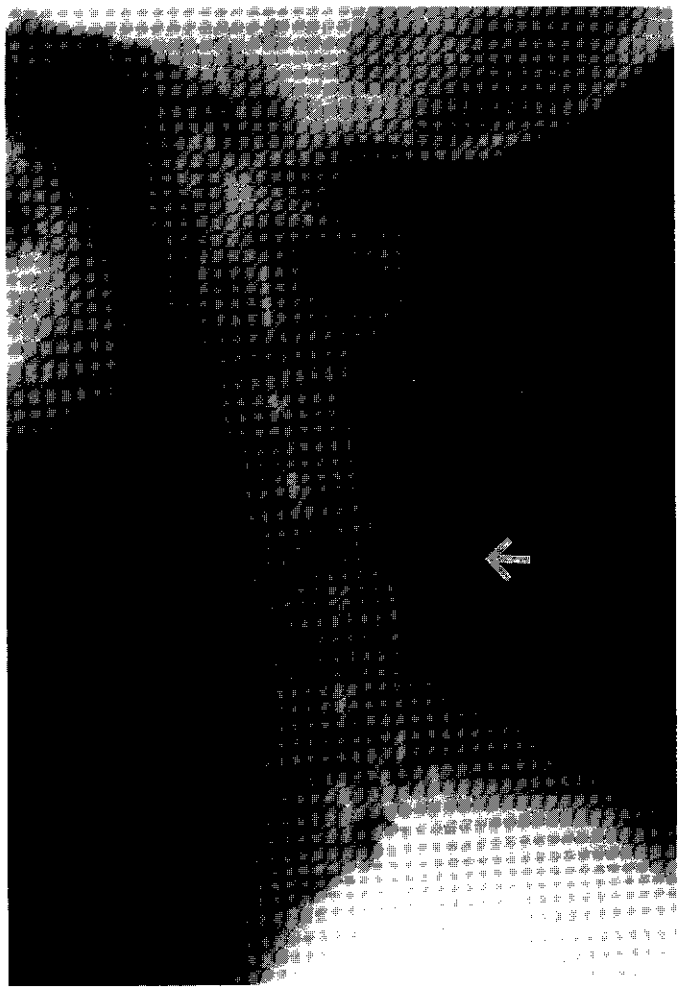
The following represents the second part of the radiology cases which were presented in the June issue of JCCA. The radiographic findings and a brief discussion of the cases are provided for your interest.

These cases were presented as part of a research study that dealt with radiographic interpretation by chiropractors. This research has been funded by the Chiropractic College of Radiologists (CCR). The Journal of the Canadian Chiropractic Association has also assisted in this project with the publication of these cases. It is our hope that everyone has enjoyed the case challenge, even if you were not selected as a participant in our study.

PRIMARY INVESTIGATORS:
Constance A. Columbus, BSc, DC, Radiology Resident,
Department of Radiology, Canadian Memorial Chiropractic College, Toronto, Ontario.
Sandra M. O'Connor, DC, DACBR, FCCR(C), Clinical Radiologist,
Department of Radiology,
Canadian Memorial Chiropractic College, Toronto, Ontario.
Jaroslaw P. Grod, DC, FCCS(C), Adjunct Professor, Canadian Memorial Chiropractic College, Toronto, Ontario.



CASE I

RADIOLOGIC EXAMINATION

A cervical spine Davis series was obtained. The films revealed an acute kyphotic angulation of the mid cervical spine at C4/C5. The C4/C5 disc space is severely wedged anteriorly, with slight irregularity of the superior endplate of C5 (black arrowhead). An anterolisthesis of C4 on C5 by approximately 2.0 mm is noted, with anterior tipping of C4. There is anterior translation of the posterior facets of C4 on C5 by approximately 3–4 mm. The interspinous space at C4/C5 is increased, measuring 2.0 cm (white arrow).

The radiographic findings are consistent with an unstable C4/C5 fracture dislocation (hyperflexion) of the posterior facet joints bilaterally and a possible fracture of the superior endplate of C5. The 2 column involvement necessitates intervention.

DISCUSSION

Bilateral interfacetal dislocation can occur with severe hyperflexion injury of the cervical spine, most commonly at the C4 through C7 levels in adults. The predominant feature of a hyperflexion injury is ligamentous trauma. The ligamentous holding elements are divided into three columns. The posterior column is made up of the supraspinous ligament, the interspinous ligament, ligamenta flava, and the facet joint capsules. The posterior longitudinal ligament and the posterior aspect of the annulus fibrosus constitute the middle column. The anterior column consists of the anterior aspect of the annulus fibrosus and the anterior longitudinal ligament.[1] The affected segments are classified as unstable if two or more columns are disrupted. Disc herniation may also be associated with this injury.[2]

The minimum radiographic study of the posttraumatic cervical spine consists of frontal, lateral & oblique projections. All seven vertebral segments should be identified on the radiographic study. Additional views may be required based on the adequate visualization of the osseous structures, the clinical findings, and the mechanism of the injury. Clinical signs and radiographic evidence of instability on neutral views should be sought prior to performing a flexion-extension study.[2] Clinical signs include pain and neurologic defects at the affected level, which may vary from mild to severe depending on the severity of the trauma.

An abrupt transition to kyphosis with widening of the interspinous space at a single level (divergent spinous sign) is significant, in that it suggests there has been disruption of the posterior ligaments. A kyphosis of 11 degrees greater at one level than at adjacent levels signifies posterior ligamentous disruption.[1] Interspinous gapping of 2 mm greater than other levels or 1.5 times other levels is considered significant.[1,2] The vertebral body of the dislocated segment is usually displaced anteriorly with respect to the caudal segment. An anterolisthesis of greater than 3.5 mm is significant and indicates frank dislocation.[1] Facet joint disruption may range from subluxation to dislocation. Bilateral facet joint subluxation will appear as offset of the articular surfaces with possible gapping of the posterior aspect of the articulations. Often the tips of the articular processes are fractured in association with bilateral facet dislocation. The prevertebral soft tissue fat stripe will be displaced anteriorly with hemorrhage.[2]

Of clinical importance is the high incidence of spinal cord injury occurring with this type of trauma. Bilateral interfacetal dislocations/subluxations may be difficult to detect, particularly at the cervicothoracic junction. However, the previous mentioned radiographic findings should raise the suspicion of this diagnosis, and should then be confirmed by computed tomography (CT) with myelography or magnetic resonance imaging (MRI).[2]

CASE II

RADIOLOGIC EXAMINATION

A lumbar spine series and AP pelvis view were performed. The overall bone density is mildly diminished. Moderate discal thinning and anterolateral osteophytic changes are present at L4/L5 (black arrow). Patchy areas of increased and decreased bone density are observed in the right proximal femur. There is accentuation of the trabecular pattern and thickening of the cortices in this same region (white arrowhead). Thickened cortical margins are also noted along the iliac crest, the subchondral region of the iliac portion of the sacroiliac joint and the left pelvic brim (black arrowheads). The radiographic findings are consistent with Paget's disease of the right proximal femur and left ilium. There is also mild to moderate degenerative changes of the visualized lower lumber spine.

DISCUSSION

Paget's disease is a disorder of bone which involves osteolysis followed by extensive disordered attempts at repair. The etiology of this condition is unknown. The most plausible theory is that the etiologic agent is a slow virus.[3]

It is a common skeletal disease, affecting middle-aged and elderly patients. This slowly progressive disorder which predominantly involves the axial skeleton, is twice as common in males as females.[4]

The presenting complaints are of gradual, progressive, dull pain present at rest and at night, not aggravated by exertion. Also, increasing size of affected osseous structures, and/or deformity may be present.[4]

Radiographic findings include basilar invagination of the skull or deformity of bones due to bone softening. Pathologic fractures, pseudofractures and secondary osteoarthritis may develop in the abnormal bone. Spinal stenosis can result from bony expansion of an affected vertebral body. On initial presentation, the disease is often polyostotic, frequently involving the pelvis, femur, skull, tibia, vertebrae, clavicle, humerus, and ribs.[3]

Four stages of the disease have been described as follows: stage one is osteolytic, stage two is a mixed presentation of osteolytic and osteoblastic changes, stage three consists of sclerotic bone changes, and stage four occurs when there is malignant degeneration. The initial phase displays focal loss of trabeculae creating a geographic lytic lesion. As the disease progresses bone repair is attempted, however abnormal bone is deposited. The result is a disordered coarsened trabecular pattern, thickened cortices and enlargement of the osseous structure. The diseased bone is structurally weak and susceptible to pathologic fracture.[3,4]

CASE III

RADIOLOGIC EXAMINATION

A left shoulder series was performed. The alignment of the glenohumeral and acromioclavicular joints are unremarkable. A patchy area of sclerosis with small luscencies is noted in the left humeral head (black arrows). There is also sclerosis and flattening of the greater tubercle on the left humerus (white arrowhead). These findings are suggestive of early stages of avascular necrosis of the left humeral head, likely a sequela of previous shoulder dislocation.

DISCUSSION

Osteonecrosis is the death of the cells and marrow components of osseous structures, specifically involving the epiphyseal or subarticular regions. In contrast, a bone infarct refers to ischemic necrosis occurring at the metaphyseal and diaphyseal regions of bones. Bone death occurs when there is a significant or complete obstruction of blood supply to a region. Blood flow can be diminished by either intravascular (thromboembolic disorders) or extravascular (trauma, or external compression) processes.[5]

There are many predisposing factors to the development of osteonecrosis, including trauma, alcoholism, corticosteroids, hemoglobinopathies (ie. sickle cell), and Caisson disease. Other cases have no known underlying risk factors and have been termed idiopathic or spontaneous osteonecrosis.[6]

Four stages of osteonecrosis have been described. The first stage involves cell death with microscopic changes in the haematopoietic elements, intracellular osteocytic constituents and marrow fat cells. During this phase there are no radiographic changes on plain films.[7]

The second stage is recognized by hyperaemia of the viable tissues surrounding the osteonecrotic zone. The hyperaemic zone is less radiodense with a coarsened trabecular pattern due to the increased vascularity and inflammatory infiltration in response to bone death. This results in osteoporosis and weakening of the bone. The ischemic region of bone is actually unchanged in density, but will appear as increased radiopacity in contrast to the surrounding hyperaemic zone.[7]

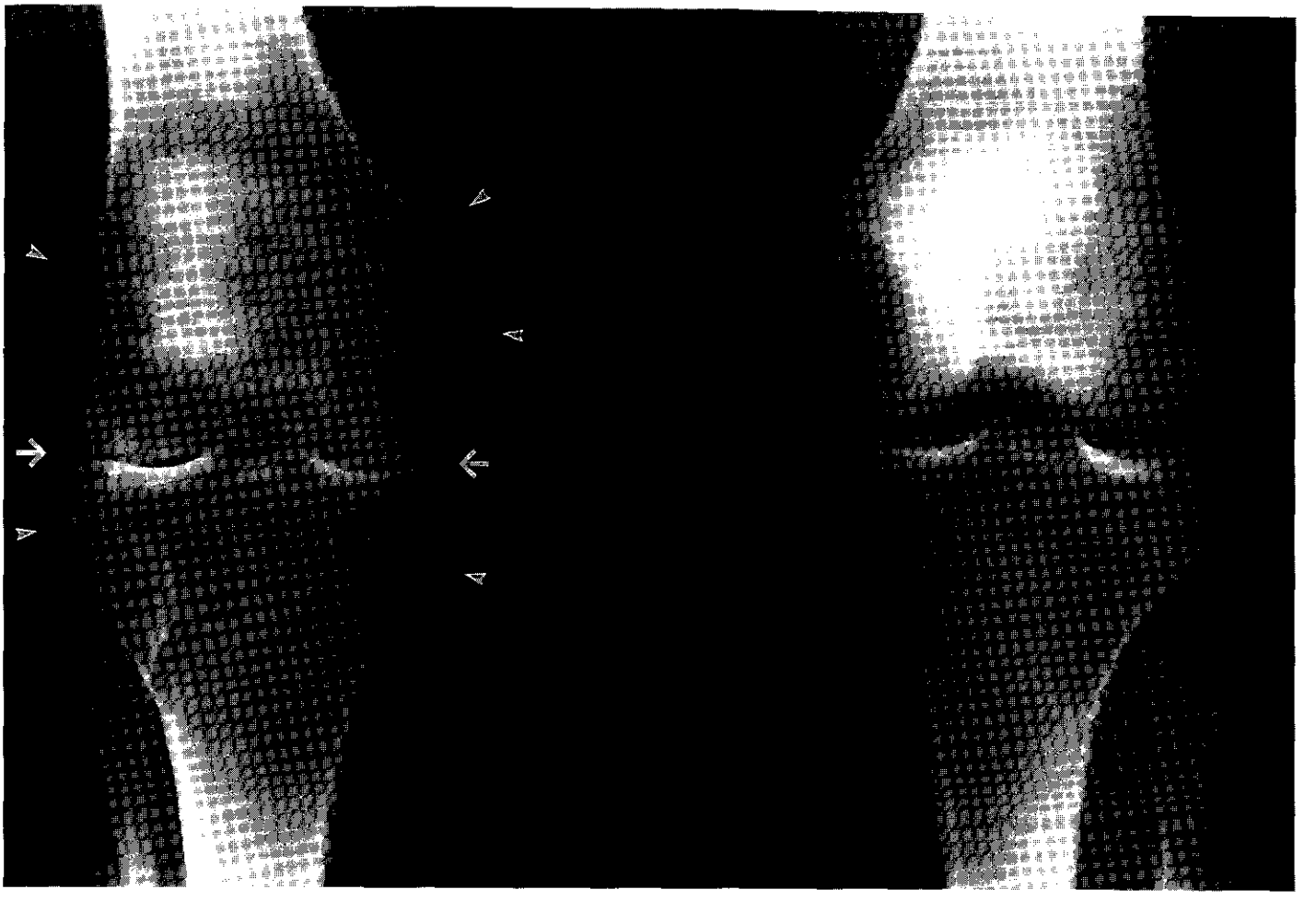
Phases three and four relate to compensatory osteoblastic activity at the viable region of cancellous bone to reinforce the bony architecture which has been weakened by resorption. This is demonstrated by a faint radiolucent zone where osteoclastic activity predominates, surrounded by a dense zone of bone where trabecular reinforcement has taken place.[7]

The supporting osseous structures may be sufficiently weakened by the continued resorption at the reactive interface that fracture and fragmentation of the subchondral bone occurs. On plain film radiographs this stage is characterized by the crescent sign representing subchondral fracture and flattening of the articular surface.[7]

Considerable long-term morbidity can follow osteonecrosis if it is allowed to progress to the late stages. Therefore early detection is extremely important to attempt to arrest progression and facilitate bone healing.[6] The best hope is to identify patients at high risk of osteonecrosis, to educate these persons with regards to early symptoms and to pursue appropriate investigations when suspected. Key points in the history should raise suspicion, such as onset of symptoms related to a particular activity on a specific day, with a degree of symptomatology inappropriate for the type and amount of trauma described. A lack of improvement or progression of symptomatology with time, aggravation of symptoms with activities that load the particular joint, and a lack of other new musculoskeletal problems are also important factors in the patient's history.

Physical examination reveals nonspecific findings including localized and referred pain, antalgic posture, reduced and painful ranges of motion, and adjacent muscular atrophy.[6] If there is a clinical suspicion of osteonecrosis and relatively little physical examination findings, then further investigation is warranted. The first of these investigations should be plain film radiography. In the early stages findings may be subtle or nonexistent on radiographs. Even when radiographic images are unrevealing and there is sufficient clinical suspicion a more sensitive imaging technique such as magnetic resonance imaging (MRI) or a bone scan is appropriate. Magnetic resonance imaging is considered to be the gold standard in diagnosing osteonecrosis.[5]

CASE IV



RADIOLOGIC EXAMINATION

A bilateral AP view of the knees and a left knee series were performed. Mild to moderate periarticular osteopenia is observed involving the left knee joint (arrowheads). There is a mild increase in the size of the left femoral condyles and tibial plateaus in comparison to the right (arrowheads). The left tibiofemoral joint displays severe uniform joint space narrowing (arrows). There is no evidence of osseous spurring or erosive changes. The remaining osseous and soft tissue structures are unremarkable. The radiographic findings are indicative of an inflammatory arthropathy, with asymmetrical involvement of the knee joints. The uniform loss of joint space, marked juxtaarticular osteopenia and enlarged epiphyses of the left tibiofemoral joint combined with the patient's age, duration and location of symptoms are highly suggestive of juvenile rheumatoid arthritis.

DISCUSSION

Juvenile rheumatoid arthritis (JRA) is one of a group of arthritic disorders whose onset occurs prior to age 16. Like the adult form of rheumatoid arthritis, the etiology is unknown. However, the pattern of articular and systemic involvement are different and the prognosis as well. 20% of JRA cases are seronegative and termed Still's disease. This is a systemic disease which can present with a rash, fever, hepatosplenomegaly, carditis, anemia and articular disease.[8]

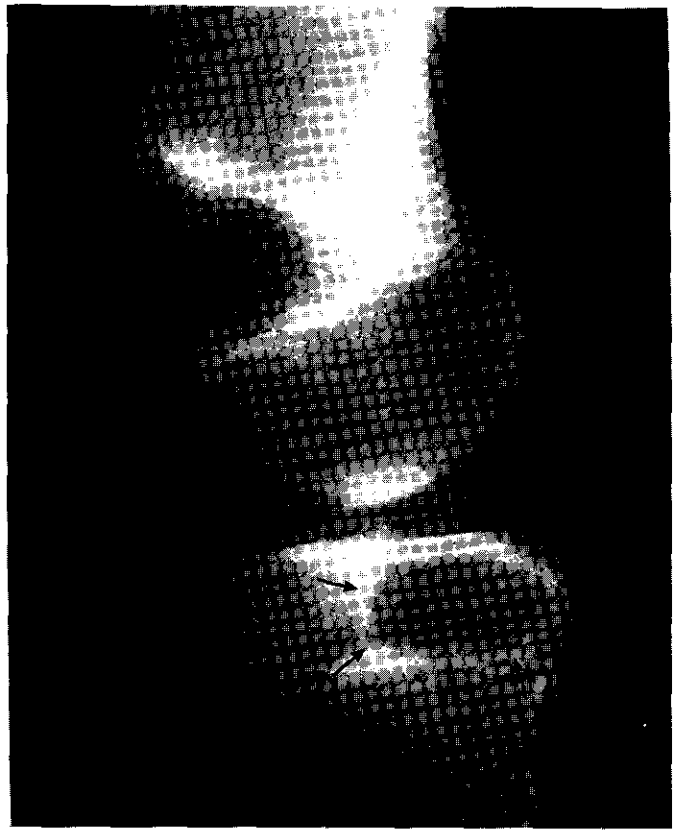
Approximately 20% of patients with this condition will have polyarticular disease. There is bilateral, symmetric involvement with pain and swelling of the small joints of the feet, the hands, the ankles, and the wrists, along with the knees.[9]

However, 30–70% of patients will present with pauciarticular or monoarticular disease, defined as four or less joints involved. The larger joints are more commonly involved, such as the knee, ankle, hip, elbow, or wrist. The most common monoarticular site is the knee.[8,9]

The patient will typically complain of an insidious onset of pain, with mild joint swelling and stiffness. Laboratory testing is not diagnostic. Females are more commonly affected. The prognosis of the juvenile form of disease is good, with long periods of remission.[9] Less than 20% of patients with JRA will develop progressive destructive disease.[8]

The pathophysiology of this disease is identical to the adult form. Therefore, radiographic examination may reveal soft tissue swelling overlying the affected articulation(s), juxtaarticular osteoporosis due to hyperaemia, periostitis, and articular erosions. Later uniform loss of joint space will occur once the inflammation has diminished. Other sequelae include joint subluxation/dislocation, bony ankylosis, and epiphyseal fractures. The most distinctive radiographic findings of JRA are the growth disturbances, consisting of enlarged and expanded epiphyses while the adjacent metadiaphyses appear narrowed. In addition, the long bones may be too long or too short.[8]

CASE V



RADIOLOGIC EXAMINATION

A right knee series including a tunnel view was performed. There is a geographic, rounded, homogenous, lytic lesion located in the posterolateral epiphyseal region of the proximal tibia (small arrows). The margins of the lesion are well defined with no sclerosis, except along the medial aspect where the zone of transition becomes wider with patchy areas of sclerosis (large arrow). Along the inferior margin of the lesion a well defined, linear radioluscency appears to extend through the physis (arrow heads). There is no evidence of expansion, cortical disruption or periosteal reaction. Based on the radiographic findings, this is highly suggestive of a Brodie's abscess (infection). However, a differential diagnosis of an epiphyseal lesion should include chondroblastoma.

DISCUSSION

Brodie's abscess is described as a localized form of suppurative osteomyelitis. This represents a subacute or chronic stage of osteomyelitis, which may occur when an infective organism has a low virulence level or when the host has a diminished resistance to infection. The disease predominantly affects male children, involving the metaphyses of tubular bones. The most common sites include the bones around the knee, ankle, or wrist. Staphylococcus aureus is the main etiologic agent to be isolated from these lesions.[10]

These patients will present with localized limb pain, often nocturnal and alleviated with aspirin. Historical questioning should focus on any recent infections, or dental surgery. Radiographic features of a Brodie's abscess include a geographic lucent lesion located in the medullary portion of bone, surrounded by a zone of sclerotic bone (involucrum). The defect may be oval, elliptical or serpiginous in shape, which may connect to the adjacent growth plate via a tortuous lucent channel. Necrotic debris (sequestration) may be evident with the fluid (purulent or mucoid material) filled cavity.[10,11]

These lesions should be differentiated from a benign tumour called osteoid osteoma, which presents with similar clinical and radiographic findings. The size of the lesion may be helpful in differentiation; the nidus of an osteoid osteoma is usually less than 1 cm in diameter, however, the nidus of an abscess is often larger. Isolation of a causative organism by bone biopsy is diagnostic.[11]

In this case the lesion was located in the epiphysis and mimicked another benign tumour, a chondroblastoma. However, the irregular sclerotic rim and evidence of a lucent channel or tract extending toward the physis was suggestive of Brodie's abscess. Surgical removal of the bone abscess is the treatment of choice. Following appropriate treatment relatively low recurrence rates have been documented.[11]

References

1 Panthria M. Physical Injury: Spine. In: Resnick D. Diagnosis of Bone and Joint Disorders, 3rd ed. Philadelphia: WB Saunders Co, 1995:2859–2864.
2 Yochum TR, Rowe LJ. Essentials of Skeletal Radiology, 2nd ed. Baltimore: Williams & Wilkins, 1996:689–693.
3 Resnick D, Niwayama G. Paget's Disease. In: Resnick D. Diagnosis of Bone and Joint Disorders, 3rd ed. Philadelphia: WB Saunders Co, 1995:2923–2964.
4 Yochum TR, Rowe LJ. Essentials of Skeletal Radiology, 2nd ed. Baltimore: Williams & Wilkins, 1996:1128–1158.
5 Simkin PA, Gardner GC. Osteonecrosis: Pathogenesis and Practicalities. Hosp Practice 1994 Mar: 73–84.
6 Yochum TR, Rowe LJ. Essentials of Skeletal Radiology, 2nd ed. Baltimore: Williams & Wilkins, 1996:1260–1273.
7 Sweet DE, Madewell JE. Osteonecrosis: Pathogenesis. In: Resnick D. Diagnosis of Bone and Joint Disorders, 3rd ed. Philadelphia: WB Saunders Co, 1995:3445–3552.
8 Yochum TR, Rowe LJ. Essentials of Skeletal Radiology, 2nd ed. Baltimore: Williams & Wilkins, 1996:872–877.
9 Resnick D, Niwayama G. Juvenile Chronic Arthritis. In: Resnick D. Diagnosis of Bone and Joint Disorders, 3rd ed. Philadelphia: WB Saunders Co, 1995:971–1003.
10 Resnick D, Niwayama G. Juvenile Chronic Arthritis. In: Resnick D. Diagnosis of Bone and Joint Disorders, 3rd ed. Philadelphia: WB Saunders Co, 1995:2338–2342.
11 Yochum TR, Rowe LJ. Essentials of Skeletal Radiology, 2nd ed. Baltimore: Williams & Wilkins, 1996:1205–1211.