect and then during maximal flexion; the thoracic measurement should increase 2-3cm, the lumbar distances 7-8cm. Other measurements employed include the finger to floor distance when the standing patient attempts to touch the floor with legs straight; and the occiput to wall distance, measured eight patient standing upright, heels back against a wall, eyes level.

Foraminal compression/distraction tests

These may be used for cervical entrapment syndromes, but are rarely positive. Passively rotate and laterally flex the neck towards affected side, then carefully press down on the head; reproduction of pain down the arm or around the scapula region suggests root entrapment or facet joint disease (foraminal compression test; 157) Conversely, upward traction on the neck, by lifting with one hand under the chin and the other under the occiput, may relieve pain due to
root compression (distraction test; 158).



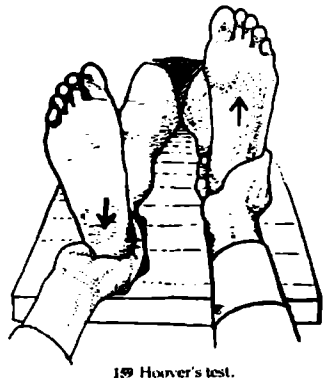
157 Foraminal compression test.

158 Cervical distraction.

(157,158)

Milgram's and Hoover's tests

These are used to distinguish 'organic' from 'functional' pain. In Milgram's test the supine patient performs active bilateral SLR to a height of 6 inches. This greatly increases thecal pressure and ability to hold this position for any time excludes significant thecal pathology. In Hoover's test the patient performs unilateral SLR with the examiner's hand under the other heel; absence of downward heel pressure indicates that the patient is not really trying (159).



159 Hoover's test.

(159)

Summary of examination of spine

(1) Inspection of the standing patient
- (a) From in front (head angulation, chest expansion)
- (b) From the side (spinal curves)
- (c) From behind (scoliosis, pelvic tilt, muscles, skin)

(2) Inspection of the walking patient

(3) Inspection during movement (restriction, pain)
- (a) Standing
  - Flexion-'touch toes' (+-Schober test)
  - Extension
  - Lateral flexion
- (b) Preferably sitting astride chair
  - Thoracholumbar rotation
  - Cervical flexion, extension, lateral flexion, rotation

(4) Palpation of patient lying on couch
- (a) 'Skin rolling' each side (hyperaesthsia)
- (b) Paraspinal muscle (tone, tenderness)
- (c) Interspinous ligaments (pain)
- (d) Facet joint region (pain)
- (e) Medial iliac crest (tenderness)

(5) Stressing of sacroiliac joints
- (a) Distraction
- (b) Knee- to- shoulder test

(6) Provocation tests for root entrapment
- (a) Straight leg rising each side
- (b) Bilateral SLR
- (c) Femoral nerve stretch test

(7) Neurological examination (power, reflexes, sensation)

(8) Detailed examination of other systems as required

Suggested Reading

Clinical Examination in Rheumatology. Michael Doherty. John doherty

NECK, DORSAL, AND LOW BACK PAIN

Acute nonspecific neck pain

Summary of Diagnosis

- Recurrent materialized neck pain based on soft tissue factors in patients with normal cervical spine.
- Causative factors include protracted and repetitive physical and psychologic stress.
- If a trigger point is found, myofascial pain may be diagnosed.
- Comorbid structural conditions of the cervical spine are frequent, particularly in the older age group.

Acute nonspecific neck pain

Key Points

- Predominates in young women under stress.
- Muscle contracture appears to be involved in most cases.
- In the repetitive stress syndrome there is vague distal pain and subsequent involvement of the periscapular region and lateral neck.
- A trigger point may be found. It is imperative in these patients to rule out fibromyalgia. If this is not present, a myofascial syndrome may be diagnosed.
- Treatment includes analgesics, heat application, and stretching of the involved muscle(s).
- Recurrences are common, usually triggered by stress.

Whiplash injury

Summary of Diagnosis

- Detailed description of circumstances of the accident and symptoms.
- Neck motion and tenderness.
- Motor, sensory, and reflex examination.
- Patients with grade I whiplash (alert, not obtunded by alcohol or drugs) do not require radiographic evaluation. Patients with grade II and III whiplash need radiographic evaluation including AP, lateral, and open mouth films.
- Additional studies are indicated when the initial radiographic evaluation is equivocal.

Whiplash injury

Key Points

- Frequent complication of motor vehicle accidents, in particular rear-end collision.
- Mechanism of injury is complex and not well understood; resulting damage includes muscles and ligamentous tears, spinal instability and sympathetic chain.
- The source of pain is a damaged facet (zygapophyseal) joint in at least 50% of cases.
- Symptoms persist more than 6 weeks in 50% of cases and more than 6 months in 30%.

- Cases with protracted, severe localized neck pain or radicular symptoms should be studied with flexion/extension radiographs and MRI seeking a surgical correctable lesion.

Cervical spondylosis

Summary of Diagnosis

- Diagnosis is based on clinical and radiographic data.
- Middle-aged to older individuals.
- Neck pain and limitation without evidence of inflammatory disease.
- Cervical radiculopathy and myelopathy may be present.
- Cervical spine films (AP, lateral, and oblique views) reveal disc narrowing, osteophytes, and possibly encroachment on neural foramina.
- Nerve conduction studies and EMG may identify additional impinging lesions such as a co incidental CTS.
- CT and MRI provide definitive data about the condition.

Cervical spondylosis

Key Points

- Degenerative process of the cervical spin secondary to intervertebral disc failure.
- Present in 40% above the age of 50; virtually universal above 70.
- Correlation between clinical findings and imaging abnormalities is poor.
- Main complications include radiculopathy (C6, 7) and myelopathy.
- Treatment should be temporizing and conservative unless progressive neurologic damage is documented.

Dorsal Pain

Summary of Diagnosis

- Diagnosis is clinical in most cases.
- Muscular pain may feature a bundle of contractured muscle which when compressed, results in pain radiation to the entire symptomatic area (trigger point).
- In fibromyalgia there are symmetric tender areas in the dorsal region and elsewhere.
- In the spondyloarthropathies, sharply localized interspinous tenderness may be elicited
- In bone pain (fracture, malignancy, osteomyelitis) pain is present day and night, there is nocturnal predominance of pain, and marked spinous processes tenderness is present at the involved segment(s).
- If radicular pain is present, consider herpes zoster in addition to bone processes and foraminal impingement. The typical herpetic skin lesion may appear days, sometimes weeks, after radicular pain sets in.
- Always ask yourself if pain could represent radiation from a visceral organ.
- X-rays are not indicated in muscular types of pain or interspinous pain [illegible] spondyloarthropathy.
- They are definitely indicated when a bone source is considered.

- If malignancy is considered, a technetium bone scan is useful to identify silent remote bone lesions.
- MRI is superior to CT in defining bone lesions.

Dorsal Pain

Key Points

- Most cases of dorsal pain are due to muscle contracture, fibromyalgia, or bone lesions.
- The most common bone lesion is an osteoporotic fracture.
- Radicular pain radiation and bone type pain are red flags of potentially catastrophic conditions.
- Neurosurgical or orthopedic referral should be made promptly.

Low back pain

Inflammatory versus mechanical low back pain

	Inflammatory	mechanical
Onset	Gradual	Abrupt
Duration	> 3months	brief
Morning stiffness	Lasts hours	Lasts minutes
Effect of activity	Diminishes pain	Increases pain
Pain at night	Present	Absent

Low back pain

Summary of diagnosis

- A careful history and physical examination, based initially on the exclusion of potentially catastrophic conditions, allows an empirical diagnosis of low back strain in most cases.
- Conditions to be excluded include an expanding abdominal aneurysm; the cauda equina syndrome; referred pain from an abdominal, retropritoneal, or pelvic source; osteomyelitis; malignant disease; and spondyloarthropathy.
- Based on clinical indications, one or more of an array of tests and imaging procedures are necessary in a minority of patients with persisting or worsening pain.
- Persistence of symptoms beyond 4 weeks justifies a thorough reevaluation.

Low back pain

Key Points

- Initial evaluation is aimed at excluding potentially catastrophic conditions, referred visceral pain, osteomyelitis, malignant disease, and spondyloarthropathy.
- Most cases run a benign, self-limited course within 2 weeks.
- Patients who continue symptomatic 4 weeks after initial evaluation should be further investigated to rule out inflammatory disease, structural disease of the sine and neoplasia.

Referred Low back pain

Summary of Diagnosis

- Mounting and tearing colicky-type pain.
- Concurrent or pelvic pain.
- Bowel, urinary, or gynecologic abnormalities.
- Ancillary studies tailored to symptoms: urinalyses, occult blood in stools, serum amylase and lipase; ultrasonography, abdominal/retropritoneal CT scan, colonoscopy, and retrograde urether pyelography.

Referred Low back pain

Key Points

- Cases should be rapidly identified; even in straightforward instances, exclusion by history and physical examination of a complicated abdominal aneurysm and the cauda equina syndrome is necessary.
- Joint care with a vascular surgeon, gastroentrologist, abdominal surgeon, urologist or gynecologist is usually required.

ACUTE NONSPECIFIC LOW BACK PAIN (BACK SPRAIN, LUMBAGO)

Red flags to be considered in initial evaluation of patients with acute low back pain

Red flags for malignancy

- Age over 50
- A history of malignancy
- Unexplained weight loss
- Pain increased by rest

Red flags for infection

- Skin or urinary tract infection
- Intravenous drug use
- Immunosuppression
- Pain increased by rest

Red flags for cauda equina compression

- Bladder dysfunction (urinary retention or flow incontinence)
- Saddle anesthesia (anus, perineum, genitals)
- Loss of anal sphincter tone
- Major limb motor weakness

Red flags for spinal fracture

- Preceding trauma relative to age (older osteoporotic patients may fracture the spine upon a minor fall or a lift effort; in younger patients spinal fracture requires major trauma such as a fall from a height or a motor vehicle accident).

Back strain

Summary of Diagnosis

- Acute onset following unusual or unprepared-for activity.
- Absence of red flags of a serious underlying condition.
- Short duration of symptoms.
- Increasing pain with certain activities and sustained postures.
- Muscle contracture.
- Occasional presence of a trigger point.
- Hyperlordosis and unequal leg length should be sought.
- X-rays are not indicated in these patients.

Back strain

Key Points

- Self-limited, mechanically determined low back pain.
- The structure at fault cannot be determined from the symptoms.
- Course is spontaneously regressive with duration of symptoms less than 3 weeks.
- Laboratory studies and x-rays are not indicated.
- Treatment is with analgesics, muscle relaxants if required clinically, and local heat. Bed rest should discouraged.
- The condition tends to recur; all patients should be instructed on back protection.
- Persistence of pain beyond 4 weeks dictates the need for further investigation.

Sciatica

Summary of Diagnosis

- Acute pain and Paresthesias extending along L5, S1, or rarely L4.
- Preceding recurrent lumbar pain.
- Reflex changes in L4 (knee jerk) and S1 (ankle jerk) lesions. There are no reflex changes in L5 sciatica.
- Bilateral sciatica suggests central hernia or other extensive compressive lesion.
- A negative straight-leg raising test suggests extraspinal compression or neuropathy.
- Watch for the emergence of cutaneous herpes zoster.
- Plain radiographs are indicated in-patients with neurologic findings to assess spondylolisthesis, fracture, neoplasia, or infection.

Sciatica

Key Points

- May have multiple spinal and extraspinal causes.
- Bilateral sciatica and rapidly progressive neurologic deficit call for immediate orthopedic or Neurosurgical consultation.
- Most cases of unilateral, single-root sciatica in young individuals are caused by an extruded disc and in older individuals by degenerative changes leading to lumbar stenosis.
- In many instances extruded disc are eventually absorbed in the epidural space.
- Treatment is as in back sprain but improvement is slower.

Spinal Stenosis

Causes of spinal stenosis

CONGENITAL-DEVELOPMENTAL STENOSIS

- Idiopathic
- Achondroplasia
- Hypophosphatemic vitamin D-resistant rickets
- Morquio's syndrome
- Conjoint origin of lumbosacral nerve roots
- Other congenital disorders

ACQUIRED STENOSIS

- Degenerative (discogenic, spondylolisthesis)
- Postoperative (laminectomy, fusion, chemonoclolysis, etc.)
- Posttraumatic
- Endocrine-metabolic (Cushing, acromegaly, renal osteodysthrophy, etc.)
- Miscellaneous (Paget's disease of bone, ankilosing spondylitis, diffuse idiopathic skeletal hyperostsis, fluorosis, etc.)

COMBINED

- Developmental plus acquired
- Cervical plus lumbar (tandem)

Spinal stenosis

Summary of diagnosis

- Severe lower extremity pain.
- Neurogenic claudication
- Absence of pain when seated.
- Thigh pain induced within 30 seconds by lumbar extension.
- Presence of pedal pulses; if absent, check for trophic changes and assess flow by Doppler.
- Wide-based gait.
- Romberg signs.
- Lumbar spine films (spondylosis, primary reduction of lumbar canal diameters; normal in certain cases).
- MRI or CT of the lumbar spine shows the process well. Myelography may also be used.

Spinal stenosis

Key Points

- Spinal stenosis is usually degenerative and therefore affects elderly individuals
- Hence there is a high frequency of Comorbid conditions.
- Most cases are slowly progressive.

- Surgical decompression succeeds in about 75% of patients with degenerative stenosis.
- Barring severe comorbidity, patients with spinal stenosis should referred for surgical evaluation.
- Nonsurgical patients should be offered epidural Corticosteroid infiltration.

Chronic low back pain

Summary of Diagnosis

- Pain of more than 6 months' duration.
- Obtain thorough history and perform detailed examination including assessment of depression and fibromyalgia.
- Review occupational factors.
- Review available imaging studies.
- A gadolinium-enhanced MRI provides essential information, particularly in postoperative cases.

Chronic low back pain

Key Points

- Reassess diagnosis
- Identify treatable components, in particular depression, fibromyalgia, and gross leg length discrepancy.
- If available, offer attendance at a back school.
- Resilient cases, provided that the patient has a genuine desire to improve, should be referred to a pain service.

Suggested Reading

Rheumatology in primary care. Juan J. Canoso

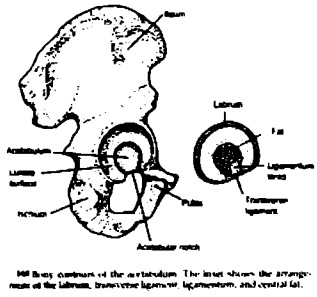
HIP PAIN

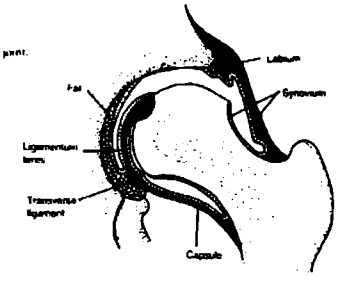
The hip is a large ball-and-socket joint that plays a major role in weight bearing, stance and locomotion (walking, running, jumping, swimming, etc.). It thus needs to permit a wide range movement while maintaining great stability. Mobility is aided by the elongated femoral neck, which offsets the shaft from the head, an arrangement that also gives great leverage to muscles acting at the proximal femur. Stability is due to:

- The powerful muscles acting across the hip
- The strong fibrous capsule
- The deep insertion of the femoral head into the acetabulum

Forces across each hip are often great: for example, standing on both feet (one-third body weight), standing on one leg (2.5 x body weight), or walking (1.5 -6 x body weight). Under low loads the joint surfaces are incongruous, but under heavy loads they become congruous, providing maximum surface contact to keep the load/unit area within tolerable limits.

The acetabular cavity is formed at the meeting point of the three bones comprising the inanimate (the ileum, the ischium, and the pubis: 160, 161). It opens outwards, forwards, and downwards, and is strongest superiorly and posteriorly (where it is subject to greatest strain in the erect and stooped position). the rim is deepened by the fibrocartilaginous labrum, which forms a collar around the femoral head, narrowing the outlet and stabilizing the femoral head within the acetabulum. A gap in the lower portion of the labrum, the acetabular notch, is bridged by the transverse ligament, converting the notch into a foramen through which blood weasels pass into the joint. The arthicular cartilage is horseshoe shaped with the open part pointing inferiorly; a fat mass fills the fosca at the bottom of the acetabulum. Hyaline cartilage covers the whole of the femoral head except at the attachment of ligamentum teres, where there is a small bony defect, the fovea.

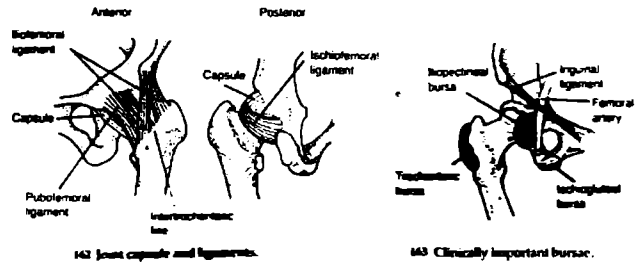




(160,161)

The strong, dense fibrous capsule arises cercumfrentially from the acetabulum, labrum and transverse ligament. It attaches distally to the intertrochanteric line of the femur anteriorly, and about halfway along the neck posteriorly. It is reinforced in front by the Y-shaped ileofemural ligament (the strongest in the body), inferiorly by the pubofemural ligament, and posteriorly by the ischiofemural ligament (162). The ligamentum teres is an intracapsular ligament that runs from the transverse ligament to the fovea; it has no joint stabilizing function but carries blood vessels, which supply a small area of the head around the fovea. The synoviom lines the capsule, labrum and fat pad, and excludes the ligamentum teres: distally, it reflects onto the femoral neck and extends to the cartilage of the head. The iliotibial

band is part of the facia lata extending from its main attachment at the iliac crest to the lateral Tibial tubercle. Clinically relevant bursae (163) around the hip include:



(162,163)

- The large, multilocular, trochanteric bursa between the greater trochanter and gluteus maximus
- The iliopectineal bursa between the anterior capsule and iliopsoas (communicating with the joint in about 15%)

The ischiogluteal bursa over the ischial tuberosity, overlying sciatic nerve

The strong muscles around the hip have complex actions, and hip movements are influenced by the position of the lumbar spine, the knee, and the opposite hip.

(E.g. flexion increases if the knee and spine also flex; extension increases if the knee is extended; abduction increases if both hips are slightly flexed). The prim movers are;

Flexors: ileopsoas (L2, L3 nerve root supply) (pectineus, rectus femoris).

Extensors: gluteus maximus, hamstrigs (L4, 5; S1, 2)

Abductors: gluteus medius, (gluteus minimus) (L4, 5; S1)
Adductors: adductor longus, magnus, brevis (L3, 4, 5; S1)

Rotation

External: piriformis, obturator, gemelli, gluteus medius (L4, 5; S1)

Internal: gluteus minimus, gluteus medius, tensor fascia latae (L4, 5; S1)

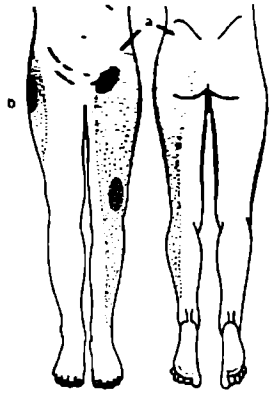
Important structures adjacent to the joint include the neurovascular bundle anteriorly, and the sciatic nerve running close to the posterior aspect.

In the adult the hip is an important site of involvement in osteoarthritis and, less commonly, other major arthropathies; Periarthicular lesions (bursitis, enthesopathy) are common. In the neonate and child, congenital dislocation, Pertes' disease, slipped femoral epiphysis, and sepsis are the principal conditions.

Symptoms

The hip joint is formed largely from the L3 segment. Hip pain is often ill defined, worsened by loading and movement (e.g. rising from sitting, standing, walking, putting on socks), and felt primarily in the anterior groin (164). However it may radiate widely to the anterior and lateral aspects of the thigh, the buttock, the anterior aspect of the knee, and rarely down the

front of the chin to above the ankle. Presentation may be with isolated knee pain (the hip and knee both contribute fibers to the obturator and femoral nerves).

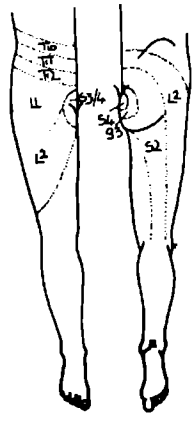


164 Pain distribution in (a) hip disease and (b) trochanteric bursitis.

(164)

Because of its wide and variable radiation, **hip pain requires differentiation** from a number of other local or distant causes, including:

- *Sacroiliac pain:* This is felt deep in the buttock, **with variable radiation down the** posterior thigh. It is often exacerbated by standing on one leg **(the affected side)**
- *Bursitis:* Trochanteric bursitis causes localized pain and **tenderness over the** trochanter, with occasional radiation down the lateral thigh. It is particularly painful when lying on the affected side (e.g. in bed) pain from ischiogluteal bursitis is felt mainly posteriorly and is particularly worsened by sitting.
- *Enthesopathy:* Adductor enthesopathy ('groin strain') usually follows sporting injury and causes pain in the medial groin, worsened by standing on the affected leg. Abductor enthesopathy produces similar pain to trochanteric bursitis but is usually worsened by walking.
- *Meralgia paraesthetica:* Entrapment neuropathy of the lateral cutaneous nerve of the thigh (beneath the inguinal ligament) causes burning pain and numbness over the anterolateral thigh. It may accompany massive or rapid-onset obesity, pregnancy, and wearing tight corsets or jeans.
- *Root pain.* Prolapsed intervertebral disc or lesions involving L1/L2 nerve roots (both rare) may produce groin pain (165). Its sharp quality and exacerbation by straining/coughing (± accompanying back pain) help suggest its nature.
- *Symphysitis:* This may produce supra-pubic pain and tenderness, worse during the stance phase of walking.



165 Dermatomes around the hip and thigh.

(165)

Examination

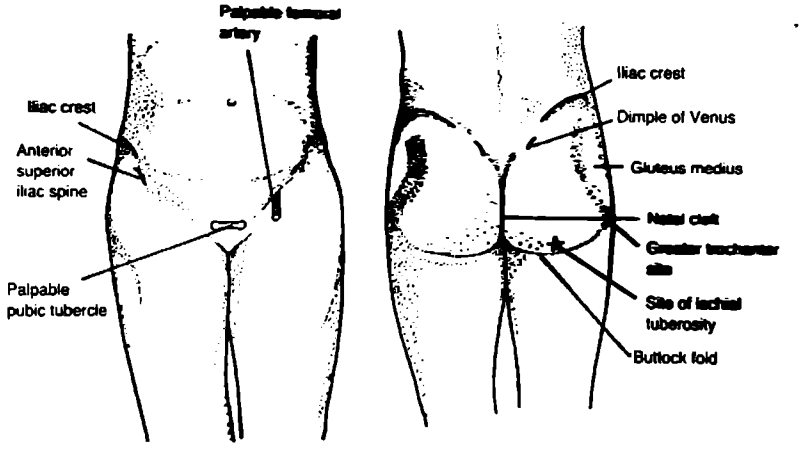
The patient, undressed to underwear, is examined standing, walking, and then lying.

Inspection of the standing patient

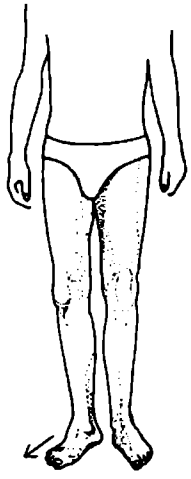
Ask the patient to point to the site of maximum pain and to delineate the area over which pain **is felt. Inspect** from in front, the side, and from behind.

Readily identifiable landmarks include the iliac crests, running between the anterior and posterior superior iliac spines, the greater trochanters, the ischial tuberosities, the gluteal folds, and the rounded proximal buttock muscles (166). From in front, look particularly for:

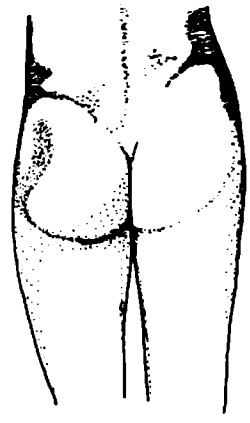
- *Pelvic tilt* - shown by loss of level between the anterior superior iliac spines. This may be due to adduction or abduction deformity form hip disease, a short leg, or primary scoliosis.
- *Rotational deformity* (167) - see whether the feet face forwards to the same degree.
- From the side, look particularly for:
- *Exaggerated lumbar lordosis* - this may indicate a fixed flexion deformity of one or both hips.
- From behind, look particularly for:
- *Pelvic tilt* (168) - shown by loss of level between the iliac crests and asymmetry of the gluteal folds. With fixed adduction, the abnormal side is elevated and the patient may be unable to place the ipsilateral foot flat on the floor. With an abduction deformity, the situation is reversed.



166 Anterior and posterior surface landmarks.



167 Rotational deformity.



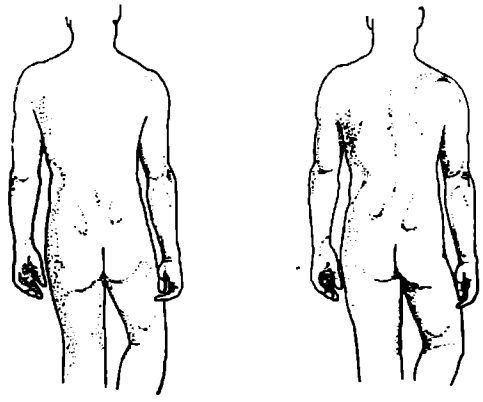
168 Pelvic tilt.

(166,167,168)

- *Scoliosis* - this often accompanies pelvic tilt.
- *Muscles wasting* - secondary to hip, primary muscle or neurological disease.

The *Trendelenburg test* shows up gross weakness of the hip abductors (gluteus medius, minimus). Ask the patient to lift one foot off the ground (169, 170). Normally, to retain balance, the abductors on the weight- bearing side contract to elevate the unsupported side. If the abductors are weak, the pelvic may drop down on the contralateral side: the patient may lose balance, stumble, and be unable to keep the foot raised. A modification is to stand facing the patient, providing support by holding the hands; as the foot is raised it is easy to appreciate the increased load transmitted from the patient with weak abductors.

The common causes of a positive trendelenburg test are hip disease (unilateral or bilateral), an L5 root lesion (unilateral) and conditions characterized by generalized weakness (usually bilateral).

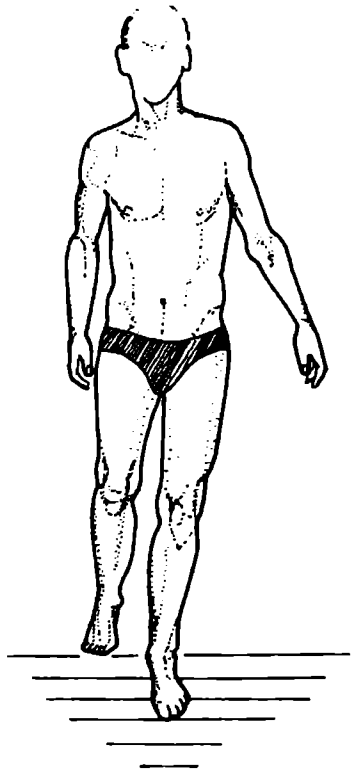


(169,170)

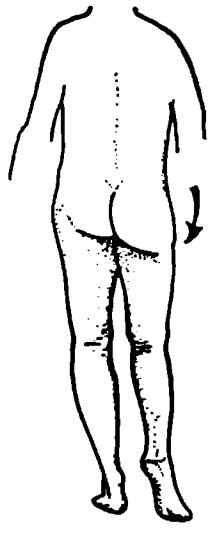
Inspection of the walking patient

Two non-specific gait abnormalities commonly result from hip disease:

- ***Antalgic gait*** (171)- usually indicating a painful hip. The patient shortens stance phase on the affected hip, leaning over the affected side to avoid painful contraction of the hip abductors.
- *Trendelenburg gait* ('abductor limp'; 172) - indicating weakness of the abductors on the affected side. During the stance phase on the affected side, the contralateral pelvis dips down and the body leans to the unaffected side. If bilateral, this produces 'waddling gait'.



171 Antalgic gait.



172 Trendelenburg gait.

(171,172)

Inspection of the patient lying on a couch

The patient, in general, should be lying straight out, as flat as is compatible with cardiorespiratory function. Insure that both anterior superior iliac spines are level and two legs are aligned.

Inspection

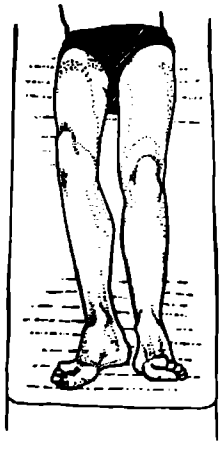
Look particularly for:

- *Skin changes* (especially scars, inguinal rash).
- *Swelling:* Swelling of the iliopectineal bursa may occasionally be apparent in the medial aspect of the groin. The hip joint is deep and swelling is usually not apparent. Anteromedial swelling extending down the thigh may occur with large synovial (Baker's) cyst extensions.
- *Deformity:* Particularly fixed flexion, external rotation or abduction deformity (these often accumulate sequentially as hip disease progresses; 173). With a severe flexion deformity the patient will be unable to straighten the legs without sitting up. With fixed adduction the affected leg may cross the other. Rotational deformities are obvious by looking at the position of the patella and the feet on the

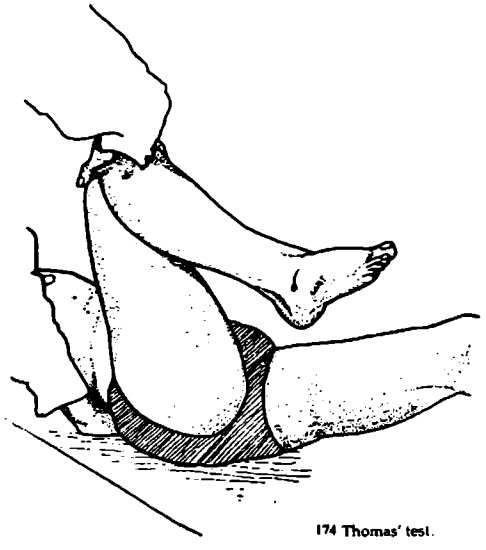
two sides. Restricted hip flexion may be compensated by an increase in lumbar lordosis which then 'masks' the fixed flexion deformity. If fixed flexion is not readily apparent utilize *Thomas' test.* (174) Flex the other hip to 90 degree to eliminate the lumbar lordosis (confirmed by placing a hand under the patient's lumbar spine) and watch for flexion of the affected hip.

- *Leg length inequality:* Apparent by looking at the heels. If there is apparent discrepancy use a soft tape measure to estimate at each side:

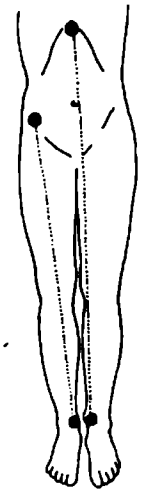
(a) The *true leg length* - between the anterior superior iliac spine and the medial malleolus (175). If one leg is flexed or externally rotated the other must be positioned similarly before measurement. Shortening (>1 cm) is often due to, but is not specific to hip disease.
(b) The *apparent leg length* - from the medial malleolus to a fixed point on the trunk (the xiphisternum is more 'fixed' than the umbilicus; in children the manubriosternal junction is more readily palpable). Inequality most commonly results from pelvic tilt.



173 Deformities: fixed flexion, external rotation, abduction.



174 Thomas' test.



175 True and apparent leg length.

(175)

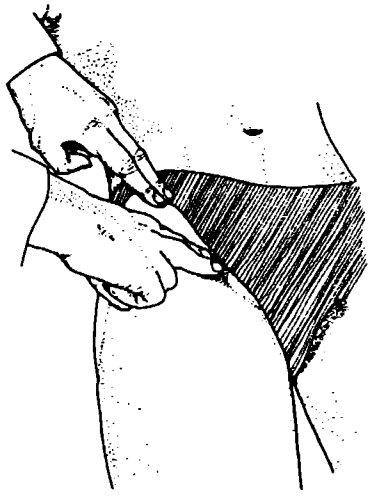
Attitude - a painful hip with synovitis is most confortable if held in mild flexion, abduction, and external rotation. Observe if this is the position the patient wants to adopt.

Palpation

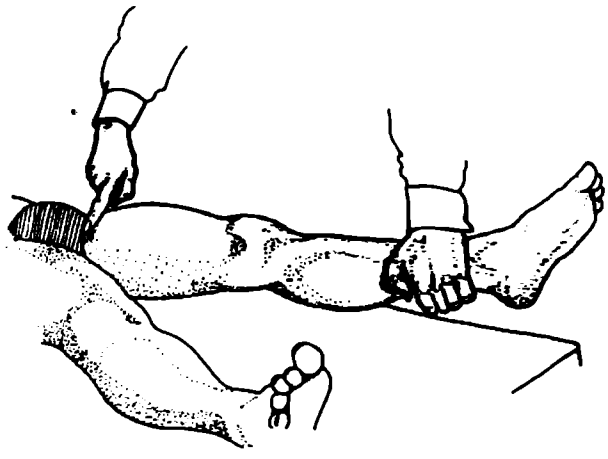
Palpate for tenderness (+- swelling) over the following areas:

- With the patient supine, palpate the anterior joint line just lateral to the femoral artery pulsation, below the middle third of the inguinal ligament (176). Tenderness here may reflect hip synovitis or iliopectineal bursitis. Bursal swelling may be palpable and give a positive balloon sign (reflecting localized bursitis or a synovial cyst communicating with an inflamed joint). Bursal swelling requires differentiation from other swelling in this region (particularly femoral hernia -

usually medial to the artery). Tenderness over the adductor origins along the superior or inferior aspect of the pubic bone may reflect adductor enthesopathy: resisted active adduction (177) may reproduce the pain.



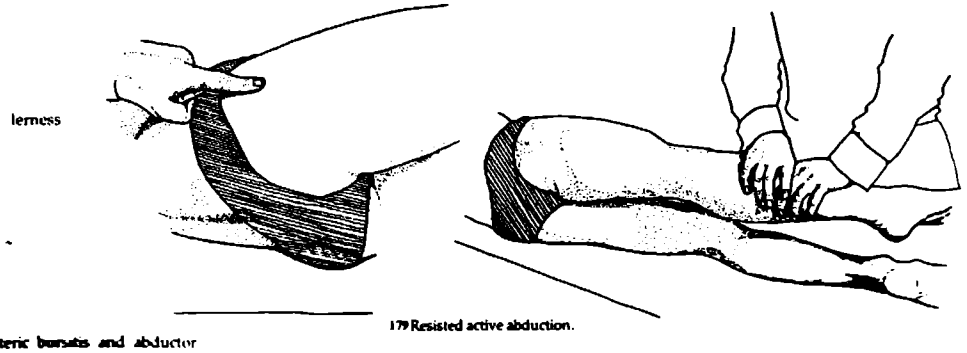
176 Palpation of anterior joint-line region.



177 Resisted active adduction and site of tenderness in adductor enthesopathy.

(176,177)

- With the patient on their side palpate around the greater trochanter for tenderness due to trochanteric bursitis or abductor enthesopathy (178). In obese subjects locate the trochanter by feeling proximally up the femur. Active abduction of the affected leg (alone or against resistance) may reproduce the pain of abductor enthesopathy (179), but will not usually worsen bursitis.



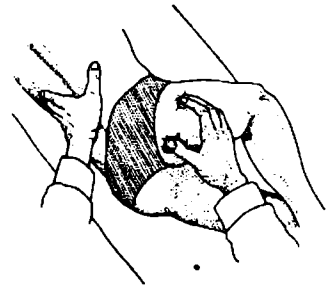
lerness

178 Palpation of trochanteric bursitis and abductor enthesopathy.

179 Resisted active abduction.

(178,179)

- With the patient still on their side flex their knee and hip and fell for prominent ischial tuberosity (180). Tenderness here suggests ischiogluteal bursitis (this is also an infrequent site for rheumatoid nodules).



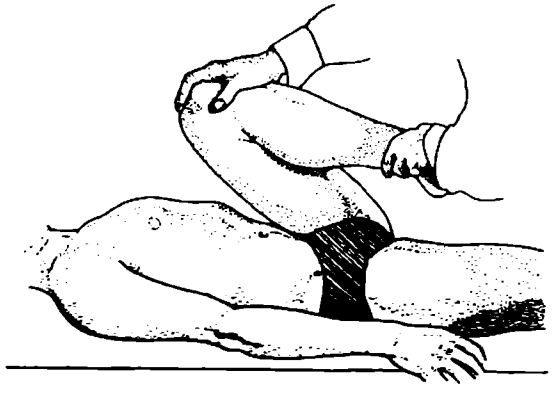
180 Palpation of ischial tuberosity and ischioglutcal bursitis

(180)

Movements

With the exception of extension, hip movements are best tested with the patient supine, look for restriction and presence of pain for each movement in turn.

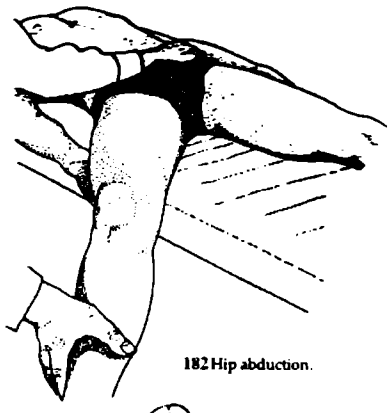
- *Flexion* (about 120-degree). This is tested with the knee flexed to relax the hamstring (181).



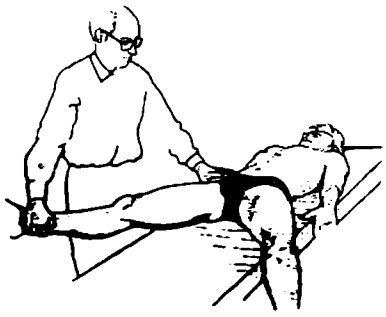
181 Hip flexion.

(181)

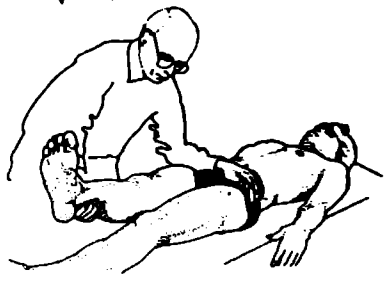
- *Abduction* (about 45-degree) and *adduction* (about 30 degree). With patient legs extended and the pelvis square, stabilize the pelvis with one hand on the opposite iliac crest, hold the ankle with the other hand and passively abduct the leg (182). The hand on the pelvis is to detect when hip abduction finishes (i.e. when the pelvis starts to move) and further lateral leg movement begins to result from lateral flexion of the lumbar spine. An alternative method is to fix the pelvis by keeping the opposite leg fully abducted, either extended along the couch or (with the knee flexed) draped over the side of the bed (183). For adduction cross the patient's leg over the other (184).



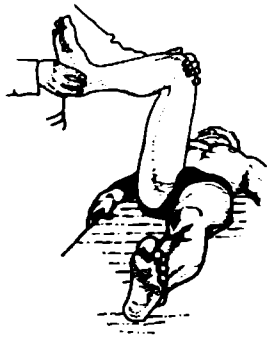
182 Hip abduction.



183 Hip abduction with pelvis fixed.



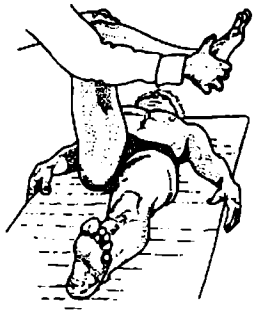
184 Hip adduction.



185 Internal rotation in flexion.

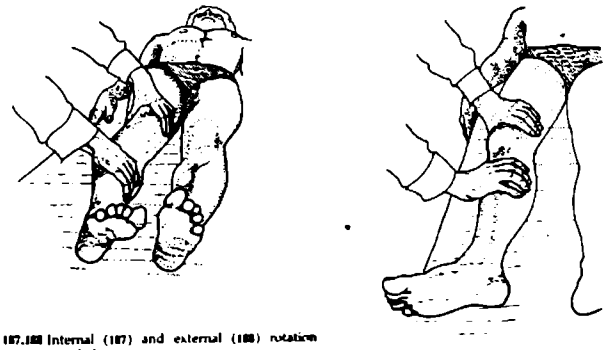
(182,183,184,185)

- *Internal* and *external rotation* (about 45-degree each). Flex the hip and knee to 90 degree and move the foot out laterally (internal rotation; 185) and medially (external rotation; 186). Internal rotation in flexion is the earliest and most constant movement to be affected by hip disease. Rotation can also be assessed with the hip extended and the leg straight: roll each leg on the couch, first one way and then the other, looking at the foot as an indicator of rotation 187,188).



186 External rotation in flexion.

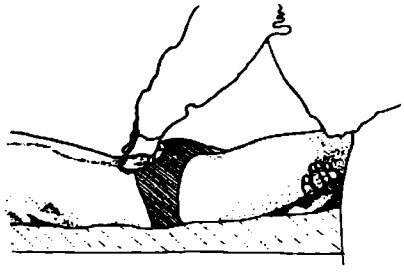
(186)



187,188 Internal (187) and external (188) rotation with hip extended.

(187,188)

- *Extension* (about 15-degree). Thomas' test detects loss of extension (i.e. fixed flexion). For smaller losses of extension lay the patient prone and attempt to immobilize the pelvis by downward pressure with one hand (over the sacrum) while extending the hip with the other hand (under the thigh: 189). If the patient has difficulty lying prone, lay them on their side and get them to flex the lower leg and hold it firmly (to stabilize the pelvis); stand behind the patient and support the upper leg while extending the hip, checking with the other hand over the lumbosacral junction for any spinal/pelvis movement.



189 Extension assessed in prone position.

(189)

Summary of hip examination

(1) Inspection of the standing patient
  (a) From in front (pelvic tilt, rotational deformity)
  (b) From the side (increased lumbar lordosis)
  (c) From behind (pelvis tilt, scoliosis, wasting) Trendelenburg test
(2) Inspection of the walking patient (antalgic gait, trendelenburg gait)
(3) Inspection of the patient lying on a couch
  (a) Inspection
    Skin
    Swelling
    Deformity
    Thomas test (fixed flexion)
    Leg length inequality (true + apparent leg length)

(b) Palpation:
Anterior joint line
Adductor origins
Greater trochanter (patient on side)
Ischial tuberosity (patient on side)
(c) Movements:
Flexion
Abduction, adduction
Internal, external rotation
Extension (patient prone or on side).

Suggested Reading

Clinical examination in Rheumatology. Michael Doherty. John Doherty

HIP PAIN

Differential diagnosis of lateral hip pain

- Adiposa dolorosa — prominent panniculus; tender to pinching
- Fibromyalgia — tenderness contralaterally and elsewhere
- Hip disease — pain is with all directions of motion; passive limitation
- Trochanteric — *Usually present* greater trochanter tenderness usually
- *Bursitis* — *at the posterior corner*
- Strain of the — tenderness proximal to trochanter;
- *Abductor muscles* — *resisted abduction painful*
- *Spinal disease* — *pain triggered by motion, usually hyperextension*
- Compressive — Burning quality; suspect subcostal in pronounced
- *neuropathy of subcostal* — *scoliosis; iliohypogastric if there is reproduction*
  or iliohypogastric nerve — with digital nerve compression
- Herpes zoster — the typical rash may be delayed days to 2 weeks

Trocanteric bursitis

Summary of Diagnosis

- Pain in the lateral hip region.
- Absence of paraesthesia.
- Absence of a palpable mass (if present, consider true bursitis and sarcoma).
- Tenderness at greater trochanter.
- Pain on leaning on the affected side.
- Usually, pain on resisted hip abduction.
- X-rays are indicated if there are reasons to suspect acute calcific tendinitis or a structural problem of the coxofemoral joint.

Key Points

- Several mimicking conditions must be excluded, in particular lumbosacral spine conditions and compressive neuropathies (iliohypogastric nerve, subcostal nerve).
- Seek an underlying musculoskeletal disturbance involving the spine, pelvis or lower extremities.

- Injection treatment is highly effective in the short term, but long-term improvement requires correction or palliation of the underlying problem.

Meralgia paraesthetica

Summary of Diagnosis

- Paresthesias in anterolateral thigh.
- Point tenderness medial to anterior superior iliac spine.
- Hyposthesia within the paresthetic area.
- Consider clinically spinal, retropritoneal, abdominal, and pelvic disease, as well as other causes of neuropathy.

Key Points

- Symptoms are usually mild.
- The condition resolves spontaneously in more than half of patients.
- Metabolic neuropathy; pelvic, abdominal, or retropritoneal compression; and spinal disease may masquerade for Meralgia paraesthetica.
- Treatment includes removal of compression (weight reduction), local infiltration, and in rare cases decompressive surgery.

Iliopsoas bursitis

Summary of Diagnosis

Irritative.

- Pain in groin, buttock, and anterior thigh.
- No pain on rotations or resisted flexion and external rotation.
- Reproduction of pain with passive flexion.

Effusive

- Asymptomatic.
- Edema, ischemia, femoral nerve paralysis, or pelvic organ compression in an individual with coxofemoral joint disease.
- Documentation by ECHO, CT, or MRI that shows cystic collection in front of coxofemoral joint.

Key Points

- The condition may present with pain resembling hip joint disease.
- These cases, which are due to trauma, respond to rest plus anti-inflammatory medications or to a Corticosteroid infiltration.
- In most cases, however, symptoms reveal vascular or neurologic compression of pelvic organs by a distended bursa.
- ECHO, CT or MRI may document the bursal cyst.
- Treatment of iliopsoas bursal distension is the treatment of the hip joint disease that causes the bursal cyst.

PATIENT 4:An elderly man who was in the waiting list for bilateral knee replacement was referred for evaluation of back pain. His spine was stiff and he could hardly move the neck. Hips only had a jog of extremely painful motion. Interestingly, rather than in the vicinity of the hip, pain was perceived in the anterior knee Finally, by having the patient lie supine near

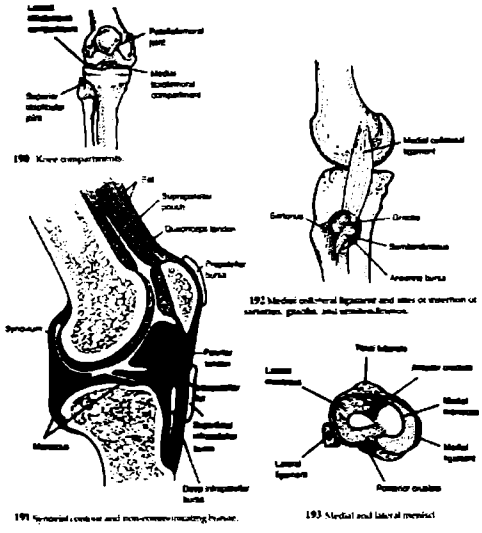
the edge of the examining table, both knees could be brought without difficulty from full extension to at least 90 degrees flexion, which further ruled out a knee origin of his pain. As expected, an AP pelvic radiograph revealed bilateral sacroiliac joint fusion and destroyed hips. ankilosing spondylitis was diagnosed and bilateral total hip (rather than knee) replacement gave the patient over 10 years of acceptable function.

Suggested Reading

Rheumatology in primary care/ Juan J. Conaso

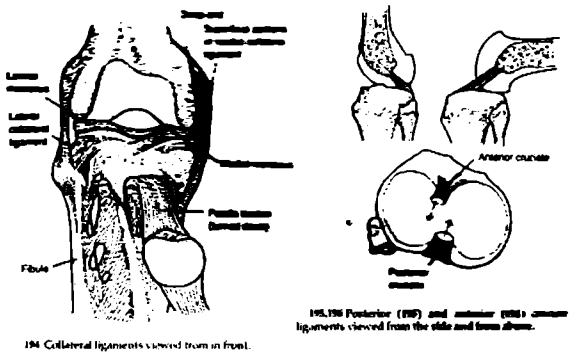
KNEE PAIN

The knee is the largest synovial joint and contains the largest sesamoid (the patella). The three compartments (medial and lateral tibiofemoral and patellofemoral) share a common cavity (190). The suprapatellar synovial reflection (pouch) is more extensive on medial aspect and offers little resistance to fluid distension. Posteriorly in the popliteal fossa, the synovial cavity is more constrained; its contour is molded by tendons into convoluted recesses the largest of which is semimenbranosus and lateral and medial gastrocnemius bursa and the subpopliteal recess (all of which communicate with the main cavity). Non-communicating bursa also occurs, the most important clinically being prepatellar bursa, the superficial and deep infrapatellar bursa (191), and the anserine bursa (192).



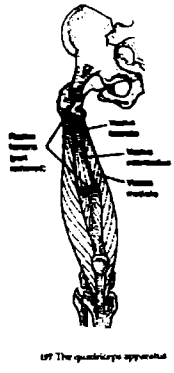
(190,191,192,193)

The two-fibrocartilage menisci (semilunar cartilage) are important loads transmitting structure (193). The medial meniscus has a thickened outer and thin inner edge; it attaches centrally to the intercondylar tubercles and medially to the capsule. The lateral meniscus attaches to the popliteus and is more mobile than the medial meniscus (and therefore less easily torn). The medial collateral ligament is broad, flat, and attached firmly to both the capsule and the medial meniscus (192). The lateral collateral ligament (194) is a longer cord-like structure attaches to the femur and fibula independent of the capsule. The two Cruciate ligaments (195, 196) are named according to their Tibial attachments: they are intracapsular, partly covered by synoviom, and attaches to the condyles in the notch. Knee stability depends on the collateral and cruciate ligaments, the capsule, the patellar ligament and good muscle tone.



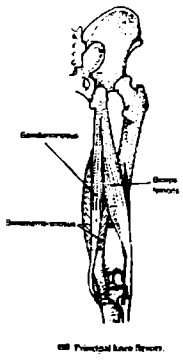
(194,195,196)

The patella keeps the quadriceps tendon 1-2 cm anterior to the femur, increasing its mechanical advantage. The tendency for the patella to undergo lateral translocation (Due to the shallower lateral angle of the groove and the lateral pull of the main bulk of quadriceps) is mainly prevented by the pull of the vastus medialis. The vastus medialis, vastus intermedius, and vastus lateralis arise from the femur (197) and provide the strong extensor apparatus that stabilize the knee, particularly on weight bearing: the vastus medialis produces the more distal muscle bulge and contracts maximally in the last 10 degree of extension, taking part in the locking or 'screw home' medial rotation of the femur on the tibia. The rectus femoris (the fourth quadriceps component) arises from the anterior inferior iliac spine and thus acts across two joints.



(197)

The main flexors are the hamstrings (the semimenbranosus and semitendinosus medially and the biceps femoris laterally, (198): they are most effective when the hip is flexed.
Ancillary flexors are the gracilis, sartorius and the medial gastrocnemius (medially) and popliteus and the lateral gastrocnemius (laterally). When the knee is flexed ('unlocked') the tibia can rotate on the femur, 40-degree externally and 30-degree internally: the lateral hamstring and tensor facia lata externally rotate the tibia, and the medial hamsrings and the popliteus internally rotate the tibia.



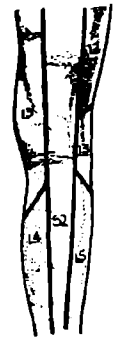
(198)

The knee is involved in most forms of arthropathy; it is also a common site of direct and indirect trauma, which may result in cartilage or ligament damage, enthesopathy, or bursitis.

Symptoms

Pain From the knee (199) is predominantly felt anteriorly, often with localization to - compartment involved (e.g. anteriorly in patellofemoral disease and anteromedially and anterolaterally in medial and lateral compartment problems, respectively). Pain rarely radiates far from the knee: prominent radiation down the tibia normally implies marked subconderal bone collapse or intraosseous hypertension. The front of the knee represents the L2/3 dermatomes (200, 201) and pain may be referred to this site from an L3 root lesion or from the hip. Referred pain often differs from pain originating in the knee in being (1) less clearly demarcated, (2) frequently accompanied by pain and aching above the knee and (3) exacerbated by different factors. For example, L3 root pain often begins in the buttock, later affecting the front of the thigh and the knee; it is not aggravated by walking but may be exacerbated by coughing. The back of the knee represents the S1/2 dermatomes (201). Posterior knee pain alone suggests a complication of arthropathy (e.g. popliteal cyst, posterior Tibial subluxation) or an S2 root lesion; other local causes include hamstring or gastrocnemius enthesopathy, lymphadenopathy, and popliteal aneurysm.

199 Site of pain arising from knee (a), and sites of referred pain to the knee (b, spine; c, hip).



200,201 Dermatomes around knee: anterior (200) and posterior (201).

(199,200,201)

'Locking' is a sudden, usually transient, painful inability to extend the knee. As a symptom, it is important in suggesting a mechanical derangement, e.g. torn meniscus, 'loose' body, or trapping of a fold of the synoviom ('plica syndrome').

'Giving way' describes a feeling of apprehension and loss of confidence in weight bearing on the knee. It predominantly accompanies problems relating to the qudriceps/patellar mechanism or stability. Weakness of the quadriceps, particularly the vastus medialis or patellofemoral disease, alters vertical 'tracking' of the patella as it moves on the femur and gives rise to this odd feeling of apprehension. Ligamentous instability also alters the mechanics of the knee during weight bearing, so that the patient knows that 'things are not right'.

Patellofemoral abnormalities commonly give rise to two characteristic features in the history:

Anterior knee pain, which is much worse going up and down (particularly down) stairs or negotiating an incline than walking on the flat. This is because of the maximal stress through that compartment when weight bearing on a flexed knee.

Progressive anterior knee pain/aching that develops during prolonged sitting with the knee flexed. The patient typically gets up, stretches the legs, and the aching disappears, only to return after 20 min or so of again sitting with knees bent.

Inspection

The patient should be inspected while weight bearing and walking and then on a couch at rest. As usual, comparison of one side with the other may help show abnormality associated with unilateral lesions.

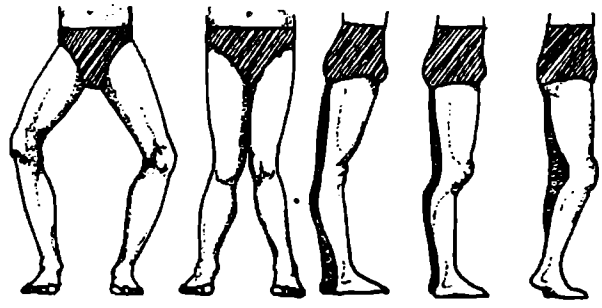
Inspection of the standing patient

The patient stands upright and is inspected from the front, the side, and behind. The main features to observe are deformity and swelling in the posterior popliteal fossa, since these are more apparent when standing than lying.

Deformity

At birth the knee is usually in marked varus: during the toddler and early childhood phases, valgus is common; during adolescence, the knee tends to straighten again. Conditions that cause cartilage loss in both tibiofemoral compartments commonly allow the knee to resume valgus, which is the natural tendency in the majority of individual.

All deformities other than fixed flexion are best assessed while the patient is standing (202-206):



202-206 Knee deformities: (202) genu varus; (203) genu valgus; (204) genu recurvatum; (205) posterior tibial subluxation; and (206) fixed flexion.

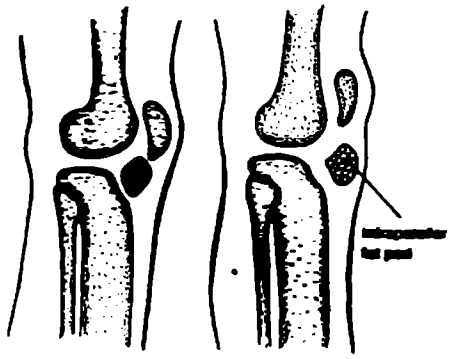
(202,203,204,205,206)

varus and valgus may be noted with the patient lying on a couch, but these are inevitably exaggerated on weight bearing. Principal deformities are:

- *Genu varus* (bow legs). This commonly reflects isolated medial compartment disease (cartilage loss ± subchondral bone collapse) and is the characteristic deformity of uncomplicated osteoarthritis.
- *Genu valgus* (knock-knees): this is the usual deformity of arthropathies characterized by synovitis and tricompartmental involvement, leading to cartilage loss throughout the knee
- *Genu recurvatum:* This is particularly characteristic of generalized hypermobility.
- ***Posterior Tibial subluxation:*** This produces a step back deformity and is particularly characteristic of arthropathies that affect the developing knee.
- *Fixed flexion.* The knee cannot extend and is always in some degree of flexion. This may complicate a variety of arthropathies, but is particularly common in conditions characterized by synovitis and resolution by fibrosis (e.g. seronegative spondyloarthropathies). If the patient experiences pain on weight bearing, and the deformity is obvious, manual correction of the deformity (e.g. reducing varus or valgus by pushing from the side) will help suggest whether the pain is predominantly mechanical and, therefore, likely to be helped by correction of the deformity.

Swelling

A popliteal cyst may produce a prominent swelling in the popliteal fossa when the patient is weight bearing and the leg is extended. An abnormally high patella (patella alta) may produce a 'camel sine' (207, 208): because high patella (hump 1) the infrapatellar fat pad (hump2) becomes more prominent. When sitting with knees flexed at 90 degree, the patellae of such patients may point upwards and be laterally placed ('frog's eyes' appearance). Varicose veins should also be noted.



207,208 Normal patella position (207) and two swellings ('camel sign') due to patella alta (208).

(207,208)

Inspection of the walking patient

Gait is described in Chapter 2. Look particularly for antalgic gait, a short step due to fixed flexion, and circumduction of the leg due to fixed extension.

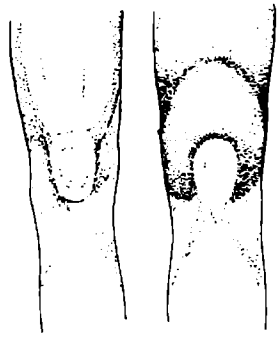
Inspection of the patient lying on a couch

Skin changes

The anterior (extensor) surface of the knee is a common site for psoriasis. Look also for erythema (either localized over a bursa, or more generalized if the knee is involved), scars, or other abnormality.

Swelling

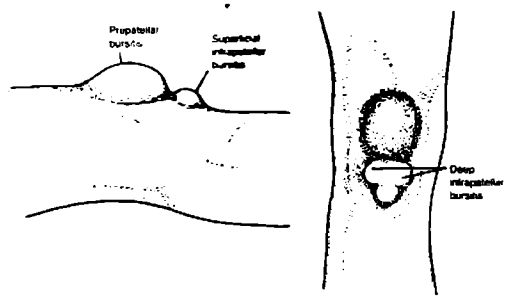
Knee effusion: fluid collecting in the knee first fills in the medial dimple at the side of the patella and then expands the suprapatellar pouch, giving a horseshoe swelling above and to either side of the patella (209, 210).



209,210 Normal knee contours (209); swelling of knee effusion (210).

(209,210)

Bursa/fa: pads: localized swelling in front of the patella suggests prepatellar bursitis (211). Localized swelling apparent below the patella, in front of the patellar tendon, suggests superficial infrapatellar bursitis (211); less prominent swelling either side of the tendon suggests deep infrapatellar bursitis or a large infrapatellar fat pad (212). A prominent medial fat pad (especially in obese women) may produce a large swelling with ill-defined boundaries above or below the medial joint line; more subtle swelling below the medial joint line may be seen with anserine bursitis.



211,212 Swellings of prepatellar bursitis and superficial infrapatellar bursitis (211 side view); swelling of deep infrapatellar bursitis (212; front view).

(211,212)

Muscle

Inspect the quadriceps for wasting (comparison with the other side is helpful in unilateral lesions). Although all the quadriceps wastes uniformly, wasting of the bulky vastus medialis (particularly in a fit young male) may be the most conspicuous.

Quadriceps wasting is a difficult sign, particularly in the middle-aged/elderly, and especially in women. Some asymmetry of muscle bulk is common and not necessarily abnormal (e.g. it may relate to usage and 'footedness'). Measuring quadriceps (thigh) girth with a tape measure at a fixed point (e.g.10 cm) above the patella on each side is often recommended, but has problems with reproducibility and lack of agreement as to what difference between the sides constitutes abnormality.

Deformity

Fixed flexion is best assessed while the patient is lying and attempted to straighten the legs. Other deformities may also be noted but are usually greater on weight bearing.

Attitude

The way the patient positions the leg and their ease in getting on and off the couch may give an idea of pain severity. The patient will keep returning to a flexed-knee position if there is synovitis or a tense effusion causing intra-arthicular hypertension.

Palpation

Temperature

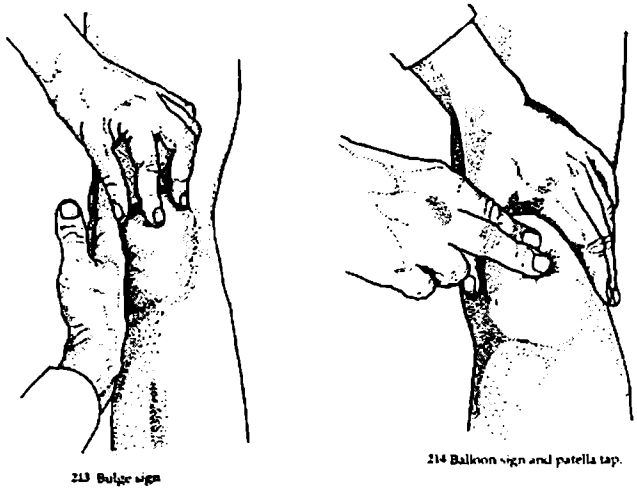
Run the back of the hand over the leg anteriorly and down each side, comparing skin temperature above and below the knee to that over the knee. If anything, the knee normally feels cooler than the thigh or shin. Increase in temperature may reflect synovitis (widespread,

mainly felt over the suprapatellar pouch) or bursitis (localized). If increased warmth is found, be careful this does not relate to varicose veins (often most apparent when standing).

Swelling

Fluid within the joint can be detected by one of three signs.

Bulge sign (213): This detects a small amount of fluid (and is not necessarily abnormal). Stabilize the patella while gently massaging down either side of the patella, in turn, observing the opposite side around the medial and lateral dimples. A small amount of fluid may flick from one side of the pouch to the other.



213 Bulge sign

214 Balloon sign and patella tap.

(213,214)

Balloon sign: With a moderate/tense effusion the bulge sign is usually lost and the balloon sign becomes positive (214). Place the palm of one hand over the patella with the fingers and thumb off to the medial and lateral sides; then press firmly down and in with the hand - this automatically encourages fluid to flow down towards the main cavity at the tibiofemoral junction. If pressure is now applied onto the patella or inferior joint region with the other hand 'ballooning' of the first hand is felt. This is the most specific test for fluid in the knee.

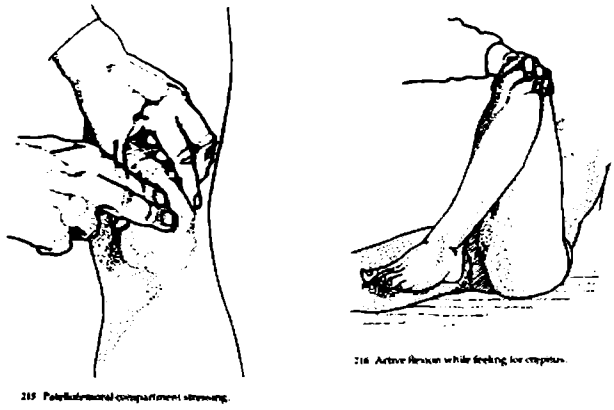
Patella tap: While testing for a balloon sign, the patella may be felt to move through the displacing fluid and then 'tap' or 'clunk' onto the femur (214). Although common with a large effusion this also occurs with marked retropatellar or anterior femoral fat.

Fluid may be detected in prepatellar or superficial infrapatellar bursitis by placing a finger and thumb either side of the swelling and pressing at its apex for a balloon sign. For deep infrapatellar bursitis, press over the patellar tendon and feel for ballooning to either side. A balloon sign is less commonly present with anserine bursitis.

Patellofemoral compartment

Press the patella back onto the femur with one hand while steadying it with the other (215): medial and lateral movement of the patella may then elicit tenderness and give rise to

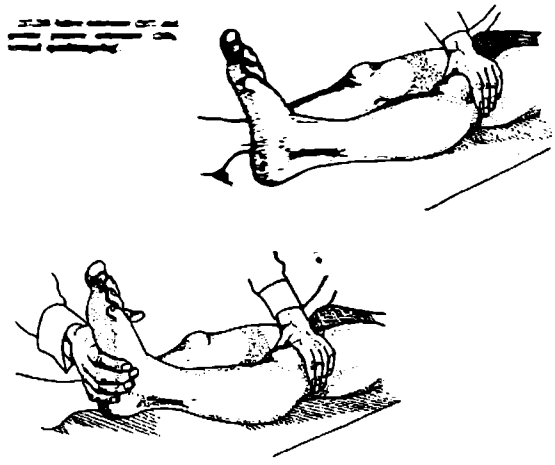
crepitus, felt by both hands. Alternatively, stress the compartment by asking the patient to tighten the quadriceps by pushing the knee backward into the couch; this pulls the patella onto the femur and may reproduce the pain, of which they complain, (This procedure produces no hip or other knee compartment movement). Localization of predominant tenderness to either the medial or lateral facet can be determined by pushing the patella medially and then laterally, in turn, out of its tracking, with the quadriceps well relaxed, while palpating firmly the medial and then lateral facets from each side.



(215,216)

Active assisted and passive movement

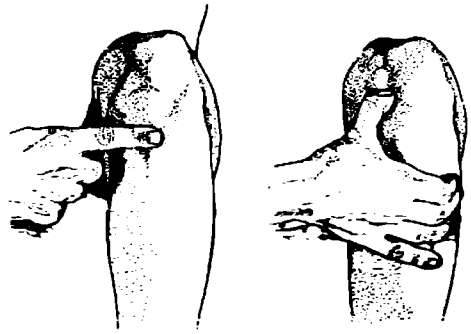
The range of active flexion is assessed with the examiner's hand draped over all three compartments (palm over the patella, fingers medially and thumb laterally) to detect crepitus at each site (216). Observe flexion from the side as the patient attempts to put the heel to the bottom (normally about 115-135 degree), and inquire concerning pain, particularly in a 'stress pain' pattern (i.e. maximal towards the extremes of limited flexion and extension). Compare active extension from the flexion position with passive extension (i.e. lift the heel upward from the couch): passive extension will correct any 'quadriceps lag' (217,218). Decreased active extension usually results from muscle atrophy (to complete the last 15 degree of extension a 60% increase in force of quadriceps is required). Extension may also be reduced in joint disease (similar for active and passive), or increased (>10 degree) in an unstable, damaged knee or in generalized hypermobility.



(217,218)

Tibiofemoral compartments

To identify joint lines, position the knee in moderate flexion. The Tibial tubercle is readily found in the midline (219), and may be locally tender in Osgood-Schlatter disease.



(219,220)

As the palpating finger is taken medially ad then proximally from the tubercle the expansion of the Tibial plateau is readily identified; as the finger goes higher the anterior joint line is felt as a valley dipping backwards between the tibia (below) and the femoral condyle (above) (220). Internal and external rotation of the tibia will open up the lateral and medial joint lines, respectively, and permit easier identification in difficult cases (e.g. obese subjects). Having found the anterior medial joint line, press firmly just medial to the patellar tendon; then, follow the medial joint line around, pressing firmly all the way. Tenderness localized to the anterior joint line is characteristic of medial meniscal injury, whereas more generalized medial joint-line/capsular tenderness suggests arthropathy. Repeat the same procedure for the lateral tibiofemoral joint line; again, localized anterior tenderness suggests meniscal pathology whereas generalized tenderness favors arthropathy.

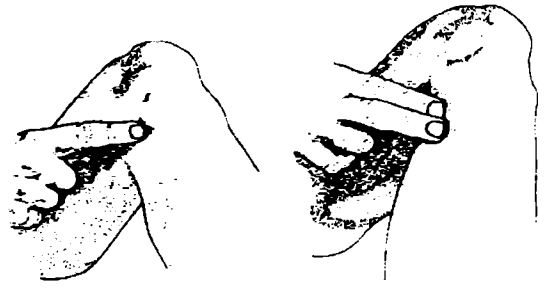
While palpating both anterior joint lines, the examiners assess the presence of any soft-tissue swelling. Synovial thickening may cause fullness at both anterior joint lines, with a visible convex bulge: if pressed it will give, but then immediately reform as pressure is released. This may be a misleading sign for a synovial thickening, since a prominent infrapatellar fat pad may appear identical. Deep infrapatellar bursitis is another cause of swelling either side of the patellar tendon; however, it may feel warm, give rise to a balloon sign, and have a more definite medial and lateral boundary. Localized swelling arising laterally, or occasionally medially, from the tibiofemoral joint lines may be due to meniscal cyst: such a swelling may pop in and out of the joint line as the knee flexes/extends.

Periarthicular lesions:

Having identified and palpated both tibiofemoral joint lines, palpate for localized sites of Periarthicular tenderness. There are no visual landmarks and it is best to take a single finger and palpate firmly over a wide area below and then above, the joint line on each side. The following lesions are most commonly found.

Inferior medial collateral ligament enthesopathy

This produces localized tenderness inferior to the medial joint line, roughly in the midline of the tibia when observed from the side (this condition is very common, 221).



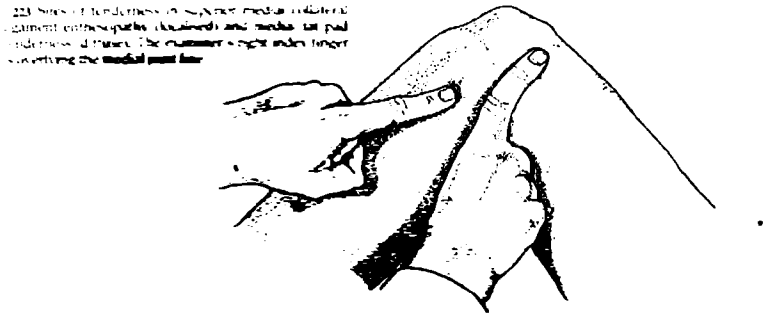
(221,222)

Anserine bursitis

This produces diffuse area of tenderness inferior to the medial joint line, often overlapping the site of the inferior medial collateral ligament insertion (222). It may additionally produce localized swelling, warmth, and, occasionally, a balloon signs. The bursa is named because of the similarity of its contour to that of the webbed goose foot; it lies between the medial collateral ligament and the tendons of the sartorius, gracilis, and semitendinosus, close to their insertion (this lesion is common, particularly in middle-aged and elderly subjects).

Superior medial collateral ligament enthesopathy

This produces localized tenderness above the medial joint line, fairly centrally on the femur when observed from the side (223).



(223)

Medial fat pad syndrome

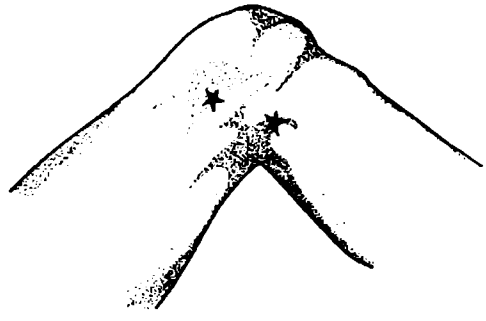
This produces a wide area of tenderness and 'doughy' swelling superior to , and often overlapping, the medial joint line (this lesion is common, even in non obese patients).The fat pad is also a common site for tenderness in fibromyalgia.

Inferior lateral collateral ligament enthesopathy

This produces localized tenderness over the fibula head, felt posteriorly on the lateral side (this condition is rare).

Superior collateral ligament enthesopathy

This causes localized tenderness superior to the lateral joint line, centrally on the femur when observed from the side (224). Lateral ligament strains are uncommon sporting injuries that characteristically produce a painful arc under flexion loading at 15-30 degree.



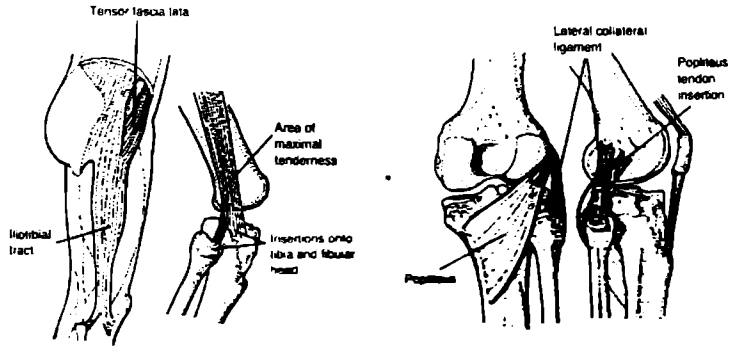
224 Sites of tenderness in inferior and superior lateral collateral ligament enthesopathy.

(224)

Iliotibial tract ('band') syndrome

This produces a line of tenderness that extends from anterolateral tibia, across the joint line, and up the side of the thigh; tenderness is usually maximal over the lateral femoral condyle (225). Predominantly a sporting injury, this syndrome also causes painful arc at about 30

degree. Pressure over the lateral femoral condyle as the knee is passively moved from full flexion to extension may produce pain at about 30 degree of flexion (Noble compression test).



225 [illegible] 226 Popliteal tendon enthesopathy.

(225,226)

Popliteal tendon enthesopathy

This is predominantly a sporting injury that produces localized tenderness on the lateral femoral condyle in a more anterior position than the superior insertion of the collateral ligament (226). It arises, particularly, from running on a cambered, sloped, or uneven track, which strains the popliteus as it attempts to reduce the rotational movement of the tibia on the femur (popliteus problems may be associated with injury to the lateral meniscus, to which it is attached).

The popliteal fossa

This is palpated with the knee in mild-to-moderate flexion: swelling and tenderness are the main features of interest.

Swelling

If a swelling is felt, it may be possible to confirm communication with the joint by massaging its contents back into the main synovial cavity with the knee in flexion. Maintain pressure on the popliteal fossa, extend the knee, then remove both hands: the swelling will not reappear until the patient flexes the knee several times, confirming a valve-like communication between the main cavity and the 'cyst'. The connection is usually patent only with the knee in mid-flexion, permitting fluid to pass in either direction: as the knee is fully extended or flexed, tendons and other posterior structures close off the valve.

Tenderness

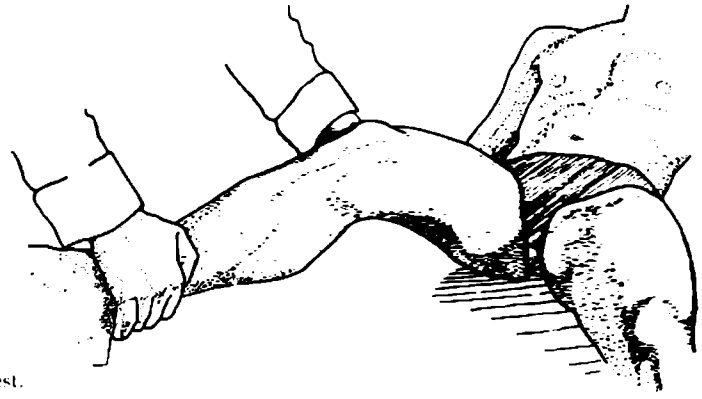
The medial or lateral hamstring tendons or their insertions, or the medial or lateral insertions of gastrocnemius may be tender to palpation in runners with hamstring or gastrocnemius enthesopathy (particularly following sprinting or running uphill when these muscles are working at full stretch). Such strains are occasionally accompanied by fine crepitus over the tendons.

Stability

Although a large number of tests for instability exist, none is totally specific for a single lesion. The following are standard screening tests for ligament or capsule damage:

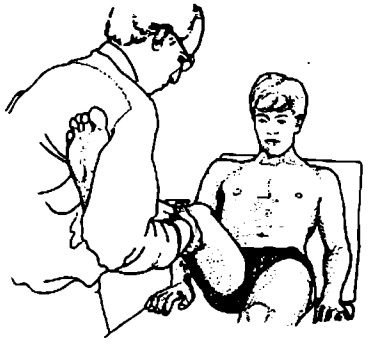
Collateral ligaments

Assess stability with the knee 'unlocked' in mild flexion (with the leg straight, the cruciates also prevent lateral movement). Push the femur medially with one hand and the tibia laterally with the other hand (227), looking for excess lateral movement of the tibia (medial ligament instability). As long as sufficient purchase can be achieved to demonstrate this sign the method of holding the leg is immaterial (some examiners place the patient's foot in their armpit and grasp the tibia firmly with both hands to apply greater pressure: 228). In addition to excess lateral movement, also note:



227 Medial ligament stress test.

(227)



228 Medial ligament stressing using firmer grasp of lower leg.

(228)

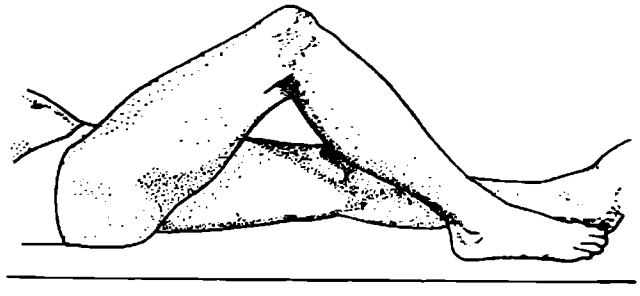
- Opening up of the medial joint line ('gap sign').
- Medial knee pain, particularly at the inferior insertion of the ligament, suggesting collateral ligament enthesopathy (this maneuver is basically a stress test for the medial collateral ligament).

The lateral collateral ligament is similarly tested with the knee in mild flexion, the examiner pushing the tibia medially and the femur laterally. Again, observe for excess lateral movement, a gap sign, and pain.

Cruciate ligaments

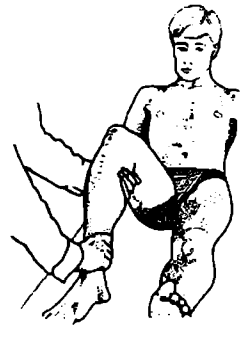
These are examined with the knee flexed to 90 degree and the hip to 45 degree (229). Before testing for excess movement:

- Palpate the hamstrings to ensure they are relaxed (otherwise, thy may restrict anterior-posterior movement of the tibia and conceal Cruciate instability).
- Observe the rounded contour of the knee from the side to ensure the tibia is not starting in a posteriorly subluxed position (posterior 'sag' sign) due to posterior Cruciate instability. Having made these checks, test for excess anteroposterior movement of the upper tibia on the femur. Steady the distal tibia with one hand while levering the upper tibia anteriorly and then posteriorly with the other hand (230); the patient's weight will hold the femur steady. Some examiners prefer to sit on the patient's foot to steady the lower leg; however, this is unnecessary and may cause pain in those with arthropathy or other painful lesions of the feet. If there is excessive anterior movement ('anterior drawer' sign) this may reflect anterior Cruciate instability, cartilage loss, or generalized hypermobility. Comparison with the other knee and other tests for hypermobility should help interpretation of a positive sign. If the tibia is pushed posteriorly, excess posterior movement implies posterior cruciate instability or, again, cartilage loss or generalized hypermobility.



229 Position for cruciate ligament testing

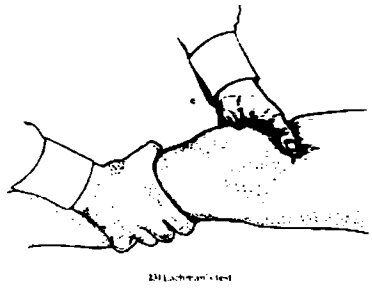
(229)



230 Anterior drawer sign

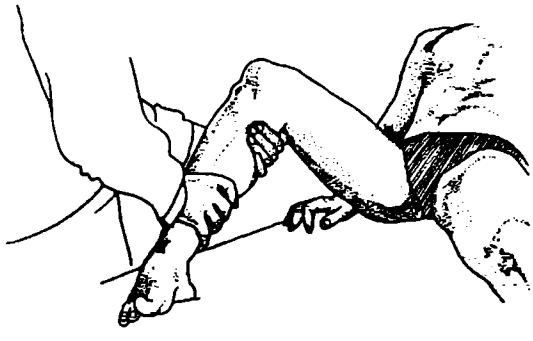
(230)

Lachman's test is a sensitive test for anterior cruciate injury (particularly of the posterolateral fibers). With the knee in mild flexion (<30 degree) and the patient relaxed, grasp the femur with one hand and the upper shin with the other hand (231), and pull the tibia anteriorly to demonstrate excessive movement and a soft 'end feel' (this requires well-relaxed patient and large examining hands).



(231)

If an anterior drawer sign is detected, *Slocum's test* for anterolateral and anteromedial instability may be performed (232). With the patient positioned as for the drawer test, sit on the couch and passively rotate the tibia medially 30 degree, keeping it medially rotated by resting the foot against your buttock. This maneuver tightens the lateral capsule, giving enough stability then to eliminate the anterior drawer sign: if the anterior drawer is still positive in this position (most anterior movement occurring on the lateral side) it is likely that the lateral capsule (and/or lateral collateral ligament) is also damaged. Similarly, externally rotate the tibia to tighten the medial capsule: a positive anterior drawer in this positions (most movement occurring on the medial side) usually implies that medial capsular fibers (and/or medial collateral ligament) are damaged.



232 Slocum's test.

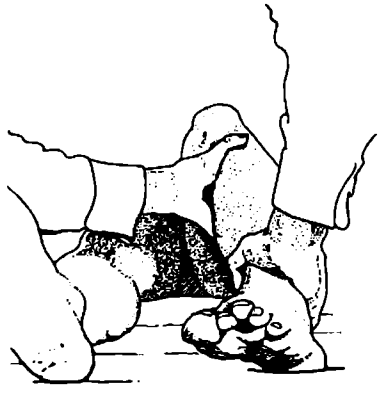
(232)

Additional tests for mechanical derangement:

If the history (e.g. 'locking') or examination suggests that the problem is primarily mechanical, further tests for mechanical derangement may be of use.

The 'pivot' shift maneuver (MacIntosh test)

Another test for anterolateral rotatory instability, this maneuver is used to demonstrate a dynamic subluxation where the tibia slips laterally and anteriorly on the femur. The patient is positioned supine, with the hip flexed (20 degree) and relaxed in slight medial rotation, and the knee slightly flexed (5 degree). The examiner medially rotates the lower tibia with one hand, the other hand pushing the upper tibia anteriorly on the femur while maintaining a valgus stress (233). As the knee is then flexed to 30-40 degree the tibia will suddenly reduce backwards with a 'clunk'. The reduction is due to the iliotibial band moving from an extensor to a flexor function, pulling the tibia back to a normal position. Normally, the knee's center of rotation changes constantly through its range of motion as a result of the shape of the femoral condyles, ligamentous restraint, and muscle pull. A positive pivot shift test usually suggests damage to the anterior Cruciate, the posterlateral capsule, or the lateral collateral ligament.



233 Pivot shift test.

(233)

The mediopatellar plica test

Pain produced by pushing patella medially with the knee flexed at 30 degree may be due to a plica squeezed between the femoral condyle and the patella.

The 'apprehension' test

If the patella is carefully pushed laterally with the knee flexed at 30 degree, the patient may resist, contract the quadriceps, and express insecurity if there is recurrent patellar subluxation or dislocation.

SUMMARY OF KNEE EXAMINATIN

(1) Inspection of the standing patient
- (a) Form in front (particularly for genu valgus, genu varus)
- (b) From the side (particularly for genu recurvatum, posterior Tibial subluxation)
- (c) From behind (particularly for popliteal cyst)

(2) Inspection of the walking patient

(3) Inspection of the patient on a couch

(a) Inspection (knee extended) for:
- Skin changes
- Swelling (effusion, bursa, fat pad)
- Quadriceps wasting
- Deformity (particularly fixed flexion)
- Attitude

(b) Palpation (knee extended) for:
- Temperature increase
- Swelling (effusion, bursitis)
- Patellofemoral tenderness, crepitus

(c) Palpation during flexion (crepitus, restriction, pain)

(d) Passive extension

(e) Palpation (knee flexed)
- Tibiofemoral tenderness, swelling
- Periarthicular tenderness
  - Collateral ligament enthesopathy
  - Anserine bursitis
  - Medial fat pad syndrome
  - Iliotibial tract syndrome
  - Popliteal enthesopathy
- Popliteal fossa (cyst, tenderness)

(f) Stability of ligaments
- Medial/lateral collateral stress tests
- Anterior drawer (Slocum's test if positive)
- Posterior drawer

Suggested Reading

Clinical Examination in Rheumatology. Michael Doherty. John Doherty

KNEE PAIN

COMMON KNEE CONDITIONS

Anterior

1-Prepatellar or pretendinous bursitis
2-Quadriceps tendon tendinitis
3-chondromalacia patella
4-Patellofemoral osteoarthritis
5-Plica medio patellaris
6-tight lateral retinaculum
7-Patellar tendon tendinitis
8-Osgood-schlatter disease
9-infrapatellar bursitis
10-Hoffa's body inflammation

Medial

1-Anserine bursitis
2- Medial meniscus tears
3-Medial (tibial) collateral tendon bursitis
4-Semimembranous bursitis

Lateral

1-Iliotibial tract syndrome
2-Bicipital tendon tendinitis
3-Lateral meniscus tears and cysts
4-Popliteal tendon bursitis (posterolateral)

Posterior (pain and/or mass)

1- Baker's cysts
2-Thrombophlebitis
3-Popliteal artery aneurysms
4-Mucoid degeneration of popliteal artery wall
5-Ganglia
6-Sarcomas

Iliotibial tract syndrome:

Summary of Diagnosis

- Pain in the lateral knee with tenderness 2-cm proximal to arthicular line.
- Rarely, lateral trochanteric pain.
- The Ober's maneuver may be positive in-patients with a tight iliotibial tract.
- X-rays or other imaging studies are unnecessary.

Key Points

- The condition causes lateral knee pain.
- Most cases are due to excessive running.
- A tight iliotibial tract may be shown in some of the patients.
- Conditions to be excluded include bicipital tendinitis, a lateral meniscus tear, a lateral miniscal cyst, and popliteus tenosynovitis.
- Patients usually improve with activity modification, local heat, a NSAID and physical therapy to lengthen a shortened iliotibial tract.
- Recalcitrant cases should referred to an orthopedic surgeon to rule out alternative diagnosis and for a Corticosteroid infiltration if diagnosis is confirmed.

Prepatellar bursitis:

Summary of Diagnosis

- Prepatellar or pretendinous swelling.
- Fluctuation.
- Redness and marked tenderness suggest infection or gout.
- X-rays are unnecessary.
- Aspirate to diagnose bacterial infection, gout, or trauma by the characteristics of the fluid

Prepatellar bursitis

Key Points

- Is often the result of recurrent local trauma.
- Traumatic bursitis tends to be subacute or chronic; septic or gouty bursitis tends to be acute or subacute.
- Diabetes and alcoholism are frequent associations of septic bursitis.
- Acute arthritis may be mimicked. However, in prepatellar bursitis, knee extension is minimally or not at all impaired.
- Septic prepatellar bursitis is a more serious and difficult to treat condition than its olecranon bursa counterpart. It usually requires hospitalization, catheter or surgical drainage, and parentral antibiotics.

Patient 5: a young woman presented to the emergency room with fever, rigors, and acutely inflamed right knee. Septic arthritis was initially considered, but six attempted knee aspirations yielded no fluid. A more experienced observer noticed that, inconsistent with septic arthritis, the involved limb could be brought painlessly to full extension and suggested the possibility of septic prepatellar bursitis. Aspiration of the prepatellar bursa yielded 0.5 cc of turbid fluid. Smears showed gram positive cocci arranged in chains, and group A streptococci grew on culture. A CT scan obtained 1 hour later revealed a large subcutaneous fluid collection at the outer aspect of the joint. The air bubbles, from 1cc of air injected in the prepatellar bursa, are proof of the rupture of the bursal sac. The fluid collection was drained with a pigtail catheter, the knee was immobilized with a plaster splint, and intravenous penicillin was begun. Drainage ceased in 4 days. A slow improvement led to full recovery within 2 months.

Anterior knee pain:

Summary of Diagnosis

Patellar

- Patellar pain mancuvers.
- Search for associated anserine bursitis.
- Knee x-rays in older individuals (to stage knee osteoarthritis in the three compartments).

Infrapatellar

- Palpate tendon, Tibial tubercle, Hoffa's body, infrapatellar bursa.
- Lateral x-rays (amorphous calcifications, Tibial tubercle fragmentation in Osgood-schlatter disease, erosion).
- Axial view of the patella with the Merchant view or the Laurin view to determine the presence of patellar subluxation or tilt.
- Bursal aspiration and HLA-B27 determination may be appropriate in some cases.

Key Points

- Determine whether pain is patellofemoral, parapatellar, or infrapatellar.
- Determine if secondary anserine bursitis is present.
- Chondromalacia patella, plica syndrome, lateral retinacular tightness, infrapatellar bursitis, patellar tendon enthesopathy, and Osgood-Schlatter disease have a good prognosis.
- Patellofemoral osteoarthritis is a progressive condition.

- The presence of secondary anserine bursitis provides a treatable pain source.
- Isometric quadriceps stretching if needed are an essential component of treatment.
- Rheumatology input should be sought in enthesopathy.
- Orthopedic consultation should be sought in all other cases if there is a failure to respond to initial treatment.

Anserine bursitis:

Summary of Diagnosis

- Diagnosis is clinical based on focal tenderness 5 cm distal to the medial arthicular line of the knee.
- Local swelling may represent a distended anserine bursa, but other lesions affecting soft tissues or bone must be ruled out.
- X-rays should be obtained if indicated for the primary condition affecting the knee.
- X-rays and other imaging procedures, in particular ultrasound and possibly MRI, should be obtained when upper medial Tibial swelling is present.

Anserine bursitis

Key Points

- A frequent cause of medial knee pain in-patients with structural knee problems.
- Processes that need to be ruled out include medial miniscal tears and bursitis of the medial collateral ligaments; painful lipomatosis; fibromyalgia; and when swelling is present, soft tissue tumors and bone tumors affecting the upper medial tibia.
- Diagnosing anserine bursitis means identifying a treatable process.
- Corticosteroid infiltration is highly effective in this condition and often renders a painful osteoarthritic knee painless.

Other bursitides in the medial knee:

Summary of Diagnosis

- Pain after over-exercise or trauma.
- Tenderness (rarely swelling) exposed by knee flexion: collateral ligament bursitis.
- Swelling sometimes tenderness around semimembranosus: semimembranous bursitis.
- MRI distinguishes bursitis from a medial meniscal tear. Both lesions may be present. MRI is important in the prearthroscopic evaluation of these patients.

Key Points

- Bursitis of the medial collateral ligament bursa is a common condition; bursitis of the semimembranosus bursa is rare.
- Both result in medial knee pain.
- A medial meniscal tear is the main differential diagnosis.
- Bursitis of the medial collateral ligament may block extension, further suggesting a meniscal tear.
- MRI clearly distinguishes these lesions.
- Treatment of bursitis includes rest, ice, and an anti-inflammatory agent; rapid regression follows a Corticosteroid infiltration.

Meniscal tears:

Summary of Diagnosis

- History of trauma.
- Pain and possible catching or locking by moving and stressing the damaged compartment.
- A positive Lachman test indicates associated anterior cruciate ligament tear. If an effusion is present, aspirate: Hemarthrosis suggests concurrent cruciate ligament tear.
- MRI and arthroscopy, depending on which is available, are diagnostic of meniscal and ligamentous tear.

Meniscal tears:

Key Points

- Meniscae are important in joint congruency and an even distribution of loads.
- Meniscal tears may occur in isolation, in association with an anterior Cruciate ligament tear, and in OA of the knee.
- Finding in meniscal tears include pain, locking, popping, and swelling, with a clear effusion.
- Anterior Cruciate ligament tears result in Hemarthrosis and an unstable knee that gives way during pivoting motions.
- Tears are best diagnosed by MRI.
- The meniscus should be saved if at all possible; this includes suturing a peripheral tear and removing the loose fragment in central tears involving the avascular zone.
- Postoperative physical therapy, including continuous passive motion and resistive exercises, is essential to minimize muscle atrophy and for muscle recovery.
- Hinged protective braces are important in the conservative and postoperative treatment of ligamentous injuries.

Baker's cysts:

Summary of Diagnosis

- Mass in medial one-third of popliteal fossa that softens in flexion (Fousher's sign).
- Antecedent or concurrent joint findings (synovitis, osteoarthritis).
- X-rays useful in evaluation of the joint; usually do not show the cyst.
- Echography: best to demonstrate intact cysts and for the initial sorting out of other popliteal lesions.
- CT scan and MRI only used for assessment of the underlying joint disease.
- Noninvasive venous studies and sometimes venography are essential to rule out thrombophlebitis. A positive diagnosis of ruptured cyst may be possible by Echography, but arthrography is required in some instances.

Key Points

- Baker's cysts are a frequent cause of popliteal discomfort in patients with knee conditions; they fluctuate with the knee condition, and their basic treatment is the treatment of the knee condition.
- Other tumoral conditions including sarcomas and popliteal artery aneurysms should be considered and excluded based on clinical findings and Echography.

- Pseudothrombophlebitis from a ruptured Baker's cyst and thrombophlebitis may be clinically identical. Furthermore, they occasionally coexist.
- Inadvertent anticoagulation in a ruptured cyst may cause serious calf hematoma.
- Symptomatic Baker's cysts in children are usually the result of irritation or trauma, are not associated with a knee condition, and tend to regress spontaneously.
- Baker's cysts occur frequently in children with arthritis; only a minority is symptomatic and therapy aimed at the cyst is not required.

A painful, swollen calf: clues that suggest pseudothrombophlebitis

1-A history of knee ailment
2-Knee swelling preceding calf swelling
3-Presence of a knee effusion
4-Knee pain preceding calf symptoms, and evidence of structural knee disease
5-Tenderness at cleft between semimenbranosus tendon and medial head of gastrocnemius

Suggested Reading: Rheumatology in primary care/ Juan j. Canoso

ANKLE AND FOOT PAIN

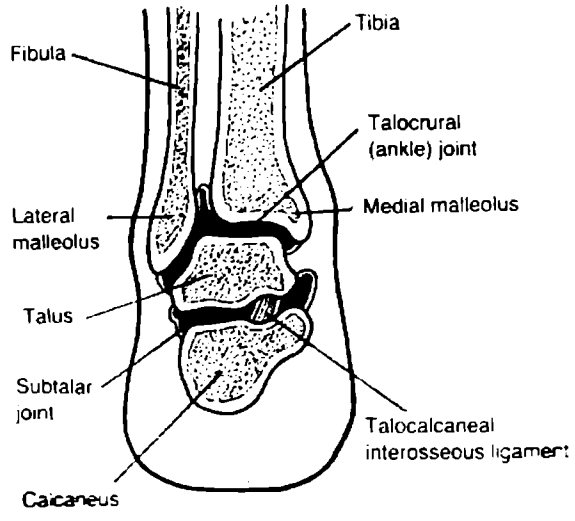
The lower leg, the ankle, and the foot are well structured for stability in weight bearing and for propulsion during bipedal gait. The large numbers of bones and their shape permit both flexibility and stability: although movement between individual joints is small, their combined motions permits controlled locomotion over of variety of ground surfaces.
The foot has three functional units:

- *The hind foot:* (the calcaneus and the talus).
- *The midfoot:* (the five small tarsal bones).
- *The forefoot:* (the metatarsals and the phalanges).

Posteriorly the bones lie over each other, while in the midfoot and forefoot they lie side by side. This makes the foot higher and narrower at the back, and produces the two (longitudinal and transverse) arches of the foot.

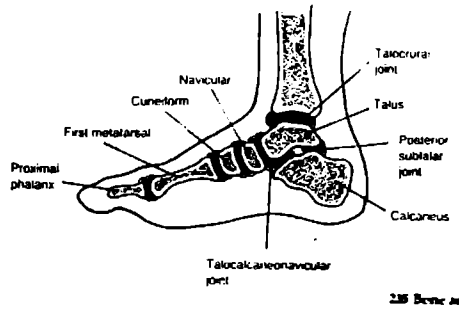
HINDFOOT JOINTS

The true ankle (talocrural) joint is a hinged synovial joint between medial malleolus (tibia) the lateral malleolus (fibula), and the talus (234, 235), permitting dorsiflexion and plantar flexion. The fibula aids stability but transmit little weight (the inferior tibiofibular joint is a syndesmosis that permit only a small 'spread' of the ankle mortise during dorsiflexion). Because the trochlea of the talus is wider anteriorly the joint is tighter and more stable in dorsiflexion (as in climbing uphill) than in plantar flexion (descending). The capsule is tightest medially and laterally, where it is bound down by ligaments, but lax in front and behind, being most extensive anteriorly. The enclosed synovial space is usually separate, having no communications.



234 Bones and joints of the ankle and the midfoot.

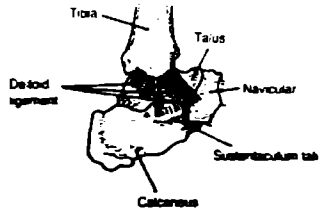
(234)



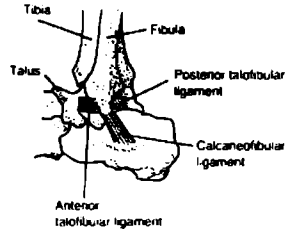
235 Bone and joint arrangement

(235)

Strong collateral ligaments maintain stability (236, 237). The single medial *deltoid* ligament resists eversion of the foot and is very strong (avulsion of the malleolus often occurs before ligament rupture). The lateral ligament is in three bands (the anterior and posterior talofibular ligaments and calcaneofibular ligament). The anterior talofibular ligament is the first to undergo stress during inversion, and is the most commonly torn.



236 Ligaments of the ankle (medial aspect)



237 Ligaments of the ankle (lateral aspect).

(236,237)

The calcaneofibular ligament may tear, but only after rupture of the anterior talofibular ligament; disruption of both leads to ankle instability. The posterior talofibular ligament is damaged only in severe trauma.

The posterior subtalar joint is between the concave undersurface of the talus and the posterior convex facet of the calcaneus (see 235). The capsule of this deep joint is tight and permits

little synovial expansion. Together with the talocalcaneal portion of the talocalcaneonavicular joint, it allows eversion and inversion of the hindfoot.

MIDFOOT JOINTS

The midtarsal joint is a functional composite formed mainly by the talocalcaneonavicular and calcaneocuboid joints, which permit forefoot eversion and inversion (some forefoot abduction and adduction is also possible). Movement is principally talonavicular but also involves movement between the coneiform and cuboid bones.

FOREFOOT JOINTS:

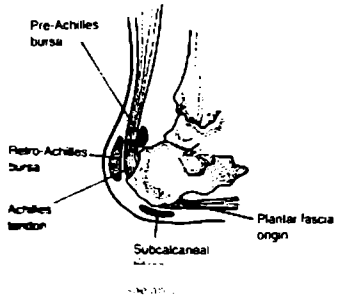
The metatarsophalangial joints (MTPJs) and proximal and distal interphalangial joints (IPJs) are synovial joints similar to the MCPJs and IPJs of the hand. Each MTPJs capsule is strengthened by a medial and lateral collateral ligament, an extensor tendon dorsally, and a plantar ligament below. MTPJ stability is dependent mainly of the capsule and if capsular function is deranged (e.g. by synovitis) the toes become unstable and follow the pull of the tendons (dorsal subluxation with valgus deformity).

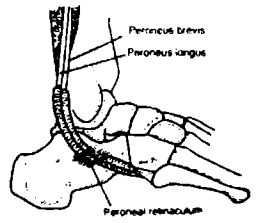
THE ARCH OF THE FOOT:

This permit equal weight distribution between the heel prominence (calcaneus) posteriorly and the heads of the lateral four metatarsals and two sesamoid bones of the first metatarsal anteriorly. It provides flexibility and spring for walking and running. The longitudinal arch has a high, flexible medial component and a low, rigid lateral component; the transverse arch is high proximally and low distally. The arch is maintained by the joint capsule and the dorsal and plantar ligaments, and is supported by the long calf muscle tendons (the role of the plantar fascia and small muscles of the sole is uncertain).

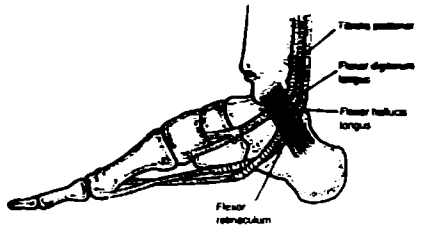
TENDONS, BURSAE, AND FASCIA

The Achilles tendon, the common insertion of the soleus and the gastrocnemius, attaches to the posterior aspect of the calcaneus and is separated from it by the retrocalcaneal (pre-Achilles) bursa (238). Other clinically relevant bursa include retro-Achilles bursa (between the skin and the Achilles tendon), the subcalcaneal bursa (between the skin and the undersurface of the calcaneus), and the adventitious bursae that form over the medial aspect of the first MTPJ ('bunion') and lateral aspect of the fifth MTPJ ('bunionette').





239 Tendons and tendon sheaths around the ankle (lateral).



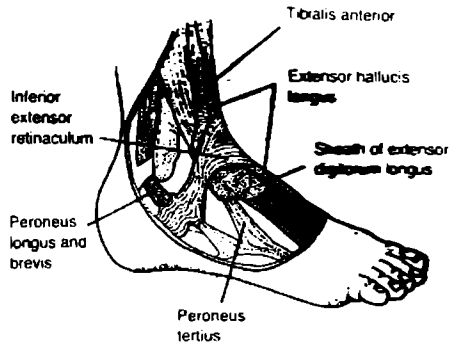
240 Tendons and tendon sheaths around the ankle (medial).

(238,239)

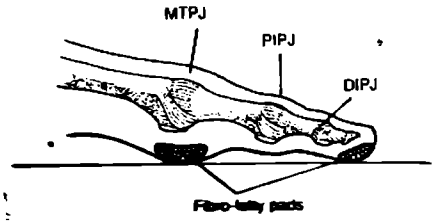
Behind each malleolus run tendons within individual tendon sheaths (239, 240):

- Lateral malleolus: the peroneus longus and the peroneus brevis (eversion) held down by the peroneal retinaculum.
- Medial malleollus: the tibialis posterior (inversion) and, more posteriorly, the flexor digitorum longus and the flexor hallucis longus held down by the flexor retinaculum (forming the tarsal tunnel through, which also passes the posterior Tibial nerve).

Anterior and superficial to the ankle are three tendon sheaths held down by the extensor retinaculum (241): the medial sheath contains the large tendon of the tibialis
Anterior (the main dorsiflexor of the foot); the central sheath contains the extensor hallucis longus and extensor digitorum longus; and the lateral sheath contains the peroneus tertius.
The plantar fascia arises from the median prominence of the calcaneus. It is tough and thick proximally, but thins as it extends and divides distally before insertion into the bases of the metatarsal heads (see 238).
The skin on the lateral aspect of the foot, toes and heel is greatly thickened, and in the subcutaneous connective tissue beneath the metatatarsal heads and tips of the toes are fibro-fatty pads that act as shock absorbers (242). As a result of the concentrated stresses they receive, the foot and ankle are common sites for traumatic arthicular and Periarthicular lesions, as well as being target sites for major arthropathies.



241 Tendons and tendon sheaths around the ankle (anterior)



242 MTPJs and fibro-fatty pads

(241,242)

SYMPTOMS

Pain arising from arthicular and Periarthicular structures is generally well localized and its sites and characteristics alone often suggest the cause.

HINDFOOT PAIN

Pain from the ankle joint is felt anteriorly in a broad line joining the two malleoli, and is characteristically worsened by standing or walking. Conversely the pain from subtalar joint is felt mainly posteriorly between the two malleoli and is particularly aggravated by walking over uneven surfaces when eversion/inversion is required.

Localized posterior heel pain and tenderness may result from retro-Achilles bursitis retro-calcaneal bursitis, or Achilles tendinitis or enthesopathy. With Achilles tendon problems, the pain often is exacerbated by standing on tip-toe (without shoes). Pain beneath the heel, which is worsened by prolonged standing or walking, is usually a result of plantar fascia enthesopathy ('plantar fasciitis').

MIDFOOT PAIN

Midtarsal joint disease gives pain in the bootlace area, often most marked during late stance and toe-off phases of walking. Loss of normal arches ('flat foot') may cause pain in the midsole.

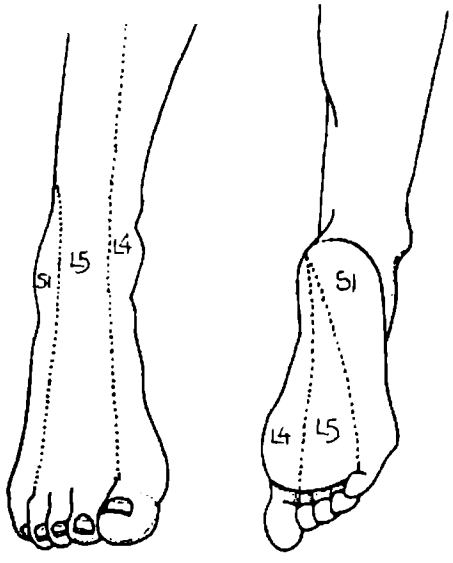
FOREFOOT PAIN

MTPJ pains felt below the metatarsal heads (metatarsalgia): it is worsened by standing and walking, and may be described as 'like walking on marbles' if several joints are involved. Burning pain in the sole and toes suggests a neurogenic cause. Morton's neuroma typically causes sharp intermittent pain between the third and fourth digits, particularly when metatarsal heads are compressed (as with restrictive shoes). Tarsal tunnel syndrome (posterior Tibial nerve entrapment) typically causes burning, tingling, and numbness in the distal sole and toes.

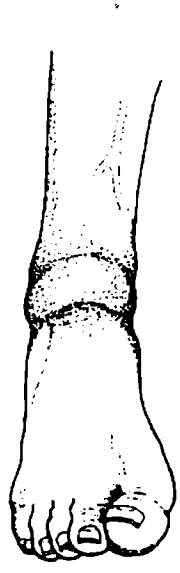
Inflammation of any tendon sheath may cause localized pain that often extends a distance along the line of the sheath and is aggravated by movement of the relevant muscle.

REFERRED PAIN

Pain may be referred to the ankle and foot from the spine and, rarely, the hip. Referred pain from nerve root irritation follows dermatomal distribution (243, 244), may be exacerbated by straining and straight leg raising, and may be accompanied by neurological signs.



243,244 Dermatomes of the lower leg and the foot: (243)dorsal aspect; (244) sole.



245 Swelling of ankle synovitis.

(243,244,245)

EXAMINATION

Inspect both feet and ankles while the patient undressed to their underwear, stands and then walks. Undertake further inspection and palpation, with the patient resting on a couch. Additional information may be obtained by inspecting the patient's footwear for abnormal molding and wear patterns of the sole and heel.

Inspection of the stand*ing* patient

Compare both sides of the patient, from in front, behind and from the side, looking particularly for the following abnormalities.

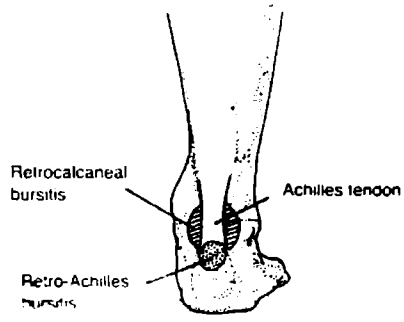
Swelling

Ankle synovitis produces diffuse swelling anteriorly (245) often filling the small depressions in front of the malleoli. Midtarsal synovitis produces only modest, diffuse puffiness over the dorsum of the midfoot. MTPJ synovitis often causes swelling over the dorsum of the forefoot, obscuring the extensor tendons and causing spreading of the metatarsals and toes. The combination of IPJ synovitis and digital flexor tenosynovitis may produce a 'sausage toe'.

Linear swelling those crosses the ankle is usually a result of extensor tenosynovitis. Peroneal or tibialis posterior tenosynovitis produces linear or diffuse puffiness around the lateral or medial malleolus, respectively.

Swelling around Achilles tendon may result from tendinitis (producing swelling of the tendon itself), retrocalcaneal bursitis (appearing more as eccentric swelling that fills in either side of

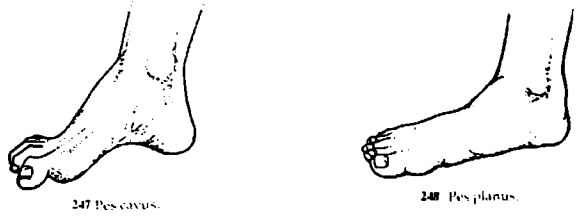
the tendon), or retro-Achilles bursitis (more prominent, superficial swelling; 246). This is also a common site for nodule formation, usually appearing as superficial, eccentric swellings over the Achilles tendon and posterior calcaneus.



(246)

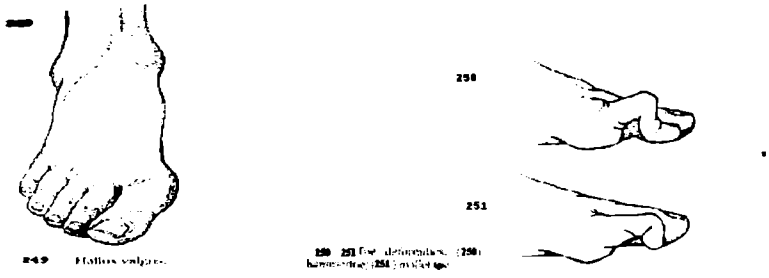
Deformity

Ensure that both arches are normal. Longitudinal arch may be increased (*pes cavus*, often a result of neurological disease; 247) or diminished (*pes planus* or flat foot; 248). in severe flat foot the displaced talus and navicular cause a prominence in front and just below the medial maleollus.



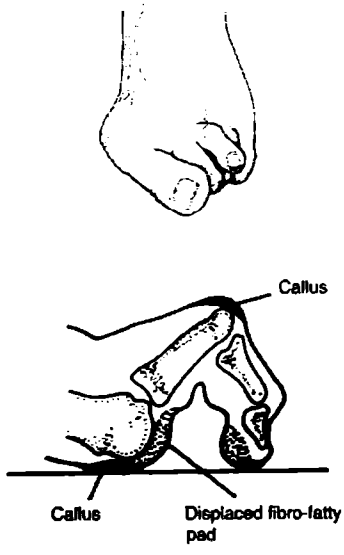
(247,248)

A large number of joint deformities may be seen. The most common toe abnormality is *hallux valgus* (249) in which there is abnormal angulation and rotation of first MTPJ: associated medial deviation of the metatarsal (metatarsus primus varus) results in a broad forefoot, and a bunion often develops. *Hammertoe deformity* (250) results of hyperextension of the MTPJ, with flexion at the proximal interphalangial joint, or PIPJ (usually the second toe, in association with hallux valgus). The distal iterphalangial joint, or DIPJ, may be straight, flexed or extended and a callus often develops over the prominent PIPJ. *Mallet toe* (251) results from flexion deformity at the DIPJ. *In cock-up toe* (252) flexion of both IPJs follows extension and often plantar subluxation at the MTPJ (usually due to polyarthritis). The fibro-fatty pad moves distally, and secondary callus may form below the exposed metatarsal head and over the flexed PIPJ.

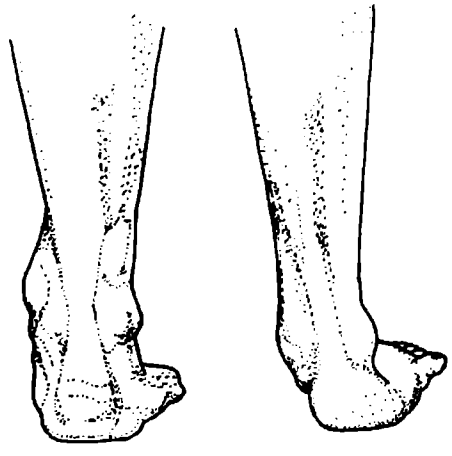


(249,250,251)

Ankle and subtallar deformities are best seen from behind. Calcaneovalgus (*eversion*) is most common (253, 254), usually reflecting damage to both the subtalar and ankle joint: calcaneovarus (inversion) only occasionally occurs. Fixed plantar flexion (*talipes equinus*) usually results from spastic paresis. Congenital clubfoot (talipes) may associate with plantar flexion of the ankle (talipes equinus) or dorsiflexion (*talipes calcaneus*). Midfoot deformity may coexist; for example *talipes equinovarus* or *talipes calcaneovalgus*.



252 Cock-up toe deformity with hallux valgus (due to polyarthritis).



253,254 Normal hindfoot (253) hindfoot eversion deformity (254)

(252,253,254)

Skin and nail changes:

Relevant observations may include loss of hair over the distal limbs (vascular insufficiency, neuropathy), vasomotor changes and discoloration (Raynaud's disease, vascular disease, cryoglobulinaemia), ulceration (gravitational, vasculitis), and psoriatic plaques. As with the fingers, inspect the nails for pitting and dystrophy (psoriasis, chronic Reiter's syndrome),

vasculitic lesions, and clubbing. Peculiar to toenails, usually the hallux, is onychogryphosis (gross distortion and hypertrophy).

Inspection of the walking patient

Gait is described in chapter 2. Pain in any part of the foot or ankle may give an antalgic gait. Other typical abnormalities may suggest the site of involvement.

Hindfoot problem

If ankle movement is reduced, the leg may be externally rotated and slightly abducted, giving a 'toe out' gait. With outward displacement of the forefoot, the patient attempts to walk by rolling the foot from the lateral to the medial side. If severe, this results in loss of the longitudinal arch so the patient walks on the medial aspect of the foot. If the heel is painful, heel strike is avoided, the step being shortened and the forefoot striking first. With Achilles tendon problems, push off is guarded or avoided, the step again being shortened.

Midfoot problem

The foot is often held inverted and push off is from the lateral side.

Forefoot problem

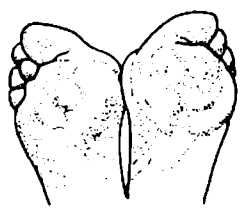
To avoid weight bearing on the forefoot the heel does not rise in late stance and there is diminished push off. The trunk, hip, and knee flex to maintain forward motion, and swing phase on the normal side shortens resulting in 'bobbing' during late stance on the painful side. Involvement of both forefeet combines to give a forward-leaning, short-stepped, shuffling gait.

Examination of the recumbent patient

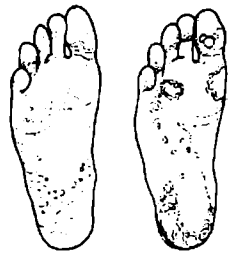
With the patient relaxed on the couch, complete inspection by examining the soles; then undertake palpation and examination of a range of movements.

Examination of the soles and the interdigital clefts

Callosities and inflamed adventitious bursa are particularly common under subluxed MTPJs (255). They appear to merge into the surrounding skin and may be tender to direct pressure. Complications that may be present (particularly in rheumatoid disease) include vasculitis, broken skin with discharge, and secondary infection. *Verrucae* are tender to direct and, particularly, laterally applied pressure, and show clear demarcation from surrounding skin. Pustular *psoriatic lesions* and *keratoderma blennorrhagica* appear identical (256, 257), and *plantar erythema* is occasionally observed, as on the palms. Part and inspect the interdigital clefts: macerated skin with fissuring is typical of *'tinea pedis'* (athlete's foot)-common in-patients with toe deformities and abnormal skin clefts.



255 Bursae beneath subluxed MTPJs.



256,257 Keratoderma blennorrhagica (256) early, mild; (257) established, severe

(255,256,257)

Palpation

Increased warmth

Pass the back of the hand down the dorsum of the foot to feel for increased warmth overlying the ankle, Midtarsal joints, or the MTPJs.

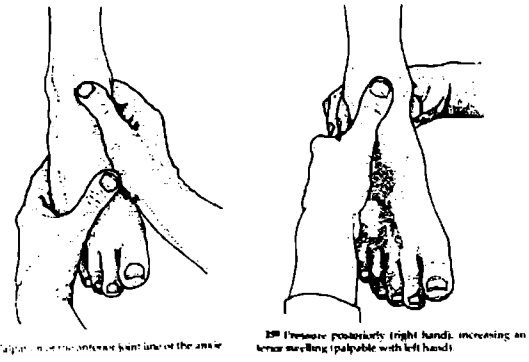
Joint tenderness, swelling, and movement

Work through each joint group looking for:

- Joint-line tenderness
- Soft-tissue swelling arising from the joint
- Restriction of passive movement
- Pain (especially stress pattern) during movement

Ankle

Identify the anterior joint line by palpating with one or both thumbs while gently dorsiflexing and plantarflexing the ankle (258). Note any crepitus during this movement. Having identified the joint lines presses firmly for tenderness, and feel for swelling.



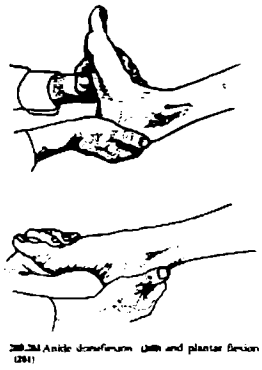
258 Palpation of the anterior joint line of the ankle

259 Pressure posteriorly (right hand), increasing an effusion (palpable with left hand)

(258,259)

Synovitis and effusion are most prominent here because of the slack, extensive anterior capsule: such intracapsular swelling becomes more obvious during passive dorsiflexion of the ankle and if pressure is applied over the posterior capsule by a cupped hand pressing beneath and behind both malleoli (259). Movement: with the knee moderately flexed and gastrocnemius relaxed, support the lower leg with one hand, holding the foot firmly with the

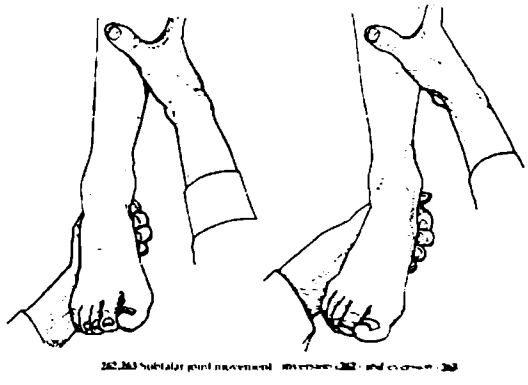
other hand, and passivcly move the ankle into dorsiflexion (about 20 degree; 260) and plantar flexion (about 45 degree; 261).



(260,261)

Subtalar joint

This joint is to deep for palpation and swelling cannot be seen.
Movement: stabilize the distal leg with one hand and, grasping the heel with the other hand, move the foot into inversion (about 30 degree; 262) and eversion (about 20 degree; 263).

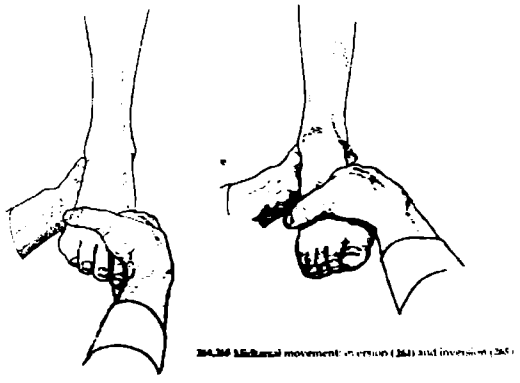


(262,263)

Midtarsal joints

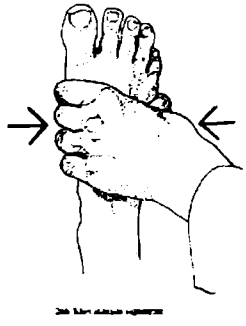
Feel for tenderness and soft tissue swelling over the dorsum of the midfoot. The muscle belly of the extensor digitorom brevis on the lateral aspect of the dorsum may simulate synovial thickening of the ankle or Midtarsal joint: it is distinguished by active toe extension, which shortens, bunches up, and hardens the muscle. Bony swelling over the dorsum occurs with talonavicular osteoarthritis, and with prominence of the talus in pes cavus (sometimes with overlying callus formation).

Movement: stabilize the calcaneus in one hand and, holding the forefoot in the other hand, rotate the foot along its long axis into eversion (about 40 degree; 264) and inversion (about 30 degree; 265).



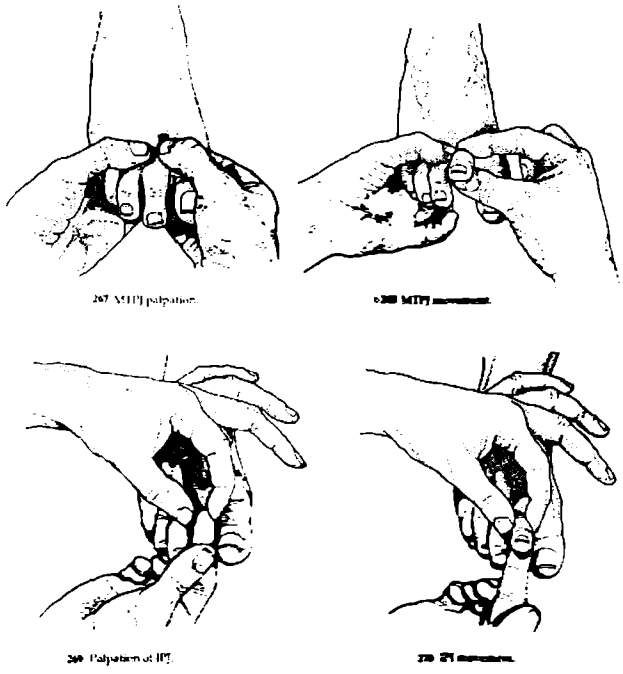
(264,265)

MTPJs. MTPJs tenderness is elicited by squeezing the forefoot laterally across the metatarsal heads (266). If tenderness is elicited the responsible joints are located by palpating each MTPJ in turn, squeezing between both thumbs (on the posterolateral aspect) and forefingers (plantar aspect; 267). Synovitis of MTPJ produces dorsal swelling that comes proximally, filling in the spaces between the metatarsal heads. Tenderness of a single metatarsal head may indicate stress fracture (most commonly the second or third 'march fracture'). Sharply localized tenderness between the third and fourth (less commonly second and third) metatarsal heads is characteristic of Morton's interdigital neuroma: altered sensation may be detected on the lateral and medial aspects of the third and fourth toes, respectively, and rarely a large neuroma may be felt.



(266)

Movement: test each MTPJ by supporting the metatarsal head between the finger and thumb, and moving the proximal phalanx into extension and flexion (268). The first MTPJ has about 80-degree extension and 35 degree flexion; the other MTPJs have about 40 degree of extension and flexion.



(267,268,269,270)

IPJs: Palpate for tenderness of symptomatic or abnormal IPJs by squeezing the posterolateral aspects of each IPJs between the finger and thumb (269). Swelling of synovitis is most prominent on the posterolateral and lateral aspects.
Movement: IPJ are tested by fixing the more proximal and moving the more distal phalanx (270). PIPjs flex to about 50 degree and DIPJs to about 40 degree, with varying extension up to 30 degree.

Periarthicular structures.

Palpate behind and under each malleolus for soft tissue swelling, tenderness and warmth relating to tendon sheaths. Crepitus may be detected, and pain reproduced, by palpating relevant tendon sheath with one hand (271) while the other hand passively moves the forefoot into eversion (stressing the peroneals around the lateral malleolus).
Tenosynovitis overlying the anterior aspect of the ankle is distinguished from ankle synovitis by:

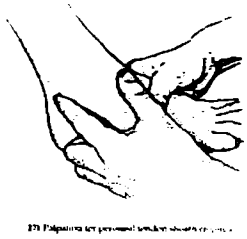
- Its more superficial, linear configuration.
- Tenderness extending well beyond the joint line.
- Pain produced by the appropriate resisted active movement (dorsiflexion of the foot, extension of the hallux and toes).

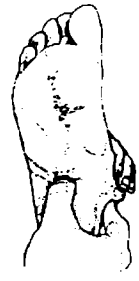
Plantar fasciitis is confirmed by reproducing pain by firm pressure over the midpoint of the heel (272).

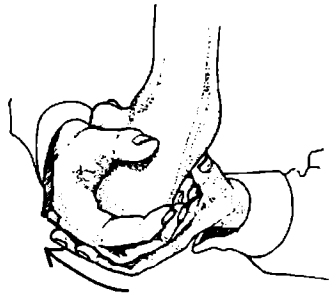
Examination of the posterior heel and Achilles is best undertaken with the patient lying prone, with the feet extending beyond the end of the couch. Palpate between the forefinger and thumb) for tenderness and swelling of:

- The Achilles tendon (Achilles tendinitis, partial rupture).
- The tissues anterior and lateral to the tendon (pre-Achilles bursitis).
- The tendon insertion site into the calcaneus (retro-Achilles bursitis or Achilles enthesopathy).

Then test for resisted active plantar flexion by asking the patient to push their foot down against your hand (273). This may reproduce pain in Achilles tendinitis, enthesopathy, or partial rupture, but not bursitis. In partial rupture, a defect in the tendon, which becomes more noticeable during resisted plantar flexion, may be palpable.







275 Test for lateral instability.

(273,275)

With complete rupture of the tendon, resisted active movement is absent, and firm squeezing of the relaxed gastrocnemius (shortening it) will not produce any passive plantar flexion. Old (healed) partial rupture may leave a palpable nodule in the tendon.

Palpable other nodules (due to generalized disease) to determine attachment to the skin or to underlying structures.

Additional test for stability

Anterior stability

The anterior drawer sign tests the integrity of the talofibular ligament. With the patient sitting, push back on the lower tibia with one hand while pulling the calcaneus and talus anteriorly with the other hand (274). Any movement of the foot relative to the tibia indicates instability.

Lateral instability

This results from anterior talofibular and calcaneofibular ligament damage. Hold the calcaneus in both hands and palpate beneath the lateral malleolus with one thumb (275). Slowly invert the heel, looking for excess movement and the development of a palpable 'gap' beneath the laterally placed thumb. For deltoid ligament insufficiency (an uncommon condition), evert the calcaneus and feel for a gap on the medial side.

SUMMARY OF FOOT EXAMINATION

(1) Inspection of the standing patient
  (a) Swelling (synovitis, tenosynovitis, tendlnitis, bursitis, nodules)
  (b) Deformity (arches, joints)
  (c) Skin, nails
(2) Inspection of the waking patient
(3) Examination of the recumbent patient
  (a) Inspection of soles, interdigital clefts
  (b) Palpation (warmth, swelling, tenderness) and movement (restriction, pain), Crepitus) of joints:
    Ankle (dorsiflexion/plantarflexion)
    Subtalar (inversion/eversion of calcaneus)
    Midtarsal (inversion/eversion of midfoot)
    MTPJs (flexion/extension)
    IPJs (flexion/extension)
  (c) Palpation (warmth, swelling, tenderness) with or without movement (pain, crepitus) of Periarthicular structures:

    Tenosynovitis (extensors, Peroneals, Tibialis posterior)
    Plantar fascia insertion
    Achilles tendon and insertion (patient prone)
    Pre-Achilles, retro-Achilles bursitis (patient prone)
  (d) Tests for stability (anterior, lateral)

suggested Reading : Michael Doherty. John Doherty. Clinical Examination in Rheumatology: 123-136, 1992

FOOT PAIN

Foot pain: common causes

POSTERIOR CALCANEAL PAIN

- Superficial bursitis
- Achilles tendinitis
- Retrocalcaneal bursitis
- Haglund' s deformity

PLANTAR CALCANEAL PAIN

1-Central tenderness
- Fat pad atrophy (primary, secondary to steroid infiltration)
- Obesity

2-Medial tenderness
- Compression neuropathy (calcaneal branches, nerve to abductor digiti quinti)

3-Medial-central tenderness (at medial calcaneal tubercle)
- Plantar fasciitis
- Avulsion fracture
- Fascial microtears

4- lateral compression tenderness
- Calcaneal stress fractures
- Calcaneal cysts

PLANTAR FOREFOOT PAIN

- Morton' s neuroma
- Stress fracture
- Metatarsophalangeal joint arthritis
- Tarsal tunnel syndrome

Ankle sprain

Summary of Diagnosis
- Inversion or eversion ankle injury.
- Pain.
- Swelling developing within a few hours.
- Difficulty bearing weight.
- Inability to bear weight and bone tenderness suggests fracture and dictates the need of confirmatory x-rays (see Ottawa Ankle Rules).

Key Points
- Ankle sprain is the most common athletic injury.
- The main issue is to determine whether a fracture is present.
- Ankle ligamentuos injuries usually result from forced inversion.
- Predisposing factors include, joint laxity, muscle weakness, and previous ankle sprains.
- Ankle sprain should be treated with ice packs during the initial 48 hours plus compression bandage and elevation of the ankle for several days.
- Chronic discomfort occurs usually as a result of a faulty diagnosis and therapy.

Causes of persistent pain after ankle sprain

1- Incomplete rehabilitation, most often after grade 3-ligamentous injury.
2- Intraarticular injuries.
3- Chronic instability, mechanical or functional. Subtalar (talocalcaneal or talonavicular) sprain.
4- Distal tibiofibular syndesmosis pain.

5- Impingement problem. Sinus tarsi syndrome.
6- Chronic tendon disorders.
7- Stress fractures
8- Nerve injuries
9- Reflex sympathetic dystrophy.
10- Tumors.
11-In-children, undetected traumatic epiphyseal injuries.

Posterior calcaneal pain

Summary of Diagnosis

- New shoes, excessive exercise, familial hypercholesterolemia inflammatory-type back pain, gout, RA?
- Was a snap felt?
- Which structure is involved (Achilles tendon, superficial bursa, calcaneus, retrocalcaneal bursa, periosteum)?
- If Achilles tendon, which portion is involved?
- Are nodules present?
- Laboratory studies aimed at proving the likely cause.
- Lateral x-rays in plantar flexion.
- MRI when interstitial tendon rupture is suspected.

Key points

- Superficial bursitis, Haglund's deformity, sports-related trauma, spondyloarthropathy, and a variety of additional causes may cause it.
- Diagnosis is based on a detailed history and physical examination.
- In cases of Achilles tendinitis, etiologies associated with a high probability of rupture should be identified.
- A tender nodule in noninsertional tendinitis indicates interstitial rupture and portends gross tendon rupture.
- MRI is useful to confirm interstitial rupture.
- Surgical tendon release and reinforcement is indicated in cases of impending rupture.

Plantar heel pain

Summary of Diagnosis

- Running history, recent weight gain, inflammatory type low back pain.
- Location of tenderness (central, at medial calcaneal tubercle, at medial border of heel).
- Increase in pain by great toe dorsiflexion (plantar fascia enthesopathy).
- Lateral x-rays of heel to rule out an inflammatory type spur and a calcaneal lesion.

Key points

- It may be caused by fat pad disorders, entrapment neuropathy, enthesopathy (traumatic, spondyloarthropathy related), and heel bone lesions.
- Physical examination is useful in the initial classification of patients.

- Running-related pain is usually caused by deficiencies in training and the use of the wrong shoe.
- Conservative treatment of PHP including rest, a heel cup, and Achilles tendon stretching is successful in approximately 80% of patients.
- Corticosteroid infiltration is only indicated in-patients with spondyloarthropathy with refractory plantar fascia enthesopathy. The procedure is technically difficult and should be performed by an expert.
- A short leg walking cast may be tried instead.
- Surgical procedures should be considered in-patients who fail to improve after 6 months of conservative treatment. Usual procedures include nerve release and plantar fasciotomy.

Tarsal tunnel syndrome

Causes of tarsal tunnel syndrome

1. Idiopathic
2. Tenosynovitis
3. Varices
4. Valgus heel
5. Ganglia
6. Exostosis related to tarsal coalition
7. Trauma (fractures, direct trauma to nerve, adhesions)
8. Melorheostosis
9. Soft tissue and bone tumors
10. Variant muscle bellies

Summary of Diagnosis

- Plantar burning pain and Paresthesias.
- A positive Tinel test behind or just distal to the medial malleolus.
- A valgus heel or a lump (soft tissue, bone) in the medial retromalleolar area may be present.
- Decreased sensory conduction velocity or increased distal latencies to abductor hallucis (medial plantar nerve) or abductor digiti minimi (lateral plantar nerve)

Key Points

- Diagnosis depends on an awareness of the process and the elimination of mimicking conditions: sympathetic dystrophy, l5 or S1 radiculopathy, peripheral neuropathy, and erythromelalgia.
- The triad of burning plantar pain, positive Tinel, and increased latencies on Electrodiagnosis is diagnostic.
- Conservative treatment of tarsal tunnel syndrome has little value.
- Surgical release is effective in 75% to 90% of patients.

Hallux valgus

Summary of Diagnosis

- Inspect feet in the standing position.
- Look for callosities.
- Search for joint hyperlaxity and Achilles tendon contracture.

- Check pulses, reflexes, and sensation.
- Obtain weight-bearing x-rays.
- Determine intermetatarsal and hallux valgus angles, and whether first MTP osteoarthritis is present.

Key Points

- Hallux valgus is the most common condition of the first MTP joint.
- All patients with hallux valgus may be treated conservatively with an extrawide, soft-shoe with insole padding.
- Many patients opt for a surgical correction of the deformity.
- Patients with vascular insufficiency or peripheral neuropathy should not be operated on.
- The choice of the surgical procedure depends on the degree and nature of the deformity.
- Good surgical results are achieved in approximately 75% of cases.

Hallux regidus

Summary of diagnosis

- Recurrent or chronic first MTP pain.
- Bulky joint; reduced dorsiflexion.
- Lateral and AP x-rays.

Key Points

- Hallux regidus affects predominantly young men.
- The joint changes are those of osteoarthritis.
- Osteochondritis or epiphysial dysplasia may be a conditioning factor.
- Periodic flares of the condition superficially resemble acute gout.
- In addition to severe pain and limitation of motion, hallux regidus often results in compression neuropathy of the cutaneous nerve.
- May be effectively treated with wide, extra-depth shoes with a rocker- bottom sole to compensate for the loss of dorsiflexion.
- Surgery is often successful in this lesion.

PATIENT 6. A 32 years old man had a history of multiple episodes of podagra, which had affected either foot, treated with indomethacine or other NSAID and Allopurinol. Attacks had been regularly triggered by athletic efforts. Rather than in the early morning hours or during the night, as seen in gout, pain routinely appeared 1 to 2 hours after excessive activity. Examined during an Asymptomatic period, both first MTPs were bulky and exhibited limited dorsal and plantar flexion. X-rays showed overgrown bone in the superior margin of the joint. There were no erosions. The left first MTP was aspirated, and no crystals were seen in a small amount of fluid withdrawn. The patient was prescribed a rigid insole and was referred to the orthopedic department for possible resection of the superior osteophytes.

Morton's neuroma

Summary of Diagnosis

- *Diagnosis is clinical based on the characteristics of pain and location of tenderness.*
- *X-rays and laboratory studies are normal; they are important to rule out synovitis and stress fracture.*
- *MRI may reveal the mass but should not be requested unless tumoral lesion needs to be ruled out.*

Key Points

- *A frequent cause of forefoot pain, particularly in women. Plantar tenderness is confined to one intermetatarsal space, usually the third.*
- *A wide, soft-shoe relieves pain in many patients.*
- *Corticosteroid infiltration may be very effective.*
- *Surgical removal of the neuroma in refractory cases is successful in over 80% of cases.*

Flat foot

Summary of Diagnosis

- *Fallen longitudinal arch.*
- *Heel inversion maneuver.*
- *Tibial rotation maneuver.*
- *Palpation of posterior tibialis tendon.*
- *Lateral weight-bearing foot x-rays in a rigid flat foot.*
- *Additional imaging procedures (oblique views, CT) to detect tarsal coalition if clinically indicated.*

Key Points

- *A flat foot may be a result of ligament hyperlaxity, Tarsal bone abnormalities, arthritis, neurologic disease, or posterior tibialis tendon rupture.*
- *A painless flexible flat foot does not represent disease.*
- *Excessive activity, trauma, or Achilles tendon contracture may cause pain in a flexible flat foot.*
- *Pain in a rigid flat foot is usually caused by the underlying tarsal bone abnormality.*
- *Posterior tibialis tendon ruptures tend to go undiagnosed; early ruptures can be repaired before irreversible changes set in.*

***Suggested Reading**: Juan j. Canoso. Rheumatology in primary care*

CHAPTER III

CONNECTIVE TISSUE DISEASES

RHEUMATOID ARTHRITIS

Summary of diagnosis

- History and physical examination to seek symmetry and distal involvement and to rule out other rheumatic diseases such as micro-crystalline disease, CTDs, spondyloarthropathy, and certain infections.
- CBC: thrombocytosis is frequent, anemia is rare in early disease; leucocytosis in very acute cases; leukopenia occasionally present in association with splenomegaly.
- ESR, CRP: usually elevated depending on extent and degree of joint inflammation.
- RF positive in 50% at onset and 90% within 2 years
- ANA positive in 30%, low titer, homogeneous pattern

In ANA-positive cases, determine complement and specific lupus antibodies dsDNA and Sm. (in rheumatoid arthritis complement is elevated or normal; dsDNA and Sm. antibodies are negative).

- If a joint effusion is present, aspirate to rule out crystal disease and to confirm aseptic polymorphonuclear inflammation. Infections rarely mimic RA. Depending on circumstances, secondary syphilis, parvovirus B-19 infection, rubella infection, or tuberculosis may have to be ruled out.

RHEUMATOID ARTRITIS

Key Points

- Rheumatoid arthritis is a potentially disabling condition that markedly reduces life expectancy.
- Treatment of RA should be implemented early and be aggressive.
- Physical therapy is an integral component of the treatment program.
- Nonsteroidal anti-inflammatory relieves symptoms but have little effect on disease progression.
- Low-dose Corticosteroid appears to decrease the rate of erosion at a significant metabolic and structural cost.
- Second-line agents have a delayed effect on RA. They consolidate improvement and unusually cause remission.

- Corticosteroid infiltration is useful in joints that lag behind in improvement and to prevent rupture of Baker's cysts.
- Primary care physicians should work closely with rheumatologist for best treatment results.

THE AMERICAN RHEUMATISM ASSOCIATION 1987 RVISED CRTERIA FOR THE CLASSIFICATION OF RHEUMATOID ARTHRITIS

1. Morning stiffness in and around the joints lasting at least 1 hour before maximal improvement
2. Soft tissue swelling or fluid (not bone overgrowth only) in 3 or more joints observed by physician.
3. At least one joint area as above in the PIP, MCP, or wrist joints
4. Symmetric arthritis (involvement of PIP, MCP or MTP joints is acceptable without absolute symmetry)
5. Subcutaneous nodules observed by a physician.
6. Positive test for rheumatoid factor by any test that has been positive in less than 5% of normal controls.
7. Radiographic erosions or Periarthicular osteopenia in hand and wrist X-rays.

To classify a patient as having RA criteria 1 through 4 must have been present for at least 6 weeks.

THE AMERICAN RHEUMTISM ASSOCIATION CRITERIA FOR COMPLETE CLINICAL REMISSION IN RHEUMATOID ARTHRITIS

A minimum of five of the following requirements must be fulfilled for at least 2 consecutive months in a patient with a definite or classic rheumatoid arthritis:

1. Morning stiffness not to exceed 15 minutes.
2. No fatigue
3. No joint pain
4. No joint tenderness or pain on motion
5. No soft tissue swelling in joints or tendon sheaths.
6. Erythrocyte sedimentation rate less than 30 mm/hour (females) or 20 mm/hour(males)

RHEUMATOID ARTHRITIS: SYSTEMIC MANIFESTATIONS

- Systemic RA features one or more of the following: weight loss, high titer rheumatoid factor, rheumatoid nodules, episcleritis, pulmonary interstitial disease, pleuritis, pericarditis, leg ulcers, palpable purpura, periungual skin infarctions, bullous eruption, digital gangrene, and neuropathy.
- Identification of systemic RA is observer dependent. A careful history and physical examination repeated over time should focus on weight, lacrimary secretion, lymph nodes, skin status, chest status, spleen size, muscle strength, tendon reflexes, and skin sensation.
- Compressive complications of RA include crycoarytenoid arthritis, various neuropathies, and medullary compression from atlantoaxial subluxation. Inquire about

voice changes, sore throat, Paresthesias, and cervical pain with radiation to extremities.

- Voice changes and unexplained sore throat call for ENT evaluation.
- Obtain a lateral film of the cervical spine in flexion as a baseline and repeat the study to detect atlantoaxial subluxation if the patient develops neck or occipital pain or neurologic findings. If abnormalities are found, request Neurosurgical consultation.

RHEUMATOID ARTHRITIS: SYSTEMIC MANIFESTATIONS

Key Points

- Weight loss, chronic disease anemia, thrombocytosis, and a high ESR and /or CRP in rheumatoid arthritis indicate a severe, ongoing inflammatory response. The addition of low dose Corticosteroid should be considered in these instances.
- Small-vessel vasculitis improves with a step up of basic treatment plus low- dose Corticosteroid.
- Narcotizing vasculitis, extensive neuropathy, and depressed complement levels call for high dose Corticosteroid and the possible addition of an immunosuppressive.
- Symptomatic atlantoaxial subluxation requires Neurosurgical evaluation.
- In associated sjogren's, replace tears and insist on dental prophylaxis.
- Felty's syndrome often improves with MTX or intra-muscular gold. Leukocyte stimulating factor may help in intercurrent infection. Recurrent infections are an indication for splenectomy.
- Amyloidosis is a rare complication of rheumatoid arthritis in the United States but is relatively frequent in Europe and Japan.
- Pericardial tamponade usually requires a pericardial window.
- Constrictive/effusive pericarditis is treated by pericardiectomy.

PATIENT 7. A 56-year-old man was seen because of disrupting migratory arthritis occurring anywhere between 7 and 20 days apart. Arthritis affected one joint at a time, symptoms were violent, and within 1 to 3 days the episode was over. Multiple subcutaneous nodules were present on his hands. The left knee showed acute inflammation. Synovial fluid aspirated from this knee had 20,000 WBC/mm3, 90% of PMNs, and no crystals on polarizing microscopy. The rheumatoid factor test was markedly positive .The patient started on intramuscular gold unfortunately, shortly after initiation of treatment the extensor tendons for the fifth and fourth right digits ruptured. Tendon transfer was successfully performed. Four months later joint's symptoms had gone away and by 6 month of therapy the nodules were much reduced in size

PATIENT 8.

A woman with long- standing rheumatoid arthritis slowly developed an incomplete fist. On examination, on attempted flexion, several fingers in each hand did not reach the palm. To distinguish flexor tenosinvitis from interphalangial joint arthritis, the examiner gently helped finger flexion. Some of these fingers had complete passive flexion plus subtle diffuse swelling of the flexor tendon sheath. In the others no further flexion occurred and PIP's were tender. Well-targeted steroid injections greatly helped this patient's hand function

Suggested Reading

1. *American College of Rheumatology Ad Hoc Committee on clinical Guidelines for the management of rheumatoid arthritis. Arthritis Rheum 39:713-722, 1996*
2. Harris ED. Clinical features of rheumatoid arthritis. In: Kelly MN, Harris ED Jr, Ruddy S, et al.
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4. Michet CJ Jr, Evans JM, FlemingKC, et al. Common rheumatologic diseases in elderly patients. Mayo Clin Proc 70:1205-1214, 1995.
5. Van Schaardenburg D, Breedveld FC. Elderly-onset rheumatoid arthritis. Sem Arthritis Rheum 23:367-378, 1994.
6. McCArty DJ, O'Duffy JD, Pearson L, etal. Remitting seronegative symmetrical synovitis with pitting edema. JAMA 254:2763-2756, 1985.

ELDERLY- ONSET RHEUMATOID ARTHRITIS

Summary of diagnosis

- Abrupt-onset polyarthritis involving predominantly upper extremity joints.
- Shoulder involvement is common.
- Lower extremity findings may be spared; only 50% of cases have MTP arthritis.
- Fifty percent of cases are rheumatoid factor positive.
- The condition must be distinguished from PMR, RS3PE syndrome, late-onset spondyloarthropathy, crystal synovitis, and malignancy-related arthritis.
- Obtain chest x-rays and check stools for occult blood.
- Joint aspiration is essential to rule out mimicking crystal synovitis.

ELDERLY-ONSET RHEUMATOID ARTHRITIS

Key points

- Seronegative elderly-onset RA tends to have benign course.
- Seropositive cases, however, tend to be progressive and invalidating.
- Seropositive elderly-onset RA should be treated early and aggressively.
- Seronegative cases should clearly distinguished from crystal induced synovitis and malignancy-related arthritis. Ruling out PMR and RS3PE may be difficult if not impossible.

Symptoms and findings of giant cell arteritis should be frequently investigated in-patients with presumed late-onset seronegative RA.

Spontaneous disease remission after one to a few years indicates the condition was PMR or RS3PE, rather than RA.

Suggested reading

Michet CJ Jr, Evans JM, Fleming KC, et al. Common rheumatologic diseases in elderly patients. Myo clin Proc 70:1205-!214, 1995

REMITTING SERONEGATIVE SYMMETRICAL SYNOVITIS WITH PITTING EDEMA (RS3PE)

Summary of diagnosis

- Age over 60.
- Male predominance
- Abrupt onset
- Distal synovitis
- Distal pitting edema
- Consider gout, CPPD, RA, spondyloarthropathy
- Obtain hand x-rays, a pelvic film to rule out sacroiliitis, UA level, RF, and joint aspiration
- Obtain serum protein electrophoresis to rule out multiple myeloma

REMITTING SERONEGATIVE SYMMETRICAL SYNOVITIS WITH PITTING EDEMA (RS3PE)

Key Points

- Accuracy in diagnosis is essential (rule out crystal disease and make an effort to separate RS3PE from late onset RA and PMR).
- Although it was originally described as a self-limited disease transition to RA spondyloarthropathy or connective tissue disease may occur, at least in the French experience.
- The evidence in support of the previous statement is retrospective. Differences in genetic background may also be involved.
- Treatment of RS3PE and late onset RA is similar.
- Response to treatment is excellent, at least in the short term.

Suggested Reading

Juan J. Canoso. Rheumatology in primary care; 1997: 72-73

SLE

Summary of Diagnosis

- Multi-system involvement by history and physical examination
- CBC, platelet count, serum creatinine, ALT, CPK, urinalysis with detailed examination sediment, TSH
- Chest x-rays.
- Consider alternative diagnosis: connective tissue diseases, infection, and neoplasm
- ANA determination on Hep-2 cells (sensitivity).
- Serum C3, C4, CH50; dsDNA, Sm (specificity).

MILD SLE

Key Points

- Probably the most common form of SLE
- Transition to a more serious SLE is more likely to happen in early disease (less than 3 years after diagnosis).

- Treatment should be conservative using sound general advice and low-toxicity medications.
- The combination of nonacetylated salicylates plus hydroxychloroquine is often successful in these patients.
- If low-dose Corticosteroid is used, they should be in addition to, rather than instead of, the previous regime and at each visit dose reduction should at least be considered.
- Calcium and vitamin D supplementation is essential in Corticosteroid-treated patients.
- Corticosteroid reduction should be made by the milligram; this may lead to discontinuation or to the use of the lowest effective dose.

RENAL SLE

Summary of Diagnosis

- Diagnosis of renal SLE rests on an accurate and repeated assessment of urinalysis, particularly a well examined urinary sediment.
- Red cell casts indicate active lupus nephritis.
- Renal function tests lag behind, and tend to underestimate, pathologic changes.
- Beware of decreasing C3 and CH50 and rising titers of dsDNA antibodies: renal disease may be on the horizon.
- Kidney biopsy is useful for: (1) uncovering severe focal Proliferative or diffuse Proliferative GN with only minor changes on urinalysis and a normal renal function, and (2) determining activity index (reversibility) of the renal lesion.

RENAL SLE

Key Points

- Prognosis in SLE largely depends on the presence or absence of severe renal disease
- Absence of nephritis at diagnosis should not give a sense of security; renal disease may ensue on follow-up.
- Histologic transition may occur in 10% to 45% of cases: from non-renal to renal, from class II or III to class IV, and from class V to class IV (ascending severity); and from class IV to class V (descending severity); as a result of Corticosteroid and immunosuppressive treatment.
- Aggressive treatment of lupus nephritis decreases irreversible scarring and delays or halts progression of renal insufficiency.

NEUROPSYCHIATRIC LUPUS

Summary of Diagnosis

- A broad range of central and peripheral nervous system abnormalities may occur in SLE.
- Accuracy in diagnosis, that is, determining that the condition is indeed due to the lupus, is essential for treatment.
- CNS infection must be considered, particularly in immunosuppressed patients.
- Watch for clinical manifestations of an associated antiphospholipid syndrome (livedo reticularis, heart murmur, history of venous or arterial thrombosis, migraines).

- Perform formal neuropsychologic testing in-patients with cognitive dysfunction. Depression, hypothyroidism, and other cases of pseudodementia must be sought in these patients.
- Patients with arterial hypertension, renal failure, or immunosuppressives may have reversible syndrome that comprises headache altered mental functioning, abnormalities in visual perception including cortical blindness and seizures.
- CSF findings (present in 50%) for example, increased protein and slight lymphocytic pleocytosis are nonspecific but speak of active CNS involvement. CSF examination is essential to rule out infection.
- EEG findings (present in 50% to 80%) are diffuse and nonspecific.
- MRI findings (present in 80%) are nonspecific: single or multiple, small or large high-intensity lesions on T2-weighted scans. Some of these lesions are rapidly reversible.
- Cerebral angiography is usually normal.
- Trans-esophageal echocardiography is essential in-patients with possible micro or macro cerebral embolism.

NEUROPSCHIATRIC LUPUS

Key Points

- Neuropsychiatric lupus has diverse locations, pathogenesis and significance.
- Secondary, iatrogenic, and unrelated neurologic processes challenge the clinician's diagnostic skills.
- Once Neuropsychiatric lupus is diagnosed, the next problem is to choose treatment.
- Pharmacologic treatment of psychiatric disease and seizures should not exclude drugs that are known to cause ANA and even SLE
- Recurrent embolic or thrombotic disease in-patients with Libman-Sack's endocarditis or an associated antiphospholipid syndrome may be halted by high grade anticoagulation plus low-dose aspirin.
- The posterior leukoencephalopathy syndrome improves rapidly with blood pressure control.
- Psychosis in a Corticosteroid treated patients may be caused by the steroid or the lupus: double the steroid dose to find out.
- Scar-related dementia unresponsive to Corticosteroid or Immunosuppression
- Once infection, embolic disease, and other non-lupus CNS conditions have been ruled out, Corticosteroid with or without an immunosuppressive may be started.

PLEUROPULMONARY SLE

Summary of Diagnosis

- Lupus pleuritis tends to be bilateral and often associates with pericarditis.
- Spontaneous LE cells may be present in pleural (and pericardial) fluid.
- Chest x-rays may suggest parenchymal, vascular, and neuromuscular lupus involvement.
- Differentiation among early pulmonary hemorrhage, acute lupus pneumonitis, and microbial pneumonia (particularly P.carinii, tubeculous, fungal, and mixed infections) is facilitated by bronchoscopy and BAL.

- Fibrotic and alveolar-type interstitial disease can be distinguished by CT
- Pulmonary hypertension may occur from microvascular disease or major vessel thrombi. Large perfusion defects in ventilation-perfusion scans suggest the latter.
- A precise diagnosis of respiratory muscle weakness requires involved physiologic techniques and often remains unproven.

PLEUROPULMONARY SLE

Key Points

- Varied lesions; sooner or later one or more of them will occur in the majority of patients.
- Lupus pleuritis is highly symptomatic but mild in terms of response to therapy.
- Pulmonary hemorrhage and acute lupus pneumonitis are life-threatening conditions.
- Interstitial disease progresses slowly, is readily detected by auscultation, and alveolar cases (these can be distinguished by CT) are treatable by Corticosteroid.
- Bronchiolitis obliterans with organizing pneumonia is also responsive to Corticosteroid therapy.
- Pulmonary hypertension is rare; it may occur from microvascular occlusive disease or from major artery embolism or thrombosis. The later are amenable to treatment with anticoagulant therapy.
- Neuromuscular respiratory failure is often responsive to high-dose Corticosteroid agents.

DISCOID LE (DLE) AND SUBACUTE CUTANEOUS LE (SCLE)

Summary of Diagnosis

DLE

- Well-demarcated plaque with epidermal atrophy, follicular plugging, telangiectasis, and pigmentary changes.
- Face, scalp, and ears are the most frequent locations.
- Additional below-the-neck lesions in 40%
- ANA positive in 10% of patients
- Skin biopsy is characteristic.

SCLE

- Papulosqamous or polycyclic non-indurated, non-scaring lesions; permanent pigmentary changes are frequent.
- Extensor surface of arms, upper chest, and upper back
- Mild systemic features (may fulfill ARA criteria) in 30% to 50%.
- Anti-SSA (Ro) antibodies present in 70%.
- Skin biopsy is characteristic.

DISCOID LE (DLE) AND SUBACUTE CUTANEOUS LE (SCLE)

Key Points

- Both conditions may be strictly cutaneous or may associate with SLE.
- Alopecia caused by DLE is permanent (scarring alopecia).

- Cases of DLE with positive ANA may progress to SLE, although not necessarily.
- Cases of SCLE that fulfill ACR criteria for DLE tend to have mild, predominantly cutaneous, highly photosensitive disease.
- Male cases of SCLE with extensive Papulosqamous rash are at higher risk of a severe systemic disease.
- Anti malarial agents are effective in the majority of patients with DLE of SCLE.

PREGNANCY AND SLE

Summary of Diagnosis

- Lupus pregnancy is a high-risk pregnancy.
- Expert monitoring of both the lupus and pregnancy is essential to maximize chances of success.
- Determine the presence of anti phospholipid anti bodies and anti-RO (SSA) and anti-La(SSB) antibodies in the mother, and if the latter two are positive, measure their activity against the 52-kd and 48-kd antigens, respectively.
- Lupus findings such as alopecia, facial and palmar erythema, carpal tunnel syndrome, arterial hypertension, anemia, thrombocytopenia, increased ESR, and proteinuria mat also be caused by pregnancy.
- CBC, platelet count, serum creatinine, urinalysis (with a detailed sediment), anti-dsDNA and alternative complement components should be serially followed.
- Fever true rash, oral ulcers, lymphadenopathy, arthritis, serositis, palpable purpura, urine sediment RBC casts, alternative pathway hypocomlementemia, and increased levels of anti-dsDNA antibodies remain valid indicators of lupus activity during pregnancy.

PREGNANCY AND SLE

Key Points

- Mothers with pathogenic anti-Ro/La should be monitored with serial fetal EKG and ultrasound.
- Fetal myocarditis or atrioventricular block is an indication for dexamethasone therapy (dexamethasone crosses the placenta).
- Only drugs with a record of safety for both the mother and the fetus should be used.
- If the patient has inactive lupus at the time of conception, is normotensive, has normal serum creatinine, and has no history of pregnancy loss, the chances of a successful pregnancy are virtually average.

SLE AND FEVER

Summary of Diagnosis

- Fever is a well-known manifestation of active SLE.
- Fever in inactive SLE suggests infection.
- Leukopenia , lymphopenia, and low levels of CRP suggests lupus fever.
- Shaking chills, leucocytosis, and high levels of CRP strongly indicate microbial infection.
- Concurrent lupus activity must be ruled out in overtly infected SLE patients.

- Corticosteroid-treated patients may be unable to mount a febrile response.
- Fever during immunosuppressive therapy suggests an opportunistic infection.

SLE AND FEVER

Key Points

- Infection is the leading cause of death in early and late SLE.
- Infections may trigger lupus activity and active lupus fosters infections.
- Extricating lupus activity from infection may be a difficult task: don't hesitate to onsult with a rheumatologist and an infectious specialist when an SLE patient has fever.
- Controlling lupus activity is critical in achieving control of infection.

PATIENT 9. A 43-year-old woman had had a rocky course with her lupus. A year before the current admission she had been brought to the ER with acute right upper quadrant abdominal pain, nausea and vomiting. She was admitted with a diagnosis of cholecystitis, but surgeons decided to hydrate and watch her overnight in the medical service. Interestingly, the next morning the team's attending noticed that her neck veins were markedly distended, there was hepatojugular reflux, and the kussmaul sign was markedly positive. She ended up having pericardial aspiration, which improved her immediately, rather than cholecystectomy. This time she came in with severe Arthralgias, febrile for 2 to 4 days, and with a prominent facial rash, especially over the bridge of the nose. Respiratory sounds were muffled at the bases. The WBC was 18,000/mm3 with 80% polys and 10% bands. Urinalysis showed 2+ protein and leukocytes and the granular casts in the sediment. The ESR was 90. Dermatologist consultant felt that the facial rash was erysipelas. Penicillin was administered, and one day later, without modifying the 10mg prednisone dose plus hydroxychloroquine 200-mg she was taking, the rash appeared inactive. Serum complement levels came- back very low, and the anti-native DNA was elevated. Upon discharge the hydroxychloroquine dose was doubled, and she did well.

DRUG-INDUCED SLE

Summary of diagnosis

- Positive ANAs is very common with certain medications (procainamide, quinidine, phenothiazines).
- The pattern of nuclear fluorescence is homogeneous, and antibodies are specific for histone.
- clinical manifestations include Arthralgias or arthritis, pleuritis, nonspecific rashes, and leukopenia.
- The required SLE classification criteria are usually not met.
- Nephritis or a positive anti-Sm or anti-dsDNA antibody tests are indicative of idiophatic lupus.

DRUG INDUCED SLE

Key Points

- Drug-induced ANA is much more frequent than clinical SLE.
- The condition is mild in most patients.
- Pericardial tamponade may occur.
- Clinical findings resolve within weeks of discontinuing the offending drug.
- Resolution of the ANA takes 6 months to 2 years.
- If drug discontinuation is not possible, cases may be treated like mild SLE.
- Drugs known to induce ANA/SLE are not contraindicated in spontaneous SLE.

PATIENT 10. A 74-year-old woman was sent for treatment advice for poorly responsive case of elderly-onset SLE. One year before the patient had developed symmetric arthritis in the distal extremities. Her response to choline magnesium salicylate and hydroxychloroquine was poor. She began to lose weight, became depressed, and her ability to manage by herself at home was questioned. Four months before her referral a severe bout of pleuropericarditis, initially thought to be an MI, put her in the hospital. Laboratory studies revealed leukopenia, an ANA on Hep-2 cells of 1:640 (N < 1:80) homogeneous pattern, low c3, and positive IgM anticardiolipin antibodies. Tests for anti-double-stranded DNA and Sm were negative. Her kidney was spared. She now was taking prednisone 20mg per day that could not be decreased because the arthritis flared. On taking the history it surfaced that she had been taking quinidine for the past 3 years for the prophylaxis of arrhythmia. The issue of a possible quinidine-induced SLE was raised with her physician and a decision was made to discontinue the quinidine (which the patient did not really need). Slowly, over the course of 3 months, all clinical findings went away. The ANA a year later was 1:40.

PRIMARY ANTIPHOSPHOLIPID ANTIBODY SYNDROME

CATASTROPHIC ANTIPHOSPHOLIPID SYNDROME

Skin involvement: acrocyanosis, levido reticularis, digital gangrene and ulcers
Cardiovascular involvement: Tachycardia, malignant hypertension, acute CCF, small vessel occlusive disease
Respiratory system: Tachypnea, ARDS
CNS involvement: Focal signs (CVA, mononeuritis multiplex), Epileptiform seizures (grand mal, status epilepticus), General (behavior disturbances, emotional lability, confusion, stupor)
Renal involvement: renal microthrombi, renal failure
Organ infarction: adrenal, hepatic, bowel

PRIMARY ANTIPHOSPHOLIPID ANTIBODY SYNDROME

Summary of Diagnosis

- Consistent clinical findings: recurrent venous or arterial thrombosis, or recurrent mid-trimester abortions.
- A high-titer IgG or IgM anticardiolipin antibody test by ELISA, or a positive test for the lupus anticoagulant (dilute APTT, kaolin clotting time test, or Russell viper venom time test).
- Absence of SLE by clinical criteria (a low-titer ANA is present in 30% of PAPS patients).

- Cardiac echo to determine if vegetations are present.
- A high-titer anticardiolipin test or lupus anticoagulant test, in the absence of the clinical correlates does not mean PAPS or SAPS.

Criteria for clssification of the antiphospholipid syndrome

Clinical	Laboratory
Venous thrombosis	IgG anticardiolipin antibodies (moderate to high levels)
Arterial thrombosis	IgM anticardiolipin antibodies (moderate to high levels)
Recurrent fetal loss	Positive lupus anticoagulant test result
Thrombocytopenia	

Patients with the syndrome should have at least one clinical finding plus one laboratory finding during the course of disease. The antiphospholipid antibody test must be positive on at least two occasions more than 3 months apart.

PRIMARY AND SECONDARY ANTIPHOSPHOLIPID SYNDROME

Key Points

- A positive anticardiolipin or lupus anticoagulant test is insufficient to diagnose PAPS or SAPS. Clinical finding such as recurrent venous or arterial thrombosis, or fetal losses must be present for diagnosis.
- However, a strongly positive anticardiolipin test per se is associated with a twofold increase in fetal loss.
- Thrombosis prevention in PAPS requires high-dose coumadinization (INR about- 3).
- Moderate heparinization plus low-dose ASA decrease the rate of fetal loss in-patients with PAPS.

SJOGREN'S SYNDROME

Summary of Diagnosis

- Sicca syndrome (dry eyes, dry mouth)
- A positive Schirmer's test or punctate and filamentous keratitis as shown by rose Bengal dye and biomicroscopic examination.
- Minor salivary gland biopsy is confirmatory of primary Sjogren's syndrome. Cases of plain KCS feature minimal salivary gland changes with a score less than 1.
- chest x-rays to rule out pseudolymphoma and interstitial pneumonitis.
- CBC, ESR, serum creatinine, AST, ALT, CPK, aldolase, alkaline phosphatase, GGT, urinalysis, TSH, ANA, RF, SSA, SSB (anti-DNA and anti-Sm if ANA is positive), serum protein electrophoresis, and quantitated IgG and IgM (serum immuonoelectrophoresis if an "M" component appears to be present).

SJOGREN'S SYNDROME

Key Points

- Primary Sjogren's syndrome is more severe than secondary sjogren's in terms of salivary and lymphoid tissue growth, visceral involvement, vasculitic complications, and the development of pseudolymphoma and lymphoma.

- Autoimmune thyroid disease is quite common in primary Sjogren's syndrome. Secondary sjogren's is often overlooked; it is present in approximately 30% of patients with RA, SLE, and scleroderma. Diagnosis depends on asking the right questions.
- Hepatitis C virus infection and HIV infection may closely resemble Sjogren's.
- Treatment of Sjogren's includes the local care of dry eyes and mucosae, prophylaxis and treatment of secondary Suppurative and fungal infection, and the use of systemic measures, in particular hydroxychloroquine.

Suggested Reading

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POLYMYOSITIS

Summary of Diagnosis

- History of muscle weakness, coarse skin on hands, Raynaud's, upper dysphagia, regurgitation, palpitations, dyspnea, and Arthralgias.
- Physical examination showing proximal muscle weakness, mechanic's hands, crackling rales, and synovitis.
- Request alkaline phosphatase, AST, ALT, CPK, aldolase; ANA; anti Jo-1.
- Request TSH, K, Ca, and Mg.
- Chest x-rays, PFT's with diffusive capacity.
- Barium swallow to detect upper esophageal dysfunction.

- EMG on one side to confirm myopathic changes, determines abnormal muscle, and excludes myasthenia gravis and Eaton-Lambert's syndrome.
- Document myositis by contralateral muscle biopsy in a muscle homologous to the one showing EMG changes.

POLYMYOSITIS

Key points

- Polymyositis occurs in several clinical patterns that range from acute rabdomyolysis to a chronic, indolent disease with wasting and contracture.
- Complications of PM include acute renal failure from rabdomyolysis; respiratory failure (from PM-related respiratory muscle weakness, pneumonitis, or aspiration; MTX pneumonitis; or opportunistic infection); and myocarditis.
- Treatment of Polymyositis is with high-dose Corticosteroid plus, in severe cases an immunosuppressant drug.
- Physical therapy is an integral component of the treatment program.
- Enzyme normalization occurs within a few weeks of treatment; strength improvement lags behind a few weeks to 6 months.
- There is no association between PM and cancer (other than detection bias).
- Long-term course of PM depends on the clinical form. In general the condition tends to recur following interruption of therapy.

PATIENT 11. A 65 year-old man was referred for evaluation of Arthralgias, general debilitation, and marked elevation of serum ALT and AST. Autoimmune hepatitis and mixed cryoglobulinemia had been diagnostic considerations. On examination skin was normal and liver and spleen were not palpable. Neck and hip flexors were weak with relative sparing of distal strength. Pronounced muscle weakness prevented the patient from rising unaided from the examining table. Myositis was suspected CPK and aldolase determinations were requested. Both were elevated about 50 fold and alkaline phosphatase was normal. Muscle biopsy confirmed the diagnosis of Polymyositis, and dramatic response to high-dose Corticosteroid followed.

DERMATOMYOSITIS

Summary of diagnosis

- Childhood and adulthood condition.
- History of proximal muscle weakness.
- Dermal changes including face, neck, and chest erythema; Heliotrope hue in the upper lids; Gottron's papules; and scaly, pseudopsoriasis form acral lesions.
- Patients may have fever, polyarthralgias, Raynaud's, upper esophageal dysfunction, pneumonitis, and carditis
- Increased muscle enzymes in most patients.
- [illegible] EMG in most patients.
- Consider an associated neoplasm in-patients in their late middle age (50 or older), particularly in the absence of overlapping features with connective tissue disease and when muscle enzymes are near normal.

DERMATOMYOSITIS

Key points

- The rash of dermatomyositis is unmistakable and represents, without question, the most helpful clue to diagnosis.
- Most patients with DM have clinical and/or biochemical evidence of muscle inflammation. However, some patients don't (amyophaticDM).
- Cases of DM beginning in late middle age should be suspected of harboring neoplasm.
- Neoplasm in DM is usually detectable on routine medical follow-up.
- Treatment of DM is similar to PM, plus the special care required by the dermal component.

INCLUSION BODY MYOSITIES AND IDIOPATHIC INFLAMMATORY MYOPATHY MIMICKERS

Summary of diagnosis

- IBM should be suspected (1) when myopathic findings involve both proximal and distal muscles, is spotty, or is asymmetric, (2) when tendon reflexes are lost and (3) in steroid-resistant PM.
- A thorough review of muscle biopsy reveals the diagnostic features of IBM.
- Several conditions may mimic idiopathic inflammatory myopathy, in particular muscle dystrophy, paroxysmal neuropathies (diabetes), neuromuscular transmission disorders, infection (trichinosis, HIV), alcoholic myopathy, enzyme errors of glycogen metabolism (weakness and cramps with exercising), hypothyroidism, and hypophosphatemia.
- Make sure to include CBC with differential count, TSH, K, Ca, and P determination in-patients with presumed idiopathic inflammatory myopathy.

INCLUSION BODY MYOSITIS AND IDIOPATHIC INFLAMATORY MYOPATHY MIMICKERS

Key points

- There are myriad myopathies beyond the familiar PM and DM.
- Inclusion body myositis should be looked for in PM cases that fail to respond to standard Corticosteroid doses.
- Consider other types of muscle injury from metabolic errors, through trichinosis, through hypophosphatemia, when assessing patients with myopathic weakness.
- Don't hesitate to enlist rheumatologist or neurologist in the evaluation of patients with proximal muscle weakness.

Suggested Reading

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SYSTEMIC SCLEROSIS

Summary of diagnosis

- Raynaud's phenomenon
- Skin fibrosis, diffuse or limited
- Dysphagia
- Arthralgias or arthritis
- Inspect nail fold capillaries with an ophtalmoscope or a wide-angle microscope.
- Obtain chest x-rays.
- Determine CBC, platelet count, reticulocyte count, BUN, creatinine, CPK, aldolase, and urinalysis.
- If limited systemic sclerosis, determine anticentromeric antibodies for diagnostic support and antimitocondrial antibodies to detect early biliary cirrhosis.
- If diffuse systemic sclerosis, determine antitopoisomerase-1 antibodies, which are confirmatory if present.

SYSTEMIC SCLEROSIS

Key points

- Edematous hands plus Raynaud's are featured by early systemic sclerosis, overlap syndromes, undifferentiated connective tissue disease, and mixed connective tissue disease.
- Limited systemic sclerosis associates with pulmonary arterial hypertension, and diffuse systemic sclerosis with the scleroderma crisis (hypertension and rapidly progressive renal insufficiency).
- Risk factors for scleroderma crisis include early rapidly evolving diffuse disease; tendon friction rubs; cold exposure, and intermediate or high doses of glucocorticoids.
- Localized lesions such as morphea and linear scleroderma primarily represent a cosmetic problem. They are unrelated to systemic sclerosis.
- Treatment of systemic sclerosis includes vasodilators, D-penicillamine to decrease collagen accretion, and measures aimed at preventing or improving organ damage.
- Prophylaxis of reflux esophagitis is essential in these patients.

SYNDROMES, AND UNDIFFRENTIATED CONNECTIVE TISSUE DISEASE MIXED CONNECTIVE TISSUE DISEASE (MCTD), RHUPUS, OTHER OVERLAP (UCTD):

Summary of Diagnosis

- Diagnosis of MCTD is suspected clinically (overlap of features of SLE, scleroderma, and myositis).
- Diagnosis of MCTD is confirmed by high-titer RNP antibodies and absence of other autoantibodies.
- Diagnosis of rhupus is suspected clinically by rheumatoid like arthritis plus clinical features of lupus and confirmed by the presence radiographic erosions, positive rheumatoid factor, and positive ANA.
- Other overlaps include features of RA/scleroderma/myositis, and scleroderma/myositis.

- UCTD is suspected clinically (absence of cutaneous sclerosis), but diagnosis requires ruling out visceral manifestations of scleroderma.

MIXED CONNECTIVE TISSUE DISEASE (MCTD), RHUPUS, OTHER OVERLAP SYNDROMES, AND UNDIFFRENCIATED CONNECTIVE TISSUE DISEASE (UCTD)

Key points

- Connective tissue diseases are classified according to clinical and laboratory findings rather than etiology.
- Thus, it is not surprising that serologically defined syndromes have clinical overlaps and clinically defined syndromes have serologic overlaps.
- MCTD, is for the most part, a transition-type condition that eventually evolves into scleroderma or SLE.
- While it stays as MCTD, peculiar complications may occur including pulmonary arterial hypertension and trigeminal neuritis.
- The course of rhupus and other overlaps is less well defined.
- UCTD is not a condition with features of two or more conditions, but a condition with features of less than one, scleroderma.

RAYNAUD'S DISEASE AND SECONDARY RAYNAUD'S PHENOMENON

Summary of diagnosis

- Episodic blanching of digits in response to cold and emotion.
- Symmetric, multiple fingers, multiple episodes a day, onset at menarche: Raynaud's disease.
- Unilateral onset, few fingers, persistent episodes, and onset in adulthood: Raynaud's syndrome.
- Unilateral, adult patient, and use of a vibrating tool: vibration white finger.
- In Raynaud's syndrome do a historic review of possible causes.
- In all patients, inspect nail fold capillaries and obtain ANA. If both are negative, scleroderma is most unlikely.
- Follow the undiagnosed rheumatic syndrome patient at semiannual intervals.

RAYNAUD'S DISEASE AND SECONDARY RAYNAUD'S PHENOMENON:

Key points

- Raynaud's disease is a benign condition that often dose not require specific therapy.
- Work-up in Raymond's disease should be limited to obtaining an ANA and inspecting nail fold capillaries with an ophtalmoscope or wide-angle microscope.
- Secondary Raynaud's phenomenon is an important red flag for a number of conditions that may be in need of treatment; most of these conditions can be diagnosed by a careful history. Laboratory evaluation should be based on the clinical leads (don't do a blind work-up!).

- Treatment of Raynaud's includes stopping smoking, dressing warmly, and if required, the use of vasodilator measures ranging from biofeedback to calcium channel blockers to digital or cervical sympathectomy.

EOSINOPHILIC FASCIITIS AND EOSINOPHILIC-MYALGIA SYNDROME (EMS)

Summary of diagnosis

- Eosinophilic fasciitis presents abruptly, often after undue exercises, with myalgias and nonpitting edema of the extremities and sometimes the trunk.
- In EMS there is an initial nonspecific febrile phase prior to developing skin findings similar to eosinophilic fasciitis.
- The main lead to diagnosis is peripheral eosinophilia.
- A full thickness biopsy including skin, subcutaneous tissue, fascia and muscle is key to the diagnosis of Eosinophilic fasciitis.

EOSINOPHILIC FASCIITIS AND EOSINOPHILIC-MYALGIAS SYNDROME (EMS)

Key points

- There is a close resemblance between Eosinophilic fasciitis and the late stages of the EMS and toxic oil syndrome.
- No new cases of EMS have been reported since L-tryptophan was removed from the market in 1989.
- No new cases of toxic oil syndrome occurred after the Tained rapeseed oil was removed from the Spanish market in 1981.
- Eosinophilic fasciitis is clinically characterized by painful woody induration of the limbs. Fingers are spared, superficial skin is normal and the pathognomonic "groove sign" is present.
- Eosinophilic fasciitis arrest spontaneously in approximately 3 years; residual contractures are common.
- Treatment of Eosinophilic fasciitis is empirical. Intermediate-dose prednisone plus cimetidine and also hydroxychloroquine have been used with apparent success.
- Aplastic anemia or myelodysplastic syndromes complicate approximately 5% of cases of Eosinophilic fasciitis.

ADJUVANT DISEASE OR CONNECTIVE TISSUE DISEASE (CTD) FOLLOWING BREAST IMPLANTS

Summary of diagnosis

- The condition is diagnosed clinically by (1) the presence of breast implants, and (2) of appearance of muscoloskeletal symptoms, neurologic symptoms, or both in combination.
- Laboratory studies are largely negative including ESR.
- Antinuclear antibodies are demonstrable in 20% to 30% of patients, frequently with a nuclear pattern (unconfirmed in some studies).

ADJUVANT DISEASE OR CONNECTIVE TISSUE DISEASE (CTD) FOLLOWING BREAST IMPLANTS

Key points

- An association of connective tissue diseases such as scleroderma and SLE has not been shown in large, well-conducted epidemiologic studies.
- Fibromyalgia or chronic fatigue syndrome symptoms in implant recipients most likely represent the background in the general population, plus the possibility that patients with the psychologic makeup of fibromyalgia and chronic fatigue syndrome may be more likely to seek a breast implant.
- A recent cohort study has shown a slightly increased relative risk of "other- connective tissue disease" in breast implant recipients. However, some of the data were collected after publicity regarding the possible health hazards of breast implants had surfaced.
- Removal of the implant has resulted in symptom improvement in only a minority of patients.
- The validity of the concept of silicone-induced adjuvant disease has not been scientifically proven.

Suggested reading

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VASCULITIDES

CLINICAL FEATURES THAT SUGEST THE POSSIBILITY OF SYSTEMIC VASCULITIS

Constitutional features
Musculoskeletal features
Skin lesions
Neuropathy: Mononeuritis (multiplex), sensory peripheral neuropathy
Respiratory tract: alveolitis, pulmonary hemorrhage, infiltrates, nodules, asthma sinositis
Kidny: hypertension, abnormal renal sediment, proteinuria, necrotising glomerulonephritis
Gastrointestinal involvement: diarrhea, nausea and vomiting, abdominal pain,hemorrhage, elevated liver enzymes
Claudication: jaw or extrimity
Central nervous system: headache, visual changes, other cranial neuropathy, stroke, seizures, altered conscious ness or altered mentation

HYPERSENSITIVITY VASCULITIS

Summary of Diagnosis

- Palpable purpura, often fever, and Arthralgias, Rarely hepatitis, nephritis, carditis, and pneumonitis
- Review medications and recent history of vaccination.
- Consider infection (fever, heart murmur, presence of pustules or extensive skin hemorrhage, exposure to hepatitis, blood transfusion, parentral drug use, and other risk factors for HIV).
- Consider urticarial vasculitis (chronic urticaria, angioneurotic edema, Arthralgias) and cryoglobulinemia (history of hepatitis, abnormal liver function tests, high-titer RF).
- Consider SLE and Sjogren's (appropriate questions).
- Consider malignancy (weight loss and other findings).
- Obtain skin biopsy and additional studies as clinically indicated.

HYPERSENSITIVITY VACULITIS

Key Points

- The most common of the vasculitides
- Palpable purpura is the characteristic findings on examination.
- It may be drug or vaccine induced or secondary to a variety of conditions, particularly infections or neoplasm.
- Course may be a single crop of lesions, recurring crops, or chronic disease.
- Visceral involvement in drug-related cases is both uncommon and mild.
- Mild drug-induced HV is treated with removal of the offending drug. Extensive lesions, severe systemic symptoms, and visceral involvement are indications for the use of Corticosteroid.
- Treatment of the underlying condition leads to resolution in secondary HV.

CHRONIC URTICARIA AND URTICARIAL VASCULITIS

Summary of Diagnosis

- Both conditions feature urticarial lesions and angioedema.
- In cases without urticaria, hereditary (autosomal dominant) deficiency of C1 esterase inhibitor must be ruled out.
- Individual urticarial lesions last longer in urticarial vasculitis.
- Variants of chronic urticaria include, cold-induced urticaria, solar urticaria and cholinergic urticaria in which lesions appear, for instance, during exercise or taking a hot shower.
- Fever, Arthralgias, abdominal pain, purpura glomerulonephritis, increased ESR, and depressed complement are features of urticarial vasculitis.
- Differential diagnosis of urticarial vasculitis includes SLE and Sjogren's syndrome.
- Laboratory evaluation should include CBC, ESR, ANA, complement level, C3, C4, and urinalysis. A low C1q and presence of C1q antibodies are diagnostic of urticarial vasculitis.
- Presence of peripheral blood eosinophilia suggests parasitic disease.
- Diagnosis of urticarial vasculitis should be confirmed with a skin biopsy obtained at the edge of the lesion.

CHRONIC URTICARIA AND URTICARIAL VASCULITIS

Key Points

- Urticarial lesions and angioedema are featured by both conditions. When only angioedema is present hereditary angioedema must be ruled out.
- Urticarial vasculitis has distinctive systemic features: ocular inflammation, Arthralgias or arthritis, obstructive pulmonary disease, and glomerulonephritis.
- Peripheral blood eosinophilia suggests parasitic disease. Examine stools for ova and parasites.
- Treatment in both conditions begins with the use of H1 and H2 blockers.
- Patients who fail to improve should be treated with a short Corticosteroid course.
- Resistant cases should be referred to an allergist.

HENOCH-SCHONLEIN PURPURA

Summary of Diagnosis

- Antecedent URI in 50%
- Palpable purpura
- Arthralgias or arthritis involving knees and ankles
- Scalp or other edema
- Abdominal pain
- Blood in stool
- Hematuria, proteinuria
- Skin biopsy showing leukocytoclastic vasculitis with IgA, C3, and properdine deposits in the vessel wall.
- Most frequent in children.

- Small-vessel leukocytoclastic vasculitis involving skin, joints, gut, and kidney.
- Lesional deposition of IgA, C3, and properdine
- Complications include intussusception and progressive renal disease (2% to 4% of pediatric patients; a higher proportion of adult cases).

HENOCH-SCHONLEIN PURPURA

Key Points

- Poor prognostic factors include melena, persisting rash, heavy proteinuria, renal insufficiency, and more than 50% crescents on renal biopsy.
- Corticosteroid and immunosuppressives should be considered when poor prognostic factors are present.
- Intravenous immunoglubulins appear to halt disease progression in severe HSP renal disease.

THROMBOANGIITIS OBLITERANS (TAO)

Summary of Diagnosis

- A condition confined to young tobacco smokers.
- Embolic disease, other vasculitides, scleroderma, and thrombotic conditions such as the primary antiphospholipid syndrome must be ruled out.
- Distal ischemic ulcers and intermittent claudication in the upper and/ or lower extremities; Raynaud's phenomenon; thrombophlebitis
- Distal arterial occlusions with normal proximal pulses
- The Allen's test is usually positive
- Pulse volume recordings document distal disease
- Arteriography shows occlusion of infrapopliteal arteries or arteries distal to the elbow and normal proximal arteries. Sites of occlusion are multiple with corkscrew collateral around the occlusions.
- Histologic diagnosis should be an exception (as it is usually performed in amputated specimens).

THROBOANGIITIS OBLITERANS (TAO)

Key points

- Cessation of smoking is essential to hult the disease.
- Iloprost infusions promote ulcer healing and reduce rest pain.
- Sympathectomy may be used for the treatment of nonhealing ulcers.
- Bypass surgery is not applicable in this condition.

PATIENT 12. A 51-year-old woman, a heavy smoker, was seen in consultation for a possible giant cell vasculitis involving the popliteal arteries. During a cruise 8 months before, left calf claudication and permanent numbness of the great toe developed. Four months later similar symptoms appeared in the right leg. There had been wide fluctuations in ischemic pain. Peaks, such as after a brisk walk, had been consistently associated with watery diarrhea and rectal pain. Recently, ischemic pain was triggered by minimal efforts and the pulp of all left toes became hyperaestetic. There was no fever, but a significant weight loss had been noticed, recovering partially during the last month. Of six physicians who had examined her

during her illness, two including ship physician, had found missing pulses in the left foot; four had stated that pulses were normal. Upon further questioning she denied polymyalgic symptoms. She had had headaches, but they were no different from the usual ones she had experienced for the past 20 years. There was no upper extremity claudication, history of Raynaud's, or other features of connective tissue disease. Admission temperature was 36.6c and BP was 120/80. There were no ulcers or other ischemic lesions. Temporal arteries were normal on palpation. The Allen's test revealed excellent radial and ulnar artery flow in both hands. Pulses were missing in both lower extremities below femoral. An arteriogram obtained the morning of admission showed occlusion of popliteal artery on the left with some irregular collateral circulation and marked narrowing on the same artery on the right. Laboratory results includes Hct 38,WBC 7400 with normal differential, platelet count 244,000, and ESR 23. Serum creatinine, alkaline phosphatase, ALT, AST, and urinalysis were all normal. As a more detailed history of the headaches was obtained, however, it turned out that the patient had been taking an average of 15mg of ergotamine tartrate per week for the previous 5 years She has informed of the strong possibility that her arterial disease was due to egotism plus nicotine use. Late on the day of admission, pulses were felt in the right foot, and by the next morning, popliteal and pedal pulses had appeared on the left. Numbness resolved within days. On a 4 month follow-up she has been entirely Asymptomatic.

POLYARTERITIS NODOSA

Summary of diagnosis

- Clinical cluster with malaise, fever, weight loss, abdominal pain, mononeuritis or mononeuritis multiplex, arterial hypertension, abnormalities in urinalysis.
- Blood cultures to exclude bacterial endocarditis.
- CBC, platelet count, ESR, BUN, creatinine determination, and urinalysis
- Hepatitis B antigen and antibody and hepatitis C antibody
- Biopsy in an involved site (skin, sural nerve, muscle, kidney, testes)
- Angiography to visualize kidney vessels and mesenteric angiography when there is evidence of intraabdominal involvement. Characteristic features include saccular or fusiform aneurysms and tapering of arteries.

POLYARTERITIS NODOSA

Key points

- Febrile, catabolic multisystem disease caused by necrotizing vasculitis of medium-sized and small muscular arteries and smaller vessels.
- In at least 60% of cases there is evidence of hepatitis B or C infection.
- Peripheral nerve involvement causes stepwise incapacitation.
- Catastrophic events are associated with renal, cerebral, cardiac, and mesenteric vessels.
- Treatment is with high-dose corticosteroids plus cyclophosphamide in uncontrolled cases.
- Survival at 5 years has improved from 20% to 80%.
- Poor prognostic factors include renal involvement and cardiac involvement.
- Neurologic sequel and residual arterial hypertension are common in successfully treated patients.

CHURG-STRAUSS SYNDROME

Summary of diagnosis

- Recent onset asthma in an adult
- Intense peripheral blood eosinophilia
- Pulmonary infiltrates
- Neuropathy
- Skin lesions.
- Biopsy of skin lesions, sural nerve, and lung

CHURG-STRAUSS SYNDROME

Key points

- Asthma in the adult followed by systemic vasculitis.
- Systemic involvement include pulmonary infiltrates, upper respiratory lesions, mononeuritis multiplex or other neuropathy, skin lesions, and myopericardial lesions.
- Renal involvement, hypertension, and GI vasculitis are rare in GSS.
- Response to corticosteroids is excellent with virtually all cases undergoing remission.
- **Remission** is permanent in about 50%.

WEGENER'S GRANOLOMATOSIS

Summary of diagnosis

- Upper airway **lesions (rhinitis,** sinusitis, and otitis media)
- Ocular lesions (**sclera, uveal tract, and orbit**)
- Mouth **ulcers, skin nodules or ulcers, arthritis**
- **Chest x-ray abnormalities (nodules, pneumonitis, with or without cavitation)**
- Urine sediment abnormalities; progression to severe glomerulonephritis
- Anemia, leucocytosis, thrombocytosis
- Low-titer positive rheumatoid factor in over 50% of patients
- C-ANCA positively.

WEGENER'S GRANOLOMATOSIS

Key points

- Upper and lower airway necrotizing granulomas, necrotizing and granulomatous vasculitis, and focal segmental necrotizing glomerulonephritis with progression to diffuse, crescentic disease characterizes the condition.
- Treatment of severe cases is with cyclophosphamide and glucocorticoids. Methotrexate may be substituted for the cyclophosphamide in mild or limited cases or when there is intolerance to cyclophosphamide. Trimethoprim/sulphamethoxazole may be effective in limited disease.
- Complete remission is seen about 75% of cases.
- Relapses are frequent and usually involve previously affected areas.
- Trimethoprim/sulphamethoxazole is effective in preventing relapses in-patients with WG in remission.

GIANT CELL ARTERITIS

Summary of diagnosis

- GCA should be considered any time elderly patients have transient or permanent visual loss; new headache, facial pain, occipital pain, chewing pain, unexplained gum pain, or tongue pain/parestesia; unexplained malaise, weight loss, or anemia; PMR; or limb claudication.
- A tender, thickened temporal artery is characteristic of the condition.
- ESR and/or CRP are increased.
- leucocytosis and thrombocytosis are frequent.
- Diagnosis should be confirmed by temporal artery biopsy.
- A positive biopsy shows chronic granulomatous inflammation and giant cell formation near the elastic lamina (50%) or a panarteritis with a predominantly limphomononuclear infiltrate (50%).

GIANT CELL ARTERITIS

Key points

- GCA affects predominantly ascending branches of the aorta, particularly the external carotid, but may involve major arteries elsewhere and even the aorta.
- Clinical manifestations include a febrile and catabolic syndrome, cephalic, facial, or visual symptoms; and limb claudication.
- Some patients present with just malaise, fever, weight loops, and anemia.
- Visual loss is the most serious complication of GCA.
- An early diagnosis (prior to catastrophe) requires that the possibility of GCA be raised in the clinician's mind.
- Treatment of GCA is with high-dose corticosteroids for at least one month followed by a taper over 1-3 years.
- A positive temporal artery biopsy helps withstand unavoidable Corticosteroid side effects.
- Parameters to follow include symptoms, hematocrit, platelet count, and ESR.
- Normal ESR values increase with age.
- Life expectancy in GCA is the same as that of the general population.

TAKAYASU ARTERITIS

Summary of diagnosis

- Young age of the patient, usually a woman
- Systemic symptoms including malaise, fever, and Arthralgias in early disease
- Symptoms and findings of major artery insufficiency
- BP difference between upper extremities more than 10 mm Hg
- Bruits over subclavian(s) or abdominal aorta.
- ESR elevated in early disease and in phases of active arteritis; ESR normal in quiescent disease.
- Arteriography allows both diagnosis and classification. Findings include [illegible] major branch narrowing, poststenotic dilatation, saccular aneurysms, and occlusion.

TAKAYASU ARTERITIS

Key points

- Takayasu arteritis predominates in young women.
- The initial phase of the disease is usually not recognized.
- Predominant involvement is in the aorta, its main branches, and the proximal pulmonary artery.
- Progressive narrowing and an elevated ESR recognize active arteritis
- Prognosis used to be poor and is now good to excellent.
- Treatment of active arteritis is with prednisone alone or in conjunction with cyclophosphamide or Methotrexate.
- Impending ischemic damage, significant aortic regurgitation, and aneurysm formation are indications for angiography or vascular surgery.

PATIENT 13. A 19-year-old woman was admitted to the hospital for evaluation of bilateral arm claudication and abdominal pain. Four months before she had became febrile after falling in a cave during an archeological expedition, at which time she swallowed some still water. Severe headaches and abdominal pain followed. Approximately 4 weeks after the onset of this illness, Arthralgias and typical erythema nodosum lesions appeared on her legs. Laboratory evaluation revealed leucocytosis, a high ESR, and high titer S.typhi antibodies. Blood and stool cultures were negative. The patient was treated with ciprofloxacin for 2 weeks, and all findings disappeared.

I first sow the patient during her second admission after fever and abdominal pain had recurred followed by forearm cramps and numbness upon minimal activities. Both brachial pulses were absent, forearms and hands were cold, and skin capillaries filled slowly after releasing digital pressure. Leg pulses and perfusion were normal. Abdomen was tender in the lower quadrants an arteriogram revealed occlusion of the left subclavian, stenosis of the right subclavian and a questionable left renal artery. The night of admission she had pressure-type precordial pain and hypotension. Pre-admission laboratory studies once again revealed raised S.tiphy antibody titers and negative stool and blood culture results. With a diagnosis of type 3Takayasu arteritis with probable coronary involvement, the patient was treated with a bolus of intravenous methylprednisolone along with ciprofloxacin. She improved dramatically overnight. Two days later the right brachial pulse had reappeared. After the initial Corticosteroid bolus she was placed on prednisone 60mg per day to which Methotrexate was added 3 weeks later as a steroid sparing agent. Three months later she is on prednisone 20mg per day plus Methotrexate 15 mg per week. The patient is back to school and leads an almost normal life. Left arm claudication is still present.

CRYOGLOBULINEMIA AND CRYOFIBRINOGENEMIA

Summary of diagnosis

- Cryoglobulins cause palpable purpura, neuropathy, renal disease, and Raynaud's phenomenon. Possible associations include hyperglobulinemic purpura and hyperviscosity syndrome.
- Persue any evidence of underline disease (infectious, Autoimmune, and neoplastic)
- For cryoglobulins determination blood is allowed to clot at 37C and the serum is placed in the refrigerator at 1C for 7 days; precipitation indicates a cryoglobulins;

centrifuge the specimen to obtain cryocrit; perform Immunoelectophoresis in the precipitate.

- Analysis of the cryoglobulins is seldom indicated in the clinical setting.
- Perform serum protein electrophoresis; if an M component appears to be present, serum Immunoelectophoresis should be requested for confirmation.
- Classify the patient as having typel, II, III cryoglobulinemia and according to underlying condition.
- Cryoglobulinemia results in livedo reticularis, skin ulcers, gangrene,, and thrombosis.
- Cryofibrinogens are determined in EDTA plasma obtained at 37 C; place the sample at 1C. The test is read as positive or negative.

CRYOGLOBULINEMIA AND CRYOFIBRINOGENEMIA

Key points

- A heterogeneous group of conditions unified by immuonoglobulin precipitation in the cold.
- Biochemically, cryoglobulinemia are classified as types I, II, and III according to the composition of the precipitate.
- Skin, peripheral nervous system, and kidney are often involved.
- The clinician's task is to identify the underline condition (infective, Autoimmune, neoplastic, "essential").
- Treatment includes (1) corticosteroids for the treatment of cryoglobulin-caused leukocytoclastic vasculitis (2) removal of the cryoglobulin if clinically indicated, and (3) treatment of the underlying condition, for example, interferon treatment of hepatitis C if clinically indicated.
- Cryofibrinogenemia exhibits cutaneous features of cryoglobulinemia, plus thrombosis.

PATIENT 14. A 60- year-old man was referred for evaluation of a one month history of malaise, anorexia, weight loss, low back pain a petechial rash, plus a positive cryoglobulins test obtained elsewhere. An abdominal aneurysm had been resected 3 months before. An Autoimmune condition, probably an essential cryoglobulinemia or vasculitis were the leading diagnostic considerations. The patient appeared chronically ill. He was afebrile. A florid palpable purpura involved the dorsum of the hands (some of these lesions were bullous), lower extremities and buttocks. A faint aortic regurgitation murmur was present. The back was held rigid. No bony tenderness was noted. A small-vessel vasculitis, a tumoral cryoglobulinemia such as seen in multiple myeloma or lymphoma, and given the murmur and back pain , sub-acute bacterial endocarditis were considered on admission. By the second hospital day the patient continued to be afebrile and 6 admission blood cultures remained negative. Given his poor general condition, Corticosteroid treatment was considered. However, by the next day all of the blood cultures grow entrococcus. Cardiac echoes including transesophageal study were negative. The patient was treated for bacterial endocarditis, and all abnormalities resolved with the exception of the heart murmur. A positive cryoglobulins test, had delayed diagnosis for almost tow weeks fortunately, Corticosteroid never got started.

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THE SPONDYLOARTHROPATHIES

CLINICAL CHARACTERISTIC OF SPONDYLOARTHROPATHIES

1. Typical pattern of peripheral arthritis: predominantly of lower limb, asymmetric
2. Tendency to radiographic sacroiliitis
3. Absence of rheumatoid factor
4. Overlapping extra-arthicular features
5. Significant familial aggregation
6. Association with HLAB27

DISEASES BELONGING TO THE SPONDYLOARTHROPATHIES

Ankylosing spondylitis
Reiter's syndrome/reactive arthritis
Arthropathy of inflammatory bowel disease (Crohn's disease, ulcerative colitis)
Psoriatic arthritis
Undifferentiated spondyloarthropathies
Juvenile chronic arthritis: juvenile onset ankylosing arthritis

Diagnostic Criteria

A clinical signs or clinical history

- Nocturnal lumbar or thoracic pain and/or morning lumbar or thoracic stiffness (1)
- Asymmetrical oligo-arthritis (2)
- Non-specific buttock pain (1) alternating bilateral buttock pain. (2)
- Sausage finger/toe (2)
- Talalgia or any other enthesopathy (2)
- Iritis (2)
- Non-Gonococcal urethritis or cervicytis less than one month before onset of arthritis(1)
- Diarrhea less than one month before onset of arthritis (1)
- Presence or history of psoriasis and/or balanitis, an/or chronic enterocolopathy (2)

- Grade 3 sacroiliitis: unequivocal abnormality. Erosions, sclerosis, widening narrowing or partial ankylosis B. Radiological signs
- Sacroiliitis (stage> 2 if bilateral or stage>3 if unilateral. (3)
- C. Genetic factors
- Presence of antigen B27 and/or family history of pelvospondylitis, Reiter's syndrome, psoriasis, uveitis, or chronic enterocolopathy (2)
- D. Response to treatment
- Subsidence of pain within 48 hours with an NSAID and/or rapid recurrence (48 hours) when it is withdrawn (2)
- if the points from these 12 criteria total at least 6 the patient can be said to be suffering from spondyloarthropathies.

ANKILOSING SPONDYLITIS

MODIFIED NEW YORK CRITERIA FOR ANKYLOSING SPONDYLITIS, 1984

1. Low back pain of at least 3 month's duration improved by exercise and not relieved by rest.
2. Limitation of motion of lumbar spine in the sagittal and frontal planes
3. Chest expansion decreased relative to normal values for age and sex.
4. Bilateral sacroiliitis grade 2 to 4.
5. Unilateral sacroiliitis grade 3 to 4.

Definite ankilosing spondylitis: Unilateral grade 3 to 4 or bilateral 2 to 4 sacroiliitis and at least one of the 3 clinical criteria.

Grade 2 sacroiliitis: definite early changes. Pseudowidening with erosion or sclerosis in both sides of the joint.

Grade 3 sacroiliitis: unequivocal abnormality. Erosion sclerosis, widening narrowing, or partial sclerosis.

Grade 4 sacroiliitis: severe abnormalities. Narrowed joint space, ankylosis.

ANKILOSING SPONDYLITIS

Summary of diagnosis

- Male predominance
- Inflammatory-type low back pain (see Table)
- Absence of features of other spondyloarthropathies
- A positive Schober test: decreased chest expansion.
- Evaluation of sacroiliac joints with an AP of the pelvis with 30 degrees cephalic inclination, or oblique films: if sacroiliitis is present no other imaging studies are needed.
- Bone scans yield many false positives and false negatives.
- CT and MRI are excellent to prove sacroiliitis; the former is less expensive and is therefor the choice.

HLA determination is unnecessary if clinical and radiographic criteria are met. The test is important in pregnant patients.
CBC, platelet count, ESR, alkaline phosphatase, ALT, serum creatinine, and urinalysis are useful to determine baseline for therapy and disease complications.

Table. The clinical history as a screening test for ankylosing spondylitis
Five factors differentiate inflammatory back pain produced by spondylitis from noninflammatory back pain due to other causes. These five factors are:
Onset of back discomfort before age 40 years.
Insidious onset.
Persistence for at least 3 months.
Associated with morning stiffness.
Improvement with exercise.

ANKILOSING SPOPNDYLITIS

Key points

- Ankilosing spondylitis affects predominantly the SI joints and the spine particularly at the thoracholumbar junction, and from their spreads up and down the spine.
- Additional frequently involved joints include the manuberiosternal, the sternoclavicular, shoulders, and hips.
- Early identification (within 2 years of onset) of hip arthritis, high ESR, lack of response to NSIADs, and limitation of lumbar motion are indicators of a poor prognosis in AS.
- Local complications include bony fusion and an increased risk of fractures from early osteoporosis and increased brittleness of the spine.
- Fractures through an ossified intervertebral space (Andersson's fractures) may mimic spinal osteomyelitis.
- Extraarticular complications include relapsing anterior uveitis, aortic insufficiency, AV conduction defects, apical pulmonary fibrosis, Amyloidosis, and cauda equina syndrome (as a result of dural cysts).
- Treatment of AS includes stringent PT to prevent flexor deformity of the spine and hips, anti-inflammatory medications, and sulphasalazine.
- Prognosis in intensively treated AS is excellent. Severe complications of the disease such as cardiac valvular disease or Amyloidosis are fortunately rare.

PATIENT 15. A 40-year old man was evaluated for almost painless progressive limitation of his neck and lower back evolving over a number of years. There had been no GI or genitourinary symptoms. On examination the patient's neck was held in slight flexion with only nodding movements possible. There was minimal motion at the costovertebral joints (chest expansion 1 cm), and the lumbar segment had no measurable lengthening on attempted flexion (schober 0,nl 6-8 cm) An AP films of his pelvis with 30 degrees cephalic inclination showed fused SI joints and bamboo-type fusion between L4-5 and L5-S1. Routine laboratory studies were requested but not HLA-B27, which would not have added if positive or subtracted if negative, to a diagnosis of classical ankilosing spondilytis.

REACTIVE ARTHRITIS AND UNDIFFRENTIATED SPONDYLOARTHROPATHY

Summary of diagnosis

- Oligoarthritis involving lower extremity joints.
- Sausage toes.
- Enthesitis (Achilles tendon, plantar fascia)
- Sometimes conjunctivitis or uveitis, mucosal lesions, balanitis (balanitis circinata), palmar and plantar pseudopustules (keratoderma blennorrhagica)
- Sometimes an intense catabolic state (consider HIV in this clinical setting)
- Preceding enteric or genitourinary infection in reactive arthritis
- Anemia, thrombocytosis, increased ESR
- Positive HLA-B27 or B7 in 5-70% of cases (not required for diagnosis).

REACTIVE ARTHRITIS AND UNDIFFRENTIATED SPONDYLOARTHROPATHY:

Key points

- From a concept of aseptic joint and enthesial inflammation following enteric or urogenital infection, the pendulum has swung to either persistent infection or a reaction to bacterial products lodged at articulator and possibly enthesial sites.
- Associations between reactive arthritis and ankilosing spondylitis, rather than causal, appear to result from the link that both have with HLA-B27.
- Reactive arthritis may be dramatic and lead to wasting; however sooner or later the condition improves or goes away.
- Treatment of reactive arthritis includes (1) cradication of the pathogen if still present; (2) a NSAID, particularly indomethacine; and (3) the use of sulfasalsalazine or Methotrexate in resilient cases.
- In certain locals with high rates of re-infection with C. trachomatis, antibiotic prophylaxis has succeeded in decreasing the rate of recurrence of reactive arthritis.

PATIENT 16. A 30-year-old man was referred for evaluation of possible chlamydia induced Reiter's syndrome. Three months prior he had documented episode of clamydial urethritis treated with a7 day course of doxycycline. A few weeks after this treatment there was another documented episode of clamydial urethritis and a two- week course of cyprofloxacine was prescribed. One week into this treatment the patient developed bilateral Achilles tendinitis, which was considered a manifestation of post- venereal Reiter's. The recommendation was to continue the cyprofloxacine to complete a 2 month course. To everyone's surprise, the patient's symptoms got worse. Pain extended to his knees, elbows and shoulders and resulted in invalidation. The ESR was repeatedly normal. On examination the patient had a great deal on attempted motions. Both Achilles tendons were thickened and tender in their middle third. There was pain and tenderness at the patellar insertion of the quadriceps tendons, the wrist extensor insertion at the lateral epicondyles, and the rotator cuffs. The lack of joint findings, the unusual location of Achilles tendinitis, and normal ESR suggested untoward reaction to ciprofloxacin rather than a reactive arthritis, and the drug was discontinued. A few weeks later the patient was minimally symptomatic.

PATIENT 17. A 60-year-old woman had a bout of campylobacter diarrhea and three weeks later came down with left knee arthritis and left shoulder pain, which on examination corresponded to an inflamed AC joint. Additional findings included tenderness at several interspinous ligaments in the upper dorsal spine and a left Achilles tendonitis. Laboratory abnormalities included platelet count of 520,000/mm3 and an ESR of 80 mm. surprisingly, giving the short duration of arthritis, a pelvic film showed sclerosis on both sides of the symphysis pubis and a left sided sacroiliitis with pronounced sclerosis in the iliac side of the joint. Interestingly, the patient recalled having been febrile for one month while in college, with an ESR of about 100 that persisted a full year. Obviously, this woman who appeared to have a first bout of reactive arthritis already had late radiographic features of spondyloarthropathy. The fever of unknown origin (FUO) while in college probably represented a first bout of reactive arthritis leading in sacroiliitis and pubic symphysitis.

INFLAMMATORY BOWEL DISEASE (IBD) ARTHRITIS

Summary of diagnosis

- Lower extremity arthritis occurs in 20% of cases of IBD.
- Spondylitis similar to AS occurs in 3% to 5% of cases of IBD.
- There is a 50% rate of HLA-B27 in IBD patients with spondylitis.
- HLA- B27 determination in these patients has no practical value and is therefore not indicated.
- There is no association between IBD peripheral arthritis and the HLA-B27.
- Review the SI joints in scout abdominal films; AS may present sub-clinically.
- Erosive changes in peripheral arthritis are rare. X-rays of peripheral joints are therefore not indicated.
- It is important to aspirate the inflamed peripheral joint (1) when symptoms are acute raising the possibility of septic arthritis, and (2) to rule out crystal induced arthritis.

INFLAMMATORY BOWEL DISEASE (IBD) ARTHRITIS

Key Points

- Peripheral joint arthritis in IBD, present in 20% of patients, must be distinguished from septic arthritis, gout and pseudogout.
- Ankilosing spondylitis in IBD present in 3% to 5% of cases is similar to idiopathic AS.
- An additional 15% of patients of IBD have silent sacroiliitis, which does not portend progression to AS.
- Hypertrophic osteoarthropathy is an uncommon association of IBD.
- Behcet's disease and SLE may be mimicked.
- Treatment of bowel inflammation suppresses peripheral arthritis but is said to have no bearing on AS course.

WHIPPLE'S DISEASE

Summary of diagnosis

- A rare condition seen predominantly in middle-aged men.
- Diagnosis is suggested by fever, arthritis lymphadenopathy, serositis, neurologic and ophthalmologic findings, wasting, and steatorrhea
- Tissue biopsies show macrophages laden with PAS-positive, diastase-resistant material intra-and-extracellolar acid-fast negative bacteria are shown by light and electron microscopy
- The agent, T. whippelii can not be cultured but may be identified in tissue samples and blood by PCR.

WHIPPLE'S DISEASE

Key points

- Whipple's disease may present as a seronegative arthritis, a wasting steatorrhea, a sarcoid like multisystem disease, or an obscure neuro-ophthalmologic disease including dementia, convergent nystagmus, and masticatory movements.
- Diagnosis depends on knowledge of disease spectrum and the use of appropriate stains (PAS-diastase, acid-fast) on tissue biopsy.
- Bacteriologic confirmation may be obtained by PCR.
- Antibiotic treatment should be life long, since brain involvement has surfaced in presumably cured individuals. Secondary resistance to antibiotic may develop.

PSORIATIC ARTHRITIS

Summary of Diagnosis

- There are at least eight clinical patterns of psoriatic arthritis.
- Some of the patterns are highly suggestive of the disease: chronic Oligoarthritis, DIP arthritis, "mutilans arthritis," and the sausage digit.
- Presence of psoriasis (review overt and "occult" areas)
- Often psoriasis in the immediate family
- Often spondyloarthropathy in the immediate family
- Laboratory findings are nonspecific (increased ESR, increased CRP, sometime elevation of liver enzymes from concurrent alcoholism)
- X-rays are characteristic in late stages of the disease

PSORIATIC ARTHRITIS

Key points

- Arthritis occurs in about 20% to 30% of psoriatic patients, a 10-fold increase compared with the general population.
- Eight patterns of arthicular involvement are recognized. Several of these patterns (Oligoarthritis, Enthesitis, spondylitis, DIP arthritis, and dactylitis) are also seen in reactive arthritis).
- Extraarticular manifestations are uncommon except uveitis in cases with spondylitis.

- Treatment of psoriatic arthritis includes NSAID's, sulfasalazine, and, in severe or unresponsive cases, Methotrexate.
- Methotrexate treatment of psoriasis is said to cause cirrhosis in about 3% of patients, a 10-fold increase over the rate observed in similarly treated RA.

PATIENT 18. A 35-year-old woman was admitted for treatment of severe Oligoarthritis involving the right knee, proximal and distal interpalangeal joints of the right middle finger, and the left ankle. The condition had been present for 2 years. Recently, the right knee had flared and patient was no longer able to walk. Psoriatic arthritis had long been suspected based on the pattern of joint involvement plus extensive psoriasis in her mother. However no one was comfortable with this diagnosis in the absence of psoriasis. On detailed skin examination, including the "occult" areas, no lesions were found. A pilonidal sinus was found in the coccygeal area the case was presented to the consulting attending who reexamined the patient and concurred with the findings, except that he opened the pilonidal fistula and found psoriasis (later confirmed by dermatologist). A photograph of this lesion was not obtained, but figure 17-6 shows intertriginous lesion in the intergluteal groove in a young woman whose polyarthritis and sacroiliitis went undiagnosed for 5 years prior to this examination.

PATIENT 19. A 19-year-old boy was preparing for a career as a classical violinist. Unfortunately, as he turned 18, psoriasis appeared on his nails and 4 months prior to his referral DIP arthritis appeared in the 2 through 5 digits of his left hand, the every fingers that pressed the violin strings. In the right hand the nails were involved but the DIPs were spared. His arthritis improved on indometacin and Methotrexate, but he never recovered the dexterity he once had.

BEHCET'S DISEASE

Summary of Diagnosis

- Classic triad: recurrent painful oral aphthae, genital aphthae, and anterior and/or posterior uveitis.
- Systemic involvement includes erythema nodosom and other skin lesions, superficial and deep phlebitis, colitis, Oligoarthritis and meningoencephalitis or cerebral vasculitis.
- Ophthalmologic consultation is in order to detect uveitis.
- LP if meningismus is present
- leucocytosis; neutrophilia, thrombocytosis. Negative ANA, negative ANCA
- Colonoscopy if colitis is present to rule out ulcerative colitis and Crohn's disease.

BEHCET'S DISEASE

Key Points

- Diagnosis of Behcet's disease is clinical.
- Suspected cases should be referred to rheumatologist and an ophthalmologist.
- The risk of blindness is higher in Middle East countries; the risk of meningoencephalitis is higher in the United States.

- Oral and genital ulcers respond to local Corticosteroid plus either oral colchicine or dapsone.
- Ocular inflammation may be treated with chlorambucil, cyclophosphamide, or cyclosporine.
- CNS inflammation or vasculitis responds best to cyclophosphamide.

ACUTE RHEUMATIC FEVER (ARF)

Summary of Diagnosis

- Usually a child or an adolescent; sometimes an adult
- Severe febrile illness with very painful, additive, fully reversible arthritis.
- Is there evidence of carditis? Is there a mitral or aortic regurgitation murmur present?
- Examine purposefully the skin for the rare erythema marginatum and probably subcutaneous nodules.
- Other patients present with isolated Sydenham's chorea

ACUTE RHEUMATIC FEVER (ARF)

Key Points

- Acute rheumatic fever follows pharyngeal (not other location) streptococcal infection.
- Mitral regurgitation appears during the ARF episode.
- Mitral stenosis is a late development, sometimes appearing decades after mitral insufficiency, even without clear relapses of the condition.
- Prompt and adequate treatment of streptococcal pharyngitis prevents ARF.
- Secondary prevention is achieved with IM Benzatinic PNC every 3 weeks.
- There have been some foci of ARF in the United States over the past 10 years.
- Different from classic ARF, the condition has not been limited to the lower strata of the population.

Suggested Reading

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Juan j. Canoso. Rheumatology in primary care; 133-149: 1997.

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GOUT

Is a term representing a heterogenous group of diseases found exclusively in the human species, which include:
An elevated serum urate concentration (hyperuricemia)
Recurrent attacks of acute arthritis, in which monosodium urate monohydrate crystals are demonstrable in synovial fluid leukocytes
Aggregates of sodium urate monohydrate crystals (tophi) deposited chiefly in and around joints which sometimes lead to deformity and crippling
Renal disease involving glomerular, tubular, and interstitial tissues and blood vessels
Uric acid urolithiasis

GOUT, ACUTE AND CHRONIC

Summary of diagnosis

- Clinical features of acute gout: important subsets include podagra; acute gout involving large joints; nodal gout; polyarticular gout; gout in rarely affected joints such as the hip, shoulder, and spine; early onset gout.
- Serum uric acid is normal in 30% of gout patients at the time of an acute attack.
- Clinical features of chronic tophaceous gout (cutaneous deposits, chronic arthritis or x-ray changes, visceral)
- Crystal documentation by joint fluid analysis or tophus aspiration
- Identification of risk factors: over weight, excessive purine consumption, alcohol abuse, arterial hypertension, and diuretic use.
- CBC, serum creatinine, serum uric acid, serum triglyceride and cholesterol
- Uric acid and creatinine excretion in 24 hour urine
- X-rays may provide diagnostic features in chronic tophaceous gout but have little value in early gout.

Gout

Key Points

- Hyperuricemia, acute gout, and chronic tophaceous gout should be clearly distinguished because their health implications differ.
- Asymptomatic hyperuricemia (except in individuals with a history of urolithiasis) does not require specific evaluation or treatment above and beyond advice on weight reduction, decrease in alcohol intake, and possibly a change in anti-hypertensive medications.
- Acute gout may be treated in many different ways. The choice of treatment depends on the patient's age and comorbidity.
- Medications are not necessarily required for the treatment of acute gout; remember this in situations of multiple organ failures and when severe complications of therapy have already occurred. Nonpharmacologic treatment includes joint aspiration, immobilization, cold packs, and time.
- Uric acid lowering drugs are considered in patients with frequent acute gout, tophaceous gout (including persistent joint inflammation and bone erosion), and urolithiasis.

- The choice between probenecid and Allopurinol is based on the 24-hour uric acid excretion, adequacy of kidney function, presence or absence of urolithiasis, and cost.
- Realistically, Allopurinol is a more practical drug to use (single dose) and may have far better compliance than uricosurics.
- After 6 months or one year of using a uric acid lowering drug, gout attacks should cease and gross urate deposits should at least partially regress.

PATIENT 20. A 62 year-old man, horse trainer, was referred for evaluation of an obscure symmetric distal polyarthritis of 2 years duration. Studies had show moderate normochromic anemia, a marked increase in ESR some degree of renal insufficiency, low-grade proteinuria, normal serum protein electrophoresis, negative RF and ANA, and trace positive cryoglobulins in his serum. A renal biopsy obtained elsewhere was nondiagnostic, showing mesangial proliferation and vascular fibrosis. On examination the patient was debilitated, unkempt man with synovial thickening and tenderness at the MCPs, Pips, wrists and ankles. There was MTP synovitis with the peculiarity that tenderness was most prominent in the big toes. Admission laboratory results included Hct 35%, Westergren ESR 100 mm/1 hour, serum creatinine 1.6 mg/dl, serum UA 8/5 mg/dl and urinalysis 1+ protein without formed elements in the sediment. Low-grade polyarticular gout was suspected and several joints were aspirated traces of fluid were obtained from a wrist an MCP, and a great toe MTP. Cell count in wrist fluid was 4000/mm3 with 100% mononuclear cells. Synovial fluid smears from all 3 joints revealed intra-and extracellolar MSU crystals, proving the suspected diagnosis of gout Because gout should be overlapping with other rheumatic disease the patient was treated with oral colchicine o.6mg/hour for 8 doses followed by colchicine o.6 mg once daily. Within 3 days of treatment all joints became Asymptomatic. One month later Allopurinol was added and one year later joint examination was normal, Hct was 40%, ESR was 12mm/hour, creatinine was 1.4mg/dl urinalysis was normal and the patient was carrying on with his life at the race track.

PATIENT 21. A previously well 56-year-old man had a string of episodes of severe the patient's lifestyle was disrupted by unpredictable attacks. Serum uric acid levels were minimally elevated, and no tophi were present on examination. Several treatment options were considered: (1) the chronic use of a NSAID, (2) colchicine prophylaxis (vide infra), and (3) long-term Allopurinol treatment with prn treatment of podagra or colchicine prophylaxis during 6 months of therapy.

PATIENT 22. A 70-year-old patient was seen for evaluation of chronic arthritis. At age 50 he began to experience recurrent acute arthritis in his knees, ankles, forefeet and more recently in his wrists and elbows. A diagnosis of gout was made clinically, and individual attacks were treated with P.O. colchicine or NSAIDs. Within the past 10 years joint symptoms have become widespread and persistent, and residual limitation has appeared in his ankle, knees and wrists. Five years prior to evaluation, subcutaneous nodules were noted in both elbows, joint pain became permanent, and morning stiffness lasting hours developed. Physical examination revealed flexor deformities of MCPs, flexible and fixed swan-neck deformities, marked ulnar deviation of digits, hard subcutaneous nodules in the olecranon regions, and effusion, synovial proliferation, and limitation of passive motion at MCPs, wrists, elbows, knees, and ankles. Knees were aspirated at the initial visit, yielding an

inflammatory fluid with myriad intra-and extracellolar MSU crystals. A small amount of chalky material aspirated from one of the nodules represented a dense suspension of extracellolar MSU crystals. Hand x-rays revealed cystic changes in carpal bones and metacarpal heads, dense Periarthicular soft tissues, and a striking lack of Periarthicular osteopenia. Laboratory results included Hct 36%, ESR 60mm/1 hour, serum uric acid 10 mg/dl, and serum creatinine 1.3 mg/dl. Urinalysis was normal and rheumatoid factor test was negative. A diagnosis of chronic tophacaeous arthritis was made, and the patient was treated with Allopurinol 300mg per day plus colchicine prophylaxis during the first year. Improvement was slow but steady; within 2 years joint pain was minimal, he no longer felt stiff and subcutaneous tophi and synovial bulk had drastically decreased.

PATIENT 23. A 45-year-old man was referred for evaluation of possible psoriatic arthritis. Psoriasis had developed at age 32 and was extensive in the extremities. An almost painless joint swelling developed at age 43 involving most MCPs and PIPs. Recently, his right Achilles tendon became thickened and a few episodes minimally painful posterior tibialis tenosynovitis occurred following intensive tennis playing. Interestingly the patient had a history of familial Mediterranean fever (FMF) diagnosed at age 20, and he was one of the first patients in the United States to receive colchicine prophylaxis. The FMF was fully suppressed with colchicine 1.2 mg/day, which was continued to the present. Physical examination confirmed the hand and Achilles tendon findings and there was, in addition, painless diffuse thickening at the roof of the left olecranon bursa. It was suspected that the patient had, rather than psoriatic arthritis, silent (suppressed) tophacaeous gout. Gout was proven by aspiration of the olecranon bursa. Hand x-rays revealed punched-out erosions overlaid by high-density soft tissue thickening, findings characteristic of gout. Serum uric acid was 10.2 mg/dl. Allopurinol 300mg per day was added to the colchicine prophylaxis and psoriasis was treated topically. In the ensuing 4 years all physical findings resolved and there was some filling in of the bone erosions.

Suggested Reading
Rheumatology in primary care/ Juan. Canoso

CALCIUM PYROPHOSPHATE DEPOSITION DISEASE (CPPD)

Summary of diagnosis

- The condition may be Asymptomatic (Chondrocalcinosis) or present clinically as acute arthritis (pseudogout), unusual osteoarthritis, rheumatoid-like arthritis, pseudo AS, and pseudoneuropathic arthropathy.
- CPPD tophi are rarely present.
- Chondrocalcinosisis a descriptive radiologic diagnosis that is not confined to the presence of CPPD crystals. Other crystal species, such as basic calcium phosphate and calcium oxalate, may cast similar radiographic findings.
- Best areas to x-ray include hands
- (Wrists), knees, and pelvis (symphysis pubis).
- CPPD crystals are short, weakly birefringent, and positively birefringent in contradistinction to MSU crystals that are longer, strongly birefringent, and negatively birefringent.

- CPPD with onset below age 50 should prompt a metabolic/endocrine evaluation including serum glucose, Fe, ferritin, alkaline phosphatase, Ca, P, Mg and TSH; between ages 50 and 70,Ca, P, and TSH. Above age 70, none.

CALCIUM PYROPHOSPHATE DIHYDRATE DEPOSITION DISEASE (CPPD)

Key Points

- CPPD may be primary (older people's condition) or secondary to genetic, metabolic, or endocrine disease.
- Correction of an underlying metabolic/endocrine condition does not help established Chondrocalcinosis.
- Parathyroidectomy may be followed by an increased rate of recurrent pseudogout; colchicine prophylaxis should be used in this setting.
- No laboratory marker has been identified in primary CPPD.
- Pseudogout and gout are treated similarly.
- Chronic colchicine or NSAID administration is effective in decreasing the frequency of recurrent pseudogout
- Arthroscopic synovectomy and debridement should be considered in severely osteoarthritic knees.
- Joint replacement is an effective treatment in end-stage CPPD crystal arthropathy.

PATIENT 24. A 48-year-old man, a physical education professor, was referred for a second opinion on his rheumatoid arthritis. Two years before, bilateral wrist and MCP synovitis developed followed by painful swelling in both knees. The patient became progressively limited and got around with crutches with difficulty. He had been treated with NSAIDs, most recently with indometacin 200 mg per day, with minimal improvement. Methotrexate treatment was being considered in the clinic, the patient was unable to climb on the examining table and was therefor examined sitting in his wheelchair. He had florid synovitis in PIPs, MCPs, wrists, elbows, and shoulders with marked limitation of motion in each segment. His neck hurt in all directions of motion, and each motion was limited 50%. Lumbar spine motion appeared normal. Hips adducted and abducted without pain. He had major difficulties in his knees and ankles where pain was so severe that slightest attempted at weight bearing was impossible.

X-rays were reviewed and faint Chondrocalcinosis was noted in wrists and knees. With the patient in the sitting position one of his knees was aspirated yielded 10 ml of turbid fluid, which on examination in the polarizing microscope was loaded with intra-and extra cellular CPPD crystals. The patient was admitted to the hospital and 10 mg of prednisone daily plus colchicine 0.6 mg TID were added to the indometacin. Improvement was slow. By the third day range of motion and isometric exercises were added and the colchicine was reduced to 0.6 mg bid. The patient was discharged home on day 7. The prednisone was phased out over 2 months. The indometacin was, continued at 200 mg per day for 1 month and then reduced by 25 mg per week to 100 mg per day. The colchicine was continued. One year after discharge the patient was working full time as director of a physical education college department.

Suggested Reading
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Ryan LM, MacCarty DJ. Calcium pyrophosphate crystal deposition disease; pseudogout; articular chondrocalcinosis. In:MacCarty DJ, Koopman WJ (eds) Arthritis and Allied Conditions, 1835-1855, 1993.

Rheumatology in primary care/ Juan J. Canoso

SEPTIC ARTHRITIS: NONGONOCOCCAL

Summary of Diagnosis

- Joint sepsis may be suspected from the acuteness of the arthritis, the circumstances of development, and associated findings.
- A history of exposure and a possible portal of entry (such as a skin infection) and some weight to the hypothesis, but a negative history should not distract from considering septic arthritis.
- The patient should be thoroughly examined with particular attention paid to skin (pustules lesions, petechiae, needle marks) and genitalia.
- Suspicion of septic arthritis implies aspiration of the joint for WBC and differential, gram stain, and appropriate cultures.
- Blood cultures should be routinely obtained.
- Dry taps are not acceptable in the investigation of septic arthritis.
- Initial X-rays will show arthicular and Periarthicular swelling plus any preexistent joint changes.
- Infected arthropathies increase the ESR and/or CRP. If one or both of these tests were abnormal, perform an indium-111-labeled WBC scan. If this test is positive, proceed with arthrography and aspiration or, as a last resort, a limited bone biopsy.

SEPTIC ARTHRITIS

Key points

- Risk factors for septic arthritis include age greater than 80, diabetes mellitus, rheumatoid arthritis, a prosthetic hip or knee, joint surgery, and skin infection.
- Untreated, septic arthritis destroys the joint. Treated late (5 days or more after onset of symptoms), it frequently causes postinfectious DJD. The rate of joint damage after nongonococcal septic arthritis is 25% to50%.
- The estimated mortality rate of nongonococcal septic arthritis is 10% to25%.
- Thus, be thorough but move fast, implementing treatment early.
- An infected joint must be effectively drained by needle aspiration, tidal lavage arthroscopy, or arthrotomy.
- All antibiotics have an excellent joint penetration.
- Septic joint should be both rested and exercised to decrease pain, improved cartilage nutrition, and decrease long-term disability.

- Infection complicates about 1% of hip or knee prosthesis. Microbiologic documentation is cumbersome but essential for good results.
- Treatment of prosthetic joint infection is difficult, implying in most cases, removal of the hardware and insertion of another prosthesis either in two stages 1 year apart or in one stage using antibiotic-impregnated cement.

SEPTIC ARTHRITIS: GONOCOCCAL ARHTRITIS

Summary of diagnosis

- Usually in a young adult; close proximity to menses in women
- Arthritis, tenosynovitis, and pustular skin lesions
- Differential diagnosis includes meningococcemia, reactive arthritis, secondary syphilis, hepatitis B prodrome, acute rheumatic fever, and microbial endocarditis.
- Obtain blood, urethral, cervical, anal, and throat cultures.
- Aspirate effused joints. Synovial fluid varies from mildly inflammatory to purulent.
- Purulent fluid is likely to show N. gonorrhea on gram stain and culture.
- Consider clamydial coinfection in all cases.

SEPTIC ARTHRITIS: GONOCOCCAL ARTHRITIS

Key points

- The most common form of arthritis in sexually active young males and females
- The association of arthritis, tenosynovitis, and pustular skin lesions suggests diagnosis
- Coexistent Clymidia trachomatis infection may result in a superimposed or subsequent reactive arthritis.
- Gonococcal arthritis dramatically improves within 48 hours of initiation of therapy. Different from other etiologies of bacterial arthritis, only one week of antibiotics is required.
- Treatment of possible coexistent clamydial infection is indicated.
- Persistent synovitis may indicate post-infectious synovitis, and associated reactive arthritis, or persistent infection.

PATIENT 25. A 34-year-old woman came to the emergency room, complaining of fever, rash, and arthritis. Three days before she had transient sore throat. Since then she felt poorly, developed a fever, and had two shaking chills. Temperature was 39 C and BP 130/80. No pustular lesions were present, and the patient was sexually inactive. Her throat was red. There was an erythematous/hemorrhagic rash with single elements from 5 mm to 2 cm and partial coalescence on her knees and ankles. Knees and right wrist were swollen and painful, and there was angry tenosynovitis in her right wrist and both ankles. Left knee and right wrist aspirates contained 10,000 and 8,500 WBC/mm3, respectively, with high neutrofilia and a negative gram stain. Right knee yielded purulent fluid with intracellular gram negative diplococci on gram stain. Throat culture, blood culture, and right knee SF culture proved the clinical suspicion of meningococcemia with septic arthritis. The patient had uneventful recovery with intravenous ceftriaxone and serial joint aspirations.

MYCOBACTERIAL AND FUNGAL MUSCULOSKELETAL INFECTIONS

Summary of diagnosis

- M. Tuberculosis causes chronic monoarthritis, chronic Proliferative tenosynovitis, and spinal osteomyelitis. Atypical mycobacterium and fungal infection may cause chronic arthritis and Proliferative tenosynovitis.
- The PPD is usually positive in muscoloskeletal tuberculosis including 75% of cases that occur in HIV-infected patients. Malnutrition is a contributing cause of a false negative PPD in these patients.
- Lytic bone lesions present on initial x-rays should raise the possibility of M. tuberculosis infection.
- SF is inflammatory but otherwise nonspecific in M. Tuberculosis and fungal infection.
- Diagnosis of tuberculosis and fungal arthritis is based on fluorochrome and other stains of joint aspirate plus Arthroscopic synovial biopsy for histology, nite blue stain, PAS stain, and mycobacterial and fungal culture. Identification of acid-fast bacteria with a fluorochrome technique is quite useful in the tentative diagnosis of mycobacterial infection.
- Diagnosis of mycobacterial (usually atypical mycobacterias) and fungal tenosynovitis is usually post hoc during evaluation of tenosynovectomy specimen.
- Diagnosis of tubeculous spinal osteomyelitis requires spinal needle biopsy for histology, nite blue stain and mycobacterial culture.

MYCOBACTERIAL AND FUNGAL MUSCULOSKELETAL INFECTION

Key points

- Chronic knee or hip arthritis and proliferative tenosynovitis, particularly if bone erosion is present, should raise the suspicion of mycobacterial (usually mycobacterium tuberculosis) or fungal infection.
- Protracted back pain and stiffness, anterior erosive changes in one or more adjacent vertebra, plus evidence of perivertebral or psoas abscess formation strongly suggest tubeculous osteomyelitis.
- Identification of the etiologic agent and treatment decisions requires specialized advice.
- A positive fluorochrome stain is very useful to initiate treatment pending culture results.
- If the diagnosis is correct, chemotherapy is tailored to the case, and required orthopedic measures are implemented. Treatment results in muscoloskeletal mycobacterial and fungal infections are satisfactory.

Suggested Reading

Juan J. Canoso. Rheumatology in primary care; 1997; 162-175

OSTEOARTHRITIS
HAND OSTEOARTHRITIS

Summary of diagnosis

- Hand osteoarthritis includes Heberden's nodes (Dips), Bouchard's nodes (Pips), and first CMC OA.
- Early Heberden's nodes are characteristically inflammatory. Late lesions are bony knobs.
- Late inflammations of Heberden's nodes suggest gout (nodal gout).
- Bouchard's nodes cause pain and flexor deformity of the joint, weakening grip.
- First CMC OA causes the "square hand" deformity and a painful and weak grip.
- Hand x-rays.
- CBC, ESR, Creatinine, serum uric acid.

HAND OSTEOARTHRITIS

Key Points

- The course of Heberden's nodes is spontaneous regression of inflammation.
- Bouchard's nodes often result in flexor deformity of the digit.
- Bouchard's nodes and first CMC OA may be quite painful and cause hand disability.
- Treatment of hand OA includes hand exercises, non-opioid analgesics, NSAIDs, and intraarthicular Corticosteroid infiltration.
- Hand surgeon referral should be obtained for patients with painful PIP and first CMC OA unrelieved with conservative therapy and if requested by the patient, for the cosmetic correction of Heberden's nodes.
- Suggested Reading
- Moskowitz RW, clinical and laboratory findings in osteo arthritis, In Koopman WJ, arthritis and allied conditions, 13th edition, Baltimore, Maryland, Williams & Wilkins, 1997; 1998-2011.
- Juan J. Canoso. Rheumatology in primary care, 1997; 176-178.
- Egger P, Cooper C, Hart DJ, et al. Patterns of joint involvement in osteoarthritis of the hand; The Clingford study. J Rheumatol 22:1509-1513, 1995

KNEE OSTEOARTHRITIS

Summary of diagnosis

- Older age of patient.
- Obesity, Chondrocalcinosis, previous inflammatory disease, athletic trauma, and previous miniscal surgery are significant predisposing factors.
- Pain increased by activity and relieved by rest.
- Check for joint effusion.
- Check for patellofemoral disease (patellar pain sign, shrug sign).
- Check for angular deformity (varus or valgus).
- Is there evidence for anserine bursitis, prepatellar bursitis, infrapatellar bursitis, or a plica syndrom?
- Is there evidence for referred neurologic pain (spine, plexus, femoral nerve) or hip disease?

- X-rays: standing films and a lateral from each knee for tibiofemoral OA; add axial (patellar) views when patellofemoral OA is a consideration. Joint space-narrowing, eburnation of subchondral bone, subchondral cysts, Ostephytosis.
- Joint aspiration if fluid present to rule out crystalline disease and to rule out infection in the appropriate setting.

KNEE OSTEOARTHRITIS

Key Points

- Knee osteoarthritis may involve preferentially the pattelofemoral compartment, the medial compartment, or the lateral compartment of the joint.
- Always search for an extraarticular cause of pain: finding anserine bursitis, for instance, indicates a reversible component in the patient's condition.
- Exercises, non-opiate analgesics, the addition of low-dose NSAIDs, Capsaicin, and the use of a cane represent the first line of treatment in knee OA.
- Full doses of a NSAID require gastric protection with misoprostol.
- Joint lavage or arthroscopy is indicated when physical therapy measures and medications fail to meaningfully relieve symptoms.
- Tibial or femoral osteotomy and total knee arthroplasty are last-resort measures in knee osteoarthritis. Obesity and age younger than 60 preclude total joint replacement.

Suggested Reading

Rose S. Fife. Osteoarthritis. In: John M, Klipple, eds. Primer on the Rheumatic diseases. Atlanta: Arthritis foundation, 1997: 216-217.

Kevin G. Moder, Gene G. Hunder. Examination of the joints. In Kelly WN, Harris ED,Ruddy S, Sledge CB, eds. Textbook of Rheumatology. Philadelphia: W.B. Saunders,1997: 333-370

Juan J. Canoso. Rheumatology in primary care. 1997: 176-182

HIP OSTEOARTHRITIS

Summary of diagnosis

- Older age of patient.
- Developmental disorder of the hip joint; obesity; joint erosion (e.g., RA); occupational factors (e.g., farmers).
- Pain triggered by activity and relieved by rest.
- Restricted passive motion (flexion, internal rotation).
- Rule out mimicking conditions: trochanteric and iliopsoas bursitis, spinal disease, and various types of neuropathy.
- Hip x-rays: joint space narrowing (superior, medial); osteophytes; degenerative cysts.

HIP OSTEOARTHRITIS

Key Points

- The source of pain in a patient with hip OA must be carefully ascertained; look for extraarticular causes of pain (soft tissue, neurologic).
- Any correctable extraarticular source of pain must be treated.
- The use of a cane significantly reduces the load on the affected hip.
- Any limitations for activities of daily living must be attended to.

- Medications should be given along the hierarchy described under knee OA.
- Surgery is indicated in-patients with unrelieved night pain or severe discomfort during daily activities. The usual procedure is a total joint replacement.

Suggested Reading

Juan J. Canoso Rheumatology in primary care; 1997: 181-182.
Brandt Kd. Management of osteoarthritis. In: Kelly WN, Harris ED Jr, Ruddy S, et al. (eds) Textbook of Rheumatology 1385-1399, 1993.

INFILTRATIVE CONDITIONS

Sarcoidosis

Summary of Diagnosis

- Malaise, fever, dry cough, dyspnea.
- Chest x-rays abnormalities: adenopathies, interstitial changes, scarring, and rarely pleural disease.
- Acute ankle periarthritis in acute Sarcoidosis; rheumatoid-like findings, Proliferative tenosynovitis, and bone changes such as dactylitis in chronic Sarcoidosis
- Heerford-Waldenstrom's syndrome: fever, parotid enlargement, facial nerve palsy, anterior uveitis.
- Loefgren's syndrome: erythema nodosom, lower extremity periarthritis, bilateral hilar adenopathies.
- Skin anergy.
- Increased serum levels of angiotensine-converting enzyme in 75% of cases; hypercalcemia and hypercalciuria in some patients.
- Diagnosis can be made clinically in Loefgren's syndrome, but most cases require histologic confirmation preferably by transbronchial biopsy.
- Infectious granulomas, in particular histoplasmosis (yersiniosis in Scandinavian countries), mycobacterial disease, AIDS, and neoplastic disease must be considered in all cases.

Sarcoidosis

Key Points

- Sarcoidosis is usually diagnosed based on respiratory symptoms and chest x-ray findings
- In a substantial proportion of cases, arthicular, bone or skin findings predominate. Chest x-ray findings suggest the diagnosis.
- Histologic proof (noncaseating granulomas) is required for diagnosis in most cases.
- Infectious and neoplastic conditions must be excluded in all cases based on clinical, pathologic, and microbiologic data. An exception is Loefgren's syndrome. This entity is so characteristic (provided that histoplasmosis, coccidioidomycosis, and yersiniosis are excluded by epidemiologic data) that a clinical diagnosis justified.
- Chronic Sarcoidosis arrests spontaneously in approximately half of cases.
- Corticosteroid are the treatment of choice in progressive disease.

AMYLOIDOSIS

Summary of Diagnosis

Clinical suspicion

In all cases

- Tissue biopsy stained with Congo red and inspected under polarized light. Green birefringence is typical of amyloid.
- Pretreatment with potassium permanganate abolishes the green birefringence of Congo red-stained amyloid in AA and AH amyloid but not in AL amyloid.
- Least problematic biopsy is by needle aspiration of subcutaneous abdominal fat.
- Usefulness of abdominal fat biopsy: AL>AA>AH.
- Suspected AL
- Serum/urine electrophoresis.
- Immunoelectophoresis if an M component appears to be present.
- Bone marrow biopsy if an M component is confirmed.

AMYLOIDOSIS

Key Points

Amyloidosis should be viewed as a miscellany of unrelated conditions that share histopathologic and electron microscopic features.
It is more appropriate to consider "Amyloidosis" rather than "Amyloidosis".
Three types of Amyloidosis-AL, AH, AA-are particularly relevant to rheumatic and connective tissue disorders.
AL resembles RA, AH results in disabling shoulder disease and AA often mimics other types of renal involvement in chronic arthritis an infectious condition.
Myocardial involvement in AL and kidney involvement in AA may result in death.

Treatment in AL and AA hinges on the successful treatment of the underlying disease.
Chronic colchicine administration prevents the development of AA in FMF and may show down progression in other forms of Amyloidosis.

Suggested Reading

Juan J. Canoso. Rheumatology in primary care; 1997: 183-186.

CHAPTER IV

BONE DISEASES

OSTEOPOROSIS

MEDICAL DISORDERS OR MEDICATIONS ASSOCIATED WITH BONE LOSS

Endocrine abnormalities: glucocorticoids excess, thyroid excess, hypogonadism, prolactinomas, anorexia nervosa, hyperparathyroidism, hypercalciuria.

Processes affecting the bone marrow: multiple myeloma, leukemia, Gaucher's disease.
Immobilization: space flight
Gastrointestinal disorders: gastrectomy, primary biliary cirrhosis, celiac disease

Rheumatologic disorders: ankilosing spondylitis, rheumatoid arthritis, osteogenesis imperfecta, homocystinuria, Ehlers-Danlos syndrome.
Other medications: anticonvulsants, Heparin, methotrexate, cytoxan, lithium, cyclosporine

OSTEOPOROSIS

Summary of Diagnosis

- Risk factors assessment.
- Spine x-rays in symptomatic patients.
- Bone densitometry in perimenopausal women.
- If x-rays show osteopenia or BMD values are> 2.5 BD below the mean peak bone mass, determine:
- ESR; thyroid tests; serum alkaline phosphatase, Ca, P, and 25(OH)D3; fasting urinary calcium corrected for creatinine excretion; urinary pyridinoline.

OSTEOPOROSIS

Key Points

- Osteoporosis is caused by an imbalance between bone formation and bone resorption resulting in a decreased bone mass.
- An increased turnover magnifies any tendency to bone loss.
- Fracture rate roughly doubles per each standard deviation (SD) decline below peak bone mass of healthy young individuals.

- Measures to increase peak bone mass (sufficient Ca and vitamin D intake, an active life) are important in prophylaxis and should be implemented beginning in childhood.
- Estrogen replacement prevents the accelerated bone loss that follows menopause.
- Perimenopausal women, unless already committed to estrogen use, should be studied by bone densitometry and possibly biochemical markers of bone turnover.
- A bone mass more than 1 SD below normal peak bone mass (T-score), particularly if associated with an increased bone turnover, is an indication for estrogen, bisphosphonate, or calcitonin treatment.
- Vitamin D deficiency should be suspected in elderly patients who sustained a spontaneous or minimal trauma fracture.

PATIENT 26. A 58-year-old man was seen for evaluation of back pain and appearance of a pointy bone in the middle of his back. Three weeks before, while dressing in the morning, he felt as if something had struck him in the middle of his back. When he touched the area, he felt a bony prominence that had not been there before. There was no pain, but he felt unable to hold anything. That night pain began in the low dorsal area and rapidly became unbearable. Pain was steady and agonizing, worse at night, for the ensuing 3 days. From then on pain was worse during the day in relation with his activities. X-rays showed pronounced spinal osteopenia and a wedged fracture of T10. An MRI revealed that the fracture had a displaced posterior fragment that impinged on the anterior cord. NO abnormal signal suggested neoplasm or infection. The patient was quite osteoporotic (3/5 SD below the mean bone mass of the young). There was no electrophoretic evidence of myeloma. Because he had lost libido several months before, an endocrinologic evaluation was undertaken. Very high levels of prolactin led to head MRI, which revealed a pituitary microadenoma. Bromocriptine, calcium, and alendronate treatment was begun.

Suggested Reading

1. American Society for Bone and Mineral Research. Favus MJ (ed) Primer on the Metabolic Bone Diseases and Disorders of Mineral Metabolism, 2nd ed. Raven Press, NewYork, 1993.

2. Aloia JF, Vaswani A, Yeh JK, et al. Calcium supplimentation with and without hormone replacement therapy to prevent postmenopausal osteoporosis. Ann Intern Med 120:97-103, 1994

3. Liberman UA, Weiss SR, Broll J, et al. Effect of oral alendronate on bone mineral density and the incidence of fractures in postmenopausal osteoporosis. N Engl Med 333: 1437, 1995.

CORTICOSTEROID-INDUCED OSTEOPOROSIS

Summary of Diagnosis

- Increased bone loss is unavoidable in prolonged (more than one month) Corticosteroid treatment.
- Additional osteoporosis risk factors should be assessed.
- Baseline bone densitometry and bone turnover markers determination should be obtained when perimenopausal or postmenopausal women are placed on Corticosteroid.
- Early prophylaxis is important in the prevention of fractures.

CORTICOSTEROID- INDUCED OSTEOPOROSIS

Key Points

- The condition occurs with high-or low-dose Corticosteroid and relates to total cumulative dose.

- Pathogenesis includes decreased osteoblast function, decreased calcium absorption, and increased urinary calcium loss.
- Prophylaxis includes weight-bearing exercises, calcium and vitamin D supplementation, and hydrochlorotizide in hypercalciuric patients.
- Estrogens, bisphosphonate, or calcitonin may be used prophylactically in pre- or postmenopausal women as well as for treatment of established osteoporosis.

Suggested Reading

1. Dequeker J, Westhovens R. Low dose corticosteroid associated osteoporosis in RA and its prophylaxis and treatment: Bones of contention. J Rheumatol 22:1013-1017, 1995.

2. Laan RFJM, van Riel PLCM, van de putte LBA, et al. Low dose prednisone induces rapid reversible axial bone loss in patients with RA. Ann Intern Med 119:963-968, 1993

OSTEOMALACIA

Biochemical determinations in osteomalacia and rickets

Serum

- Low or normal calcium
- Low phosphorus
- Markedly elevated alkaline phosphatase
- Creatinine and phosphorus elevated in renal failure
- PTH levels elevated if hypocalcemia is present
- 25(OH)D low in nutritional, malabsorption, or cholestatic liver disease vitamin D deficiency
- 1,25(OH)2D normal or high owing to secondary hyperparathyroidism (if enough vitamin D is available)
- 1,25(OH)2D low in tumoral osteomalacia

OSTEOMALACIA

Summary of diagnosis

- X-rays of affected part: fracture (s) involving spine, pelvis, ribs, or hip; osteopenia; pseudofractures (Loser's zones); widened epiphysial plates in rickets.
- Serum biochemistry: Ca low or normal, P low, alkaline phosphatase high (this is a normal finding after a fracture).
- Serum 25(OH) D3 low, serum 1,25(OH) 2 D3 normal or high due to secondary hyperparathyroidism if there is enough 25(OH) D3 substrate in nutritional osteomalacia.
- Diffuse achiness, myopathic weakness, serum 1,25(OH) 2 D3 decreased: consider tumoral osteomalacia.
- In appropriate situations rule out anticonvulsant use (Phenobarbital, phenitoin, carbamazepine), vitamin D-dependent rickets, intestinal malabsorption, renal failure, renal tubular disorders, and hypophosphatasia.

Osteomalacia and Rickets

Key Points

- Nutritional osteomalacia is frequent in the geriatric group where it tends to be confused with osteoporosis.
- Tumoral osteomalacia is more frequent than previously believed; it should be considered in-patients with diffuse achiness, myopathic weakness, and low serum phosphate. It may also explain weakness and disability in some patients with known cancer.
- Multiple additional conditions associate with osteomalacia.
- Treatment of nutritional osteomalacia is with vitamin D supplementation; symptoms and findings, including unhealing fractures, resolve within 6 months of treatment.
- Tumoral osteomalacia reverses with the successful removal of the tumor. If surgery is not possible patients may be treated with calcitriol [1,25(OH) 2D3] and phosphate supplementation.

Osteitis fibrosa cystica (OFC): Hyperparathyroidism

Summary of Diagnosis

- Diagnosis may be suspected clinically if the primary or secondary hyperparathyroidism is known, and requires a high degree of clinical acumen if the underlying diagnosis is not known.
- Features of OFC include osteopenia, fractures, diffuse bon pain, rheumatoid-like symptoms focal bone pain and tumor (brown tumors), and destructive arthropathy. Chondrocalcinosis (with or without pseudogout) and myopathy are additional muscoloskeletal manifestations of hyperparathyroidism. Gout may occur from the commonly associated hyperuricemia.
- Laboratory diagnosis of hyperparathyroidism is based on (1) demonstration of persistent hypercalcemia, plus (2) an elevated serum PTH concentration by a method that is sensitive, specific and reliable.
- Radiographic findings in OFC include osteopenia, subperiosteal bone resorption (phalangeal, distal clavicles, sacroiliac joints), osteosclerosis (mainly in secondary cases), and cystic lesions (brown tumors) that occur in association with subperiosteal resorption.

Osteitis fibrosa cystica (OFC): Hyperparathyroidism

Key Points

- OFC is seen most frequently in-patients with renal failure (secondary hyperparathyroidism).
- Hyperparathyroid osteopenia can not be distinguished from osteoporosis based solely on bone densitometry. Bone biopsy may be important in this setting.
- Several rheumatic conditions may be mimicked by osteitis fibrosa including fibromyalgia (diffuse bone pain), RA, and bone tumors. In addition hyperparathyroidism may lead to Chondrocalcinosis and pseudogout and myopathic weakness.
- Profound hypocalcemia following parathyroidectomy is likely in-patients with OFC.

Renal osteodysthrophy

Summary of diagnosis

- Renal osteodysthrophy should be suspected in all patients with end-stage renal failure.
- Clinical findings includes bone pain, fractures, pseudofractures, bone cysts, tumoral calcinosis, and deformity.

- Radiographic findings include osteopenia; subperiosteal erosion [illegible] phalanges, resorption of distal end of clavicles, and erosive changes [illegible] osteosclerosis; "rugger jersey spine"; pseudofractures (Loser's fractures through [illegible] seams); and ectopic calcification.
- Serum P, Ca, alkaline phosphatase, aluminum, 1,25(OH) 2 D3, and parathormone are important in predicting the type of bone lesion present.
- Bone densitometry are unreliable because they tend to overestimate trabecular bone.
- Bone biopsy remains the gold standard to clarify the prevailing mechanism of renal osteodistrophy.

Renal osteodistrophy

Key Points

- Renal osteodistrophy comprises the skeletal manifestations of end-stage renal disease.
- Lesions according to frequency: OFC > adynamic renal bone disease >OFC plus osteomalacia > osteomalacia.
- Laboratory studies are useful in predicting type of bone lesion and mechanisms involved.
- X-ray findings are subtle in many cases and require high-quality films for assessment.
- Bone densitometry is unhelpful if normal.
- Bone scan is very useful in the assessment of fractures and pseudofractures.
- Treatment is based on the use of a low P diet, phosphate binders, control of serum Ca (Ca concentration in the dialysate), and the calcitriol.
- Parathyroidectomy is reserved for refractory hypercalcemia, intractable pruritus, progressive ectopic calcification and calciphylaxis.
- Osteitis fibrosa cystica must be proven by biopsy prior to parathyroidectomy.

Paget's disease of bone

Summary of Diagnosis

- Focal pain, pain on weight bearing, or Asymptomatic.
- Findings on x-rays: an advancing lytic edge ("flamed shaped" or "blade of grass"), sclerosis, trabecular coarsening and disorganization, expanded bone, compromise of at least one end of the bone.
- Increased serum alkaline phosphatase.
- Increased urinary excretion of hydroxyproline and pyridinoline.

Paget's disease of bone

Key Points

- A frequent condition often identified in the fifth decade of life.
- Wide range of extent and severity.
- Clinical activity is determined by assessment of serum alkaline phosphatase and urinary hydroxyproline and pyridinoline.
- Multiple complications may occur at the lesion proper, in a neighboring joint, by neural compression, and systemically.
- Rapid worsening of pain or deformity should be investigated by x-rays plus bone biopsy if suspicion of malignancy arises.

- Treatment should contemplate whether symptoms result from the pagetic lesion, a compressive lesion, or concurrent joint disease.
- Current anti-paget therapies, calcitonin and biphosphonates are helpful in improving symptoms and correcting biochemical parameters of activity.
- Ancillary therapies, including analgesic and anti-inflammatory medications, physical therapy and orthopedic surgery, should be used as needed and add importantly to the successful treatment of paget's disease.

Osteomyelitis

Summary of Diagnosis

- Focal bone pain.
- With or without fever.
- With or without leucocytosis.
- ESR usually elevated.
- A positive probe test in infected pedal ulcers (detection of a rock hard structure at the ulcer base) is proof of osteomyelitis and imaging procedures other than admission x-rays are unnecessary.
- If plain x-rays are normal, obtain bone scan; if positive, document further by MRI or CT (table; imaging in osteomyelitis).
- Unless blood cultures are positive, a bone biopsy or aspiration should be obtained for culture to arrive at a positive diagnosis and choose an appropriate antibiotic agent.

Osteomyelitis

Key Points

- Osteomyelitis should be suspected whenever focal bone pain develops
- Lack of fever and a normal peripheral WBC count are not inconsistent with a diagnosis of osteomyelitis.
- The ESR is almost always elevated.
- Routine x-rays may or may not be abnormal. The assessment of possible osteomyelitis justifies the early use of radionuclide bone scanning, and if this is abnormal, an MRI.
- Successful treatment results in gradual improvement in symptoms followed by ESR normalization and radiologic healing of the lesion.
- Early consultation with an infectious disease specialist, orthopedic surgeon, and vascular surgeon (in osteomyelitis-complicating pedal ulcers) is essential for optimal treatment results.

Hypertrophic osteoarthropathy (HOA)

Summary of Diagnosis

- Digital clubbing.
- Dyaphysial pain and tenderness from periostitis.
- Non-inflammatory joint effusions.
- Sometimes cylindrical edema, Paresthesias, and redundant and hyperpigmented skin
- X-rays of appendicular bones to determine preinstall new bone formation.
- Bone scan useful for the early diagnosis of periostitis.
- Obtain chest x-rays looking for lung cancer and other intrathoracic pathology.

- Consider subacute bacterial endocarditis (SBE).
- Consider gastrointestinal disease (cirrhosis, inflammatory bowel disease).
- Consider hereditary forms of HOA.

Hypertrophic osteoarthropathy (HOA)

Key Points

- Hypertrophic osteoarthropathy is a red flag for a variety of serious conditions.
- Synovial fluid examination, long-bone x-rays, and bone scan in early cases are important for diagnosis.
- Once the cause is identified, if a curative treatment exists (e.g., antibiotic treatment of SBE), full remission of HOA will follow.
- Symptomatic treatment is with NSAIDs, particularly indometacin.

OSTEONECROSIS

Causes of osteonecrosis:

SYSTEMIC

- Sickle-cell disease and other hemoglobinophaties
- Alcoholism
- Hypercortisolism (idiophatic, iatrogenic)
- Primary or secondary antiphospholipid syndrome and other hypercoagulable states.
- Systemic lupus erythematosous (glucocorticoids, vasculitis, antiphospholipid antibodies)
- Oral contraceptives
- Pregnancy
- Hyperlipemia type II or IV.
- Obesity
- Chronic liver disease
- Renal transplantation
- Pancreatitis
- Gaucher's disease
- Fabry's disease
- Dysbarism (decompression sickness, high altitude exposure)
- Carbon tetrachloride poisoning
- Chemotherapy
- Radiation therapy
- Idiopathic

LOCAL

Traumatic: trauma with disruption of arterial blood supply to bone.

Idiopathic:

- Wrist: Kiembock's **malacia** (lunate). Preiser's **disease** (scaphoid)
- Elbow: Osteochondritis **dissecans** (capitellum)
- Spine: Scheuermann's **disease (apophyseal rings** of vertebral bodies)
- Hip: Legg-calve-perthes **disease (femoral head)**
- Knee: Osteochondritis dissecans **(medial femoral** condyle)
- Talus: Osteochondritis dissecans
- Tarsum: Knoller's disease (navicular).
- Metatarsals: Freiberg's infarction (usually second metatarsal head).

OSTEONECROSIS

Summary of Diagnosis

- Clinical suspicion (SLE, Corticosteroid treatment, alcoholism, sickle-cell disease).
- Joint pain on loading in excess of pain during passive motion.
- Relatively preserved range of motion.
- X-rays may be normal in early stages.
- Bone scans show early focal decreased uptake followed by increased uptake around the lesion.
- MRIs are most efficient in diagnosis past the initial 3 to 4 days of the lesion and before x-rays are positive.

OSTEONECROSIS

Key points

- Should be suspected whenever focal bone pain appears in a patient with known risk factors: for example, LE, steroid use, alcoholism, or sickel-cell disease.
- Once a lesion is present, its course is progressive and has the potential, if left untreated, to destroy the joint.
- Additional lesions may appear.
- Treatment of early lesions is with core decompression biopsy. The usefulness of this technique has not been fully documented.
- Most advanced lesions eventually need arthroplasty.

PATIENT 27. An 87-year-old woman with severe coronary artery disease and neurogenic claudication due to spinal stenosis was admitted to the hospital with a protracted episode of angina pectoris. CPK levels remained normal and no EKG changes developed. On the second hospital night sever medial right knee pain developed spontaneously. No effusion was noted. There was true bony tenderness around medial tibial plateau proximal to the anserine bursa and anterior to the medial collateral ligament. The patient, who before was fully ambulatory, was now unable to place the foot on the ground. Prior to her discharge the next day, knee X-ray was obtained and were read normal the following day an MRI was obtained. A large subchondral fracture was present (spontaneous osteonecrosis). She was treated with bed rest and knee protection with a walker. One month later a band of sclerosis had appeared at the fracture site Now after 6 months she is fully ambulatory and only minimal residual sclerosis is present on follow-up knee films

Suggested Reading

Juan J. Canoso. Rheumatology in primary care: 1997; 189-208

CHAPTER V

Pain Amplification Syndromes, Related conditions, and Miscellanea

Benign hypermobility syndrome (BHS)

Summary of Diagnosis

BHS is a clinical diagnosis based on Beighton's criteria. (score>6)
Marfan's syndrome and Ehlers-danlos syndrome must be excluded (it is

not always easy).
Arthralgias, fibromyalgia, and later in life osteoarthritis complicate BHS.
Additional manifestations of collagenous hyperlaxity include skin hyperlaxity, mitral valve prolapse, recurrent joint luxation (shoulder, patella), flat feet, hernias, and prolapses.

Beighton's scoring system for hypermobility syndrome
Score 1 point on each side of the body:

a. Extension of fifth MCP past 90 degrees.
b. Thumb reaches forearm on passive wrist flexion.
c. Elbow hyperextends past 0 degrees.
d. Knee hyperextends past 0 degree.

Score 1 point:
Palms reach floor on anterior lumbar flexion while knees are kept extended.
Maximum score=9
Hypermobility syndrome is present in patients with a score of 6 or greater.

Key Points

- Patients with joint hypermobility are subject to arthicular, ligamentous, and visceral consequences of the hypermobile ligamentous tissues

- Little understood but great concern to the patients are the Arthralgias, tendinous pain, and fibromyalgic features of the condition.
- Flat feet are usually Asymptomatic, but patellar and shoulder luxation produce recurrent disability.
- Reassurance, simple analgesics, and physical therapy work well in this patient. Associated muscoloskeletal syndromes should be treated on their merits.

Marfan's syndrome

Summary of Diagnosis

- Long, thin extremities.
- Arachnodactyly.
- A positive "thumb sign"(thumb protrude from the clenched fists).
- Kyphoscoliosis.
- Reduced vision from subluxed lenses (ectopia lentis).
- Aortic dilatation that begins at the aortic root.

Marfan's syndrome

Key Points

- Marfan's syndrome is an autosomal dominant disorder of fibrillin, a protein distributed in skin, blood vessels, pericondrium, and suspensory ligament for the lens.
- Patients with Marfan's syndrome must be closely followed by a cardiologist working in close association with the primary care physician.
- Prognosis in marfan's depends on the rate of aortic dilatation.
- The rate of aortic dilatation may be slowed down with the use of beta-adrenergic blockers.
- Surgical replacement of the aorta, aortic valve, and mitral valve has been successful in some patients.
- Mimicking conditions include hemocystinuria, congenital contratural arachnodactyly, and the marfanoid hypermobility syndrome.

Ordinary leg cramps

Summary of Diagnosis

- Typically they occur at night.
- The gastrocsoleus muscle is typically involved.
- Diagnosis is clinical.
- Laboratory studies, which are not required, would be normal.

Key Points

- Cramps occur by contraction of an already shortened muscle.
- The shortened position should be discouraged by using loose bedclothes and by stretching while holding the feet in dorsiflexion.
- Quinine taken at night is useful to decrease the frequency of ordinary leg cramps.

Reflex sympathetic dystrophy (RSD)

Causes of reflex sympathetic dystrophy

1. Trauma (fracture, especially Colle's fracture, minor surgery, lacerations, infections, frostbite, burns, aortic surgery).
2. Parkinsonism
3. Hemiplegia
4. Lumbar disc prolapse
5. Amyotrophic lateral sclerosis
6. Cervical radiculopathy
7. Plexus and peripheral nerve injuries
8. Carpal tunnel and tarsal tunnel syndromes Myocardial infarction
9. Cancer
10. Diabetes
11. Hyperthyroidism
12. Isoniazid therapy
13. Uncertain

Summary of Diagnosis

- Follows minor soft tissue, nerve, or bone trauma.
- Pain out of proportion of the findings.
- Pain exacerbation with emotion and environmental warmth.
- Burning pain, hyperpathia, allodynia, and local edema.
- Increased early uptake on bone scan.
- Progression to tissue atrophy.
- Patchy osteopenia on x-rays.

Key Points

- The telltale findings in RSD are pain out of proportion to physical findings.
- Disuse atrophy appears to be central in pathogenesis.
- Treatment includes symptomatic relief, physical measures to counteract atrophy, and a thorough search for a lesion that may be acting as a trigger.
- Oral glucocorticoids and epidural blockade are useful in early disease.
- Late cases may be helped by calcitonin.
- Physicians with special training best handle RSD.

Erythromalgia/Erythromelalgia (ETA)

Causes of erythromalgia/Erythromelalgia

1. Adult, idiopathic
2. Adult, secondary
   - Thrombocythemia: essential Thrombocythemia, polycythemia Vera, myelofibrosis, chronic myelogenous leukemia
   - Vasculitis, SLE, RA
   - Diabetes mellitus
   - Pregnancy
   - Infections: respiratory poxvirus infection
   - Medications: nicardipine, nifedipine, bromocriptine
   - Miscellaneous conditions: pernicious anemia, lichen sclerosis et atrophicus, arterial hypertension, etc.

3. Early forms (may be familial)

Erytheromalgia/Erythromelalgia

Summary of Diagnosis

- Paroxysmal redness, swelling, and warmth in the limbs.
- Triggered by dependency and warmth; relieved by limb elevation and cold.
- Rule out vascular insufficiency.
- Determine platelet count. If elevated, seek a myelodysplastic syndrome.
- Check for SLE, RA, and other associated conditions.

Erytheromalgia/Erytheromelalgia

Key points

- The condition should be distinguished from vasoocclusive disease such as thromboangiitis obliterans and arteriosclerosis obliterans and sympathetic dystrophy.
- Underline conditions, in particular myeloproliferative conditions and connective tissue disease, should be ruled out.
- Cases associated with thrombocytosis usually respond to low-dose aspirin.

Fibromyalgia

Summary of Diagnosis

- Most prevalent in women (3.4% in women, 0.5% in men).
- Highest prevalence (>7%) in women aged 60 to 79.
- Pronounced fatigability.
- Generalized pain.
- Tenderness at 9 or more of 18 typical points.
- Sleep disturbance in most patients.
- Normal CBC, ESR, and TSH.

Key Points

- A chronic painful condition that predominantly affects the women.
- Characteristic feature is symmetrically placed tender points.
- Morning stiffness and fatigue often lead to a wrong diagnosis of RA.
- Pseudoneurologic symptoms are frequent and may be prominent, suggesting, for instance, MS.
- Treatment of fibromyalgia rests on an understanding of the condition, tricyclic agents, and aerobic exercise.
- Unfortunately many patients do not tolerate medications, and of those who do, only 30% improve.
- Treatment of fibromyalgia in specialized centers has a success rate of approximately 60%.
- Given the information just listed, it appears prudent to advise exercising, living life as normally as possible, and avoiding a dependent life.

PATIENT 28. A young woman was self -referred to the clinic for a further opinion on the treatment of recently diagnosed SLE. She complained of fatigue, migraines, alopecia,

morning stiffness, Arthralgias and Raynaud's phenomenon. An ANA determination done elsewhere was positive and hydroxychloroquine treatment was under consideration. The patient was asked- to describe rather than enunciate her symptoms, which she did with some disgust. The "migraine" was described as a band around her head, the reynaud's consisted of a mottled bluish-discoloration of fingers upon cold exposure, the "Arthralgias" denoted pressure pain in the lateral elbows and inner knees, and by "alopecia" she meant a fair amount of hair left on her comb each morning. Findings on physical examination were normal except widespread, symmetrically placed tender points. Tenderness was most prominent in the lateral elbows and inner knees. Laboratory results brought by the patient included a 1:40 (borderline) homogeneous-pattern ANA done on Hep-2 cells and normal CBC, urinalysis, and ESR. The patient was told that her condition was fibromyalgia rather than SLE, but reassurance did not work; she soon saw another rheumatologist corollary: in a patient without clinical evidences of SLE or other connective tissue disease, ANA determination is contraindicated.

PATIENT 29. A young woman, a physician's wife, finally decided to consult after 2 months of living a bed-to-chair existence because a diffuse achiness and lack of stamina. Symptoms had gradually developed over the past 2 years. She had initially attended some psychotherapy sessions. Recently, physician friends had suggested the possibility of fibromyalgia. Past medical history was significant for a bout of depression while the patient was in college. On examination she appeared both depressed and exhausted; general examination including muscle strength was normal; and muscoloskeletal examination revealed disseminated fibromyalgic tender points. Laboratory studies obtained elsewhere included normal CBC, ESR, CPK, and aldolase. Antinuclear antibodies and rheumatoid factor test were negative. Thyroid tests were requested and revealed a marked elevation of TSH and depression freeT4. On thyroid hormone replacement the patient improved her mood, stamina, and achiness, and became once again functional. The fibromyalgic tenderness improved but did not go away.

Myofascial pain (MFP) syndromes

Summary of Diagnosis

- Regional pain.
- Presence of a trigger point (reproduces the entire pain, a taut muscle band is felt, and there is a local twitch response).
- Many patients exhibit additional findings of fibromyalgia.
- There are no laboratory or imaging findings useful to diagnose the condition.

Key Points

- A controversial condition; many of the patients have additional characteristics that are diagnostic of fibromyalgia.
- Another subgroups of patients have occupational disease.
- Patients with typical trigger point and no evidence of fibromyalgia improve temporarily by xylocaine infiltration or the use of a cooling spray.
- Patients with fibromyalgia should be treated for this condition.
- When occupational factors are identified, work environment modification may afford lasting relief.

PATIENT 30: A 60-year-old right-handed switchboard operator was referred because of long-standing right parascapular pain with radiation to shoulder and upper arm. Several physicians had seen the patient, for the preceding 3- years, and a variety of diagnoses had been made including muscle spasm, a myofascial syndrome, and bursitis. Routine chemistries, ESR, chest -x-ray, tangential views of the scapula, a bone scan, and an MRI had been normal or negative. Twice she had been on medical leave with some improvement, but pain promptly recurred upon returning to work. Analgesics, NSAIDs, massage, ultrasound, and laser therapy had afforded minimal relief. The only treatment that had resulted in Asymptomatic periods of up to 2 months had been local infiltration with a mixture of xylocaine and methylprednisolone acetate of which she had four within the last year. Physical examination was normal with the exception of point tenderness in the soft tissue between the tip of the scapola and the spine. Firm pressure on this point failed to reproduce the shoulder or arm pain. There was right-sided pain upon bending the neck to the left as well as bringing the scapulae closer together, suggesting the pain originated within trapezius. It was also apparent that short of a job modification, little could be expected in here case.

After explaining to her my assessment, I agreed to her request of another infiltration, only this time I would use a new, powerful albeit nonsteroidal drug, bupivacaine. Pain relief was complete. Within a few weeks a receptionist position was made available for her at her company and there has been no recurrence of pain. The same effect was achieved by giving her bupivacaine, a local anesthetic whit a longer action (about 3 hours) than xylocaine (about 30 minutes), as with xylocaine mixed with long-acting Corticosteroid.

Repetitive strain injury

Summary of Diagnosis

- In most cases there is a well-defined soft tissue syndrome that needs to be accurately diagnosed if it is to be effectively treated.
- There are also instances of myofascial pain and cases of fibromyalgia that surface clinically in mechanically stressed areas.
- A minority of cases is compensation related.
- Nerve conduction studies may be normal in work-related carpal tunnel syndrome.

Key Points

- Make an effort to establish an accurate diagnosis.
- If the case appears to be work related, obtain a detailed work history and enlist an occupational therapist to inspect the work environment.
- Work environment modification or a change in job description is the physiologic way of dealing with work-related regional pain.
- Tackling the problem from the end result, that is, local or systemic treatments for the regional syndrome, at the exclusion of its cause, can be expected to fail in the majority of patients.

Items to include in the clinical evaluation Of prolonged or chronic fatigue (CFS)

Summary of diagnosis

- A thorough history.
- A thorough physical examination.
- A mental status examination.
- CBC with differential; ESR; Serum alanine aminotransferase, total protein, albumin, globulin, alkaline phosphatase, Ca, P, glucose, BUN, electrolytes, and creatinine; TSH determination; and urinalysis.

Key Points

- Patients who feel physically and mentally exhausted.
- Indeed, the reported frequency in the United States was 24%.
- A duration of symptoms of at least 6 months.
- The cause of chronic fatigue syndrome is unknown, a role of viral infection has been considered.

They complain of at least four of the following eight symptoms:

1. Impaired memory or concentration.
2. Sore throat
3. Tender cervical or axillary lymph nodes.
4. Muscle pain.
5. Multijoint pain.
6. New headaches.
7. Unrefreshing sleep.
8. Postexertion malaise

- Fibromyalgia is present in up to 80% of cases.
- There is no effective treatment for CFS, mainstays of therapy are tricyclic antidepressant agents, physical therapy, and psychologic support.

Joints that rub, squeak, crack, or snap

Summary of Diagnosis

- In most instances diagnosis is clinical
- Tendon sheath rubs are elicited by finger motion, bursal rubs by skin displacement, and joint rubs by angular motion
- Squeaks, are brought about by the motion of contacting bare bone
- Joint cracks and snaps, when associated with sudden changes of range of motion, are unequivocal evidence of an internal derangement of the joint and an indication for arthroscopy.
- Scapular snaps should be investigated with oblique or profile scapular x-rays, and possibly CT.
- Hip snaps are located by palpation during flexion/extension movements. Knee snaps are located by palpation during flexion-extension movements.

Key Points

- Soft tissue rubs in scleroderma indicate severe disease.
- Arthicular squeaks usually reveal denuded contacting bone.
- Finger cracking, which is normally harmless, if abused may lead to mechanical deterioration of the joint.

- Painful clicks, cracks, and snaps indicate internal derangement in joints or inflammation at a prominent site or hole in extraarticular snaps.

Soft tissue pain and fever

Summary of Diagnosis

PIOMYOSITIS

- Tropical piomyositis occurs in seemingly normal individuals. Local trauma may precede muscle infection.
- Nontropical piomyositis affects drug users, patients with AIDS, diabetics, and patients with other debilitating conditions.
- Focal induration and tenderness in muscle; fluctuation later in the course.
- Thighs, buttocks, paraspinal muscles, upper arm, and shoulders.
- Fever.
- leucocytosis (with eosinophilia in tropical cases).
- Documentation by ECHO, CT with contrast or MRI.
- Bacteriologic diagnosis by needle or catheter aspiration, or surgical drainage.

NECROTIZING FASCIITIS

- Type 1(mixed infections) frequently postoperative and in diabetics; predilection for abdomen or perineum.
- Type 2 (Group A streptococcus, Or GAS) usually in alcoholics or intravenous drug users; predominant limb involvement.
- Severe local pain; patch of cellulitis that evolves to bulla formation and necrosis; local anesthesia; there may be crepitus.
- Hypertermia, less often hypothermia; systemic toxicity.
- Systemic toxicity and hypotension in a patient with severe localized soft tissue pain should suggest streptococcal necrotizing fasciitis.
- Surgical debridement is both diagnostic and therapeutic.

SPONTANEOUS GAS GANGRENE

- Abrupt onset of pain, swelling, and tenderness.
- Patient appears gravely ill; pale sweaty, delirious, manic, or apathetic.
- Crepitation.
- Rapid evolution to full thickness necrosis with bulla and blebs.
- Diagnosis proven at surgery: pale or brick-colored muscle that is not contractile and does not bleed.
- Gram stain of exudate: gram positive rods.

Soft tissue pain and fever: pyomyositis, necrotizing-Fasciitis, and gas gangrene

Key Points

- Serious infection should be considered any time in a patient with local pain and tenderness has fever or systemic toxicity.

- Intravenous drug use, AIDS, malignancy, diabetes, and other debilitating illnesses are often in the background.
- Muscle induration and tenderness suggests pyomyositis.
- Severe toxemia with mental changes suggests necrotizing fasciitis and is the rule in gangrene.
- Suspected fasciitis and gangrene call for immediate surgical exploration.
- Prognosis is good in pyomyositis, poor in fasciitis and dismal in gas gangrene.

PATIENT 31. A 64-year-old man who one month before had undergone reconstructive surgery for urethral stenosis presented with pain in anteromedial proximal right thigh. He was afebrile. Some local puffiness was present, but no induration or fluctuance was noted. Hip motion was free and painless and no hernial sac was palpable in the inguinal area. An echo showed normal venous and arterial flow and no abscess. He was treated with naproxen 500mg tid.Ten days later he presented febrile, having had rigors, and marked swelling and induration were now present at the previously painful area. A helical CT with contrast showed multilocular cavity with enhancement at its margin. A large multilocular intramuscular abscess was drained surgically. The root of the abscess was adjacent to the urethra. S.epidermidis grew from the puss.

The patient with a soft tissue mass

Summary of Diagnosis

- Obtain a detailed history (trauma, pain, and growth rate).
- Rule an arthicular or bursal origin of the mass.
- Determine by palpation the structure in which the mass is lodged: subcutaneous tissue, muscle/fascia, and bone.
- Obtain plain x-rays. Phleboliths are characteristic of hemangiomas. Soft tissue osteochondromas exhibit stippled calcification. Other focal calcifications are nonspecific and occur frequently in certain neoplasm (e.g. synovial sarcoma).
- Obtain an ECHO to determine the internal structure of the mass.
- If the mass is not cystic, enlist muscoloskeletal oncologist to conduct further evaluation.
- The next step is likely to be an MRI followed by incisional (rarely excisional), fine-needle, or Tru-cut needle biopsy.

Key Points

- Soft tissue malignancy should suspected in-patients with a soft tissue mass that is not synovial or bursal.
- Superficial lipomas can be confidently diagnosed if more than one lesion is present.
- Ganglia can be confirmed or ruled out by ultrasound.
- All other patients should be suspected of having soft tissue malignancy and should be expediently referred to a muscoloskeletal oncologist.

- Ideally, the biopsy should be performed by the same surgeon who will be in charge of the definitive surgical treatment.
- Open biopsies allow an accurate diagnosis of soft tissue sarcomas. Of the closed biopsy techniques, Tru-cut needle biopsies are best because they provide tissue with the original architecture preserved. Fine needle biopsies are helpful to diagnose local recurrences as well as to sample enlarged lymph nodes.

PATIENT 32. A 60-year-old man presented with a rapidly enlarging lump in the Volar aspect of the left index finger developing during the past 3 weeks. The patient felt otherwise well. On examination the mass was fleshy and interfered minimally with finger motion. Rather than Proliferative tenosynovitis it was felt that the patient probably had a soft tissue sarcoma. A chest X-ray showed a large mass in the left lung field. Excisional biopsy revealed a monophasic synovial sarcoma. A needle biopsy of the lung mass was obtained, revealing identical tissue.

Suggested Reading

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In almost every patient , history &physical provide the key to diagnosis and management of rheumatological problems. however , few rheumatology books give adequate emphasis to the practical examination skills required . Clinical Examination in Rheumatology is a comprehensive and easy - to - follow guide , written to facilitate the full examination of patients with joint and periarticular disease . supported throughout by extensive illustrations and clear instructoins on methodology , this book enables readers to apply basic anatomy in order to recognise common and important causes of regional locomotor pain .

In addition to medical students , the trainee rheumatologist , general physician , physiotherapist and allied health professional will find this volume , which reflects the current educational emphasis on clinical skills , of great practical value .

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