Infrapatellar contracture syndrome

An unrecognized cause of knee stiffness with patella entrapment and patella infera*

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ABSTRACT

Infrapatellar Contracture Syndrome (IPCS) is an infrequently recognized cause of posttraumatic knee morbidity. Unique to this group of patients is the combination of restricted knee extension and flexion associated with patella entrapment. IPCS can occur primarily as an exaggerated pathologic fibrous hyperplasia of the anterior soft tissues of the knee beyond that associated with normal healing. It can also occur secondarily to prolonged immobility and lack of extension associated with knee surgery, particularly intraarticular ACL reconstruction. IPCS follows a predictable natural history which is divided into three stages. Symptoms, diagnostic findings, and recommended treatment are determined by the stage at presentation. Once beyond its early presentation, IPCS is best treated by an anterior intraarticular and extraarticular capsular debridement and release, followed by extensive rehabilitation. The authors review 28 consecutive cases of IPCS. At followup 3 months to 4 years postoperation, the patients had averaged 2.3 additional surgical procedures following their index procedure or injury.

The average increase in extension at followup was 12° with the average increase flexion 35°. Eighty percent of patients demonstrated signs and symptoms consistent with patellofemoral arthritis; 16% of the patients demonstrated patella infera. The authors conclude that prevention or early detection and aggressive treatment are the only ways of avoiding complication in these problem cases.

Loss of knee motion is a common sequela after knee injury or surgery. Flexion exceeding 125° is usually adequate, although the patient may complain of inability to squat. Loss of more than 10° of extension produces comparatively more subjective complaints and alteration in normal gait patterns than loss of flexion. Tolerance for extension loss appears extremely limited and patients usually complain when their knee lacks 10° of extension. Our clinical experience indicates that loss of motion in either direction affects normal knee kinematics and can lead to a progressive arthrosis. Having observed significant morbidity in patients with restricted knee motion, we now perform surgical procedures which are inherently stable, isometric, and which will tolerate early protected range of motion. Despite our aggressive approach to rehabilitation after knee surgical procedures, a few patients still develop notable decreases in knee motion and require judicious treatment to improve their final result.

We have learned that a methodical but aggressive approach is required in treating significant loss of knee motion. Various etiologies of restricted knee motion must be considered, a diagnosis made, and specific therapeutic measures instituted without creating more ligamentous or articular damage. If loss of motion has been long-standing, more than one condition must be corrected, as well as those changes in the knee resulting from immobility.

Because the causes of extension and/or flexion loss can differ greatly, different resolutions have been used. Traditionally, treatment for loss of flexion (extension deformity) has been founded on Payr's detailed study of the morbid anatomy of knee stiffness. Payr concluded that knee flexion loss started in the suprapatellar tissues, particularly the suprapatellar pouch and then led to quadriceps scoliosis, retraction of the alar folds of the patella and patellofemoral adhesions. The final stage was one of a fixed (entrapped) patella with patellofemoral cartilage destruction and tibial articualar destruction secondary to invasion by a fibroscle-
otic pannus. These same observations recur throughout the literature, along with variations of procedures designed to release, lengthen, or partially excise quadriceps muscle tissue.\textsuperscript{2,10,11,22,23}

In 1982, knee extension deformity was further defined arthroscopically by Sprague et al.\textsuperscript{24} who, in reviewing 24 patients, noted three distinct subgroups. Group 1 demonstrated discrete bands of adhesions traversing the suprapatellar pouch; Group 2 included patients with complete obliteration of the suprapatellar pouch and the peripatellar gutters; and Group 3 consisted of patients with obliteration of the suprapatellar pouch as well as the peripatellar gutters but who did not regain normal flexion after arthroscopic debridement. These patients were also found to have extracapsular involvement with bands of tissue extending from the proximal patella to the anterior femur.

It is noteworthy that most authors have dealt almost exclusively with loss of flexion but have rarely alluded to flexion deformity (loss of extension). In an extensive review of posttraumatic knee stiffness, Farrini et al.\textsuperscript{25} mentioned that flexion deformity rarely occurred after meniscectomy, but usually after neuromuscular diseases and septic arthritis. Interestingly, they observed, as we have, that the morbidity associated with loss of extension was much greater than with loss of flexion, yet paradoxically stated, "only rarely does a flexion contracture require surgical treatment." By contrast, Putti,\textsuperscript{19} Connor,\textsuperscript{7} and Heydarian et al.\textsuperscript{6} have all recommended various posterior release procedures to relieve loss of extension. Their papers, and others, on extension loss deal almost exclusively with pediatric and neuromuscular disorders, and universally recommend release of scarified posterior capsule or contracted hamstring tendons and even sectioning of cruciate ligaments to gain full extension. Other procedures designed to lengthen flexor tendons or perform corrective suprapatellar osteotomies have also been suggested for the correction of flexion deformity.\textsuperscript{4,5,26}

In summary, extension deformity has been uniformly treated by suprapatellar, quadriceps, and peripatellar release, while flexion deformity has usually been treated with posterior lengthening or release.

This paper deals with a subgroup of our patients who demonstrated uniquely different symptoms, physical findings, and morbidity. This group presented with patella entrapment in association with loss of extension and flexion (Fig. 1) and still exhibit significant loss of knee motion despite multiple corrective procedures. We have coined the term "Infrapatellar Contracture Syndrome" (IPCS) to refer to patients with this little recognized cause of postoperative knee morbidity. To better treat this select group of patients, we have conducted a retrospective clinical study to define the etiology, anatomy, pathomechanics, and treatment of restricted knee motion associated with patella entrapment (IPCS).

PATIENT REVIEW

Patients for our study had to demonstrate a significant reduction of knee motion associated with decreased patellar mobility. It is important to assess patellar mobility because not all patients with a knee contracture have associated patella entrapment, a condition which can occur secondarily in association with a number of different causes, or primarily with no secondary cause recognized. For our clinical study, only patients with primary patella entrapment were selected (Fig. 2).

At corrective surgery, attention was paid to the soft tissues around the knee as well as to articular lesions of the condyles and patella. Biopsies were frequently taken from the capsule, fat pad, retinaculum, and patellar tendon.

Because our treatment of IPCS spans several years and our protocol has evolved, we review only our last 28 consecutive cases with over 1 year followup (average, 18 months). Of these 28 patients (ages 11 to 52 years, 16 were female and 12 were male. IPCS followed an index surgical procedure in all but two, in whom entrapment occurred after minor anterior knee trauma. Index surgeries on the remaining 26 patients included: 19 anterior cruciate ligament reconstructions, 2 posterior cruciate ligament reconstructions, 2 arthroscopic meniscectomies, 1 arthroscopic lateral retinacular release, and 1 diagnostic arthroscopy, and 1 proximal and distal patellar realignment. The large number of ACL reconstructions in this study may reflect the nature of our practice, but is certainly significant.

Corrective procedures done before patient presentation in our clinic included: closed manipulation, manipulation with arthroscopic debridement, arthroscopy with lateral retinacular release, skeletal traction, and anterior-posterior arthroscopy with debridement. The time span from index procedure or injury to presentation varied from 4 weeks to 1 year (average, 4.3 months). All patients had experienced a period of immobility as a result of casting or pain and swelling.

The chief complaints were knee stiffness and pain. An antalgic, or flexed knee, gait was seen in all patients, with over half complaining of swelling following prolonged standing and walking. Crepitation was a major complaint in over 90% of the patients, with weakness and giving way also commonly present. Actual knee instability was not seen. The majority had undergone supervised physical therapy following their index procedure or injury.

All patients demonstrated a significant lack of extension ranging from 7° to 35° (average, 17°). Knee flexion ranged from 60° to 139° (average, 98°). Moderate to severe quadriceps atrophy associated with a significant antalgic gait was present in all. Patellofemoral crepitation was palpable in over 75%, as was a diffuse synovitis and peripatellar induration, with a "shelf sign" detected in most patients (Fig. 1B). This sign appears when induration in the anterior tissues extends to the distal attachment of the patellar tendon and creates an abrupt stepoff or "shelf" from the patellar tendon to the tibial tubercle.

The other key physical finding was reduced patellar mobility. Significant loss of superior-inferior (passive and active) patellar glide, particularly superior glide, was noted in all patients. Restriction of patellar motion varied depending
Figure 1. Stages of presentation of infrapatellar contracture syndrome. A, Stage I (prodromal stage): Examination typically demonstrates indurated synovial, fat pad, and retinacular tissues. A painful range of motion, restricted patellar mobility, and quadriceps lag are associated. Failure to progress with rehabilitation is an important clue. B, Stage II (active stage): The presence of a "shelvesign" is typical of this stage. Continued peripatellar swelling and induration with severely restricted patellar motion, particularly superior glide, is noted. C, Stage III (burned-out stage): Development of patella infera is the key finding for this stage of presentation. Inflamed and indurated peripatellar tissues are not prominent and patellar mobility is less severely restricted than in Stage II.

Figure 2. Etiology of infrapatellar contracture syndrome.

on the time from origin of symptoms to presentation. Patellar glides were severely restricted in patients presenting early, whereas restriction had decreased somewhat in patients presenting later. The passive patellar tilt test was 0° or less, i.e., negative to the transcondylar plane of the femur in all patients. A shortened patellar tendon with obvious patella infera was present in 16% of cases, all of which presented late. Quadriceps inhibition with a significant quadriceps lag was noted in many patients presenting early, suggesting quadriceps insufficiency prior to passive extension loss. In those presenting early (4 to 16 weeks), retinacular thickening and induration was palpable. The patellar tendon and its articular recesses were obscured by induration of the fat pad and retinacular tissues. Increased warmth around the knee was noted in these patients, but there were no dysvascular changes, hypoestesias, or other findings suggestive of sympathetic dystrophy. Routine knee radiographs of the patellofemoral joint showed mild to moderate disease atrophy in most cases. Patella infera was usually associated with patellofemoral joint narrowing and osteophytosis (Fig. 3).

When physical therapy alone proved inadequate, we revised our treatment to include earlier surgical intervention. Procedures to regain motion were used singularly or in combination and consisted of one of three types: first, closed knee manipulation under general anesthesia; second, arthroscopic adhesiolysis, including anterior fat pad and notch debride ment, and ligament replacement evaluation when indicated; or third, open debridement with lateral retinacular release, anterior debridement, partial fat pad resection, and intraarticular and extraarticular release of the patellar
tendon and patella medially, superiorly, and inferiorly. The majority of patients required open debridement of the anterior knee joint. In those patients requiring surgery, an average of 8 months separated the index procedure from their corrective surgery. The average number of surgeries performed per patient after the initial injury or index procedure was 2.3 (range, 1 to 6).

Findings at surgery were surprisingly consistent despite the varying causes of patella entrapment. In cases presenting 3 to 6 months after the onset of patella entrapment, severe induration and fibrosis of the capsular and synovial tissues and fat pad prevented insertion of the arthroscope without articular damage (Fig. 4A). Biopsy of these tissues revealed a dense, fibrosclerotic reaction with disorganized collagen (Fig. 4B). The patella was firmly adherent to a fibrotic fat pad, with the fat pad occupying the entire anterior space, extending from the intercondylar notch and the medial and lateral joint lines forward to the patellar tendon. To thoroughly release the patella inferiorly, we excised a major portion of the fat pad (Fig. 5A).

Microscopic examination of the fat pad revealed it to be encapsulated with a dense fibrotic tissue in association with an inflammatory reaction (Fig. 5B). Because this reaction usually extended to the medial side of the knee, a second medial arthrotomy was necessary to excise the thickened tissues. Once the patella was freed inferiorly, the suprapatellar pouch could be examined and adhesions released. If an ACL reconstruction had been done previously, the graft would be examined to ensure that impingement was not present in the intercondylar notch and that the ligament was placed isometrically. If impingement was seen and/or the ligament was nonisometric, a diagnosis of secondary patella entrapment was made, and the patient was eliminated from the study. (Secondary patella entrapment occurred as frequently as primary in our total patient population.)

Associated with prolonged patella entrapment were destructive changes of the articular surfaces of the tibia, femur, and patella. These changes appeared to have two origins. First, wherever the fibrosclerotic fat pad had impinged against the medial and/or lateral femoral condyle, a pressure ulceration appeared (Fig. 6A). This was particularly true when vigorous physical therapy including forced extension with traction, extension casts, or heavy traction weights had been prescribed. A second origin of articular destruction appeared to be an infiltrating inflammatory pannus extending from the medial and lateral gutters over the non-contact surfaces of the joint. This pannus was extremely adherent and, once removed, the cartilage below was discolored, softened, and fibrillated. Microscopic sections of the cartilage revealed loss of glycosaminoglycans and subchondral bone atrophy (Fig. 6B). These changes are similar to those described by Waugh et al.** in their study of flexion deformities in rheumatoid and osteoarthritic knees.

Outcome

No patient in this series required posterior capsular lengthening or excision, which should only be considered in cases

Figure 4. Findings at surgery. Stage II IPCS. Top, lateral knee arthrotomy demonstrating thickened and fibrotic capsule. Note hyperemic synovial pannus covering lateral femoral condyle, and fibrotic fat pad occupying the entire anterior retropatellar space. Bottom, hematoxylin-eosin stain of capsular biopsy. Note fibrotic and inflammatory reaction with random collagen deposition.

where surgical repair in this area was part of the original surgery. Virtually every patient achieved large increases in range of motion on the operating table after anterior debridement only. No serious complications arose from the treatment as described. If postoperative drains are used, they should not directly exit the lateral side of the knee, which has little soft tissue protection.

At postoperative review the average extension deficit was 5° (range, 0° to 10°) and the average flexion 133° (range, 115° to 145°). Overall there was an average extension gain of 12° and an average flexion gain of 35°.
Despite increased knee motion, significant residual morbidity secondary to IPCS was noted in this series of patients. Greater than 90% of those reviewed revealed signs and symptoms consistent with patellofemoral arthrosis. Thirty percent of the patients demonstrated tibiofemoral crepitation with early osteophytosis and/or joint space narrowing. It was most alarming that among patients who presented late, no athletes had successfully returned to their preinjury level of sports, and patients who were laborers had not returned to their former employment. The economic drain on these patients and their families from surgical and physical therapy bills and loss of work time was enormous. Most patients, regardless of treatment, required physical therapy daily or every other day for an average of 7.6 months (range, 5 to 14 months).

**CADAVER STUDY**

To better understand the anatomical, clinical, and histological findings of our study, we closely examined anterior knee structures in freshly obtained cadaver specimens (N = 6). The patellar tendon is surrounded by fascial and fibrofascial tissue planes. Posterior to the patellar tendon, a very vascular, fibrofatty pad is firmly adherent to the distal pole of the patella and proximal patellar tendon. It extends through both medial and lateral joint lines to become intimately associated with the anterior horns of the medial and lateral menisci (Fig. 7A). Anterior to the patellar tendon, bursal-type tissues separate the tendon from the overlying deep fascia of the knee which contains the collagenous condensations from the iliobial band laterally and the medial patellofemoral ligament medially. Patellomeniscal ligaments (Kaplan’s ligaments), which are an integral part of the deep fascial planes, extend from the inferior pole of the patella through the medial and lateral extensions of the fat pad to attach to the anterior horns of both menisci (Fig. 7B).12,13 When our findings at surgery are combined with the findings from our cadaveric dissections, the mechanism of infrapatellar contracture and subsequent patella infera appears clearer. At operation obvious pathology was noted in the inferior extensor mechanism described. The fat pad had undergone a fibrocalcification resulting in a firm tissue block to normal knee extension. Fibrous tissue bands (particularly Kaplan’s ligaments) passing through this area of inflammation and fibrosis created a very nonextensible and rigid structure. Because the fat pad extends into the intercondylar notch via the ligamentum mucosae, and it is firmly attached to the anterior horns of the medial and lateral menisci, tibial impingement (which blocks extension) becomes prominent. Failure to recognize this important point in the treatment of an associated flexion deformity could prove harmful. If one releases or lengthens normal posterior tissues when

**Figure 5.** Top, excised hematoxylin-eosin stain showing fat cells below in association with an e.
CLASSIFICATION

Loss of knee motion associated with patella entrapment and infrapatellar contracture can have many causes. A distinction should be made as to whether the patella entrapment is due to primary infrapatellar contracture or to secondary, as a result of prolonged immobility, nonisometric ligament repairs, etc. In our experience, the most common cause of patella entrapment and subsequent IPCS is intraarticular ACL surgery, which necessitates an anterior approach combined with immobility, quadriceps inhibition, and purposeful lack of extension exercises. With the use of strong intraarticular grafts such as patellar tendon, the knee can become "captured" if the graft is placed nonisometrically, thus causing patella entrapment and IPCS due to a prolonged loss of motion. An ACL graft placed too anterior on the tibia can cause impingement at the intercondylar notch, also blocking extension. Likewise, a graft placed under excessive tension...
can prevent the normal anterior tibial glide necessary for full extension. When a patient exhibits lack of extension after ACL surgery, it is important to determine whether this is caused by faulty technique or is primary IPCS. Secondary causes of restricted knee motion with patella entrapment must be ruled out prior to diagnosing primary IPCS. In reviewing the 19 ACL surgical patients with primary IPCS, we found that our cases were equally divided between acute and chronic repair, combined intraarticular and extraarticular substitution, biologic versus synthetic substitution, open versus arthroscopic replacement and associated MCL or meniscal repair procedures. No dominant cause was evident.

We do not believe that what we have observed is merely a normal healing response to knee surgery or injury. Our experience, reinforced by our clinical review, convinces us that 5% of patients having had knee surgery or injury will have an abnormal fibrosclerotic healing response through the anterior retinacular and fat pad tissues. Regardless of the cause or treatment, a small percentage of patients will develop primary IPCS. The syndrome has been alluded to in the past; for example, Andrews has referred to "fibrotic healers," and believes that they comprise approximately 10% of his knee surgery patients (personal communication, 1985). Only after following patients from early presentation to patella infera did we begin to understand this syndrome. We now recognize a continuum of pathophysiologic findings that suggests our patients exhibited various stages of the same disease process. Three stages of IPCS can be distinguished: the prodromal stage, the active stage, and the residual or "burned-out" stage.

I. Prodomal stage

Nearly every patient undergoing knee surgery or sustaining a knee injury enters the prodomal stage of IPCS as a normal consequence of healing (Fig. 1A). However, those who progress to the active stage of IPCS will have primary IPCS or, for one of the reasons discussed above, will develop secondary IPCS.

Patients who will develop primary IPCS at the prodromal stage are usually seen within 2 to 8 weeks of their surgery or injury. Periarticular inflammation and swelling combined with immobility and quadriceps weakness are common. Such patients also fail to progress during rehabilitation, are unable to gain extension, and exhibit abnormal pain and swelling. Diffuse edema is present throughout the periarticular tissues, particularly in the area of the patellar tendon and fat pad. Tenderness is noted about the patellar tendon, and a painful active range of motion is present. A quadriceps lag is present, indicative of a passive range of motion greater than an active one. Patellar glide tests demonstrate decreased patellar excursion; patellar tilt is restricted, although not rigid.

II. Active stage

Patients in this stage have progressed from Stage I unrecognized into the active stage of IPCS within 6 to 20 weeks (Fig. 1B). These patients present with significantly decreased patella mobility and usually have had previous manipulations or surgery which has exacerbated their problem. Fat pad induration and patellar tendon rigidity are now present. Obvious extraarticular involvement and a "shelf sign" are present. There is a severe decrease in patellar mobility in all directions, with a zero or negative patellar tilt test. A quadriceps lag is no longer present, in that both active and passive knee extension and flexion are restricted. Significant quadriceps atrophy is apparent, with patellofemoral crepitus on tracking tests. Upon voluntary contraction of the quadriceps, the patella fails to move superiorly. The patient demonstrates a bent knee or "short leg" gait.

III. Residual stage

Patients with Stage III disease will generally present from 8 months to years after the onset of IPCS (Fig. 1C). Depending upon the time from the onset of the syndrome, the physical findings may reveal more supple peripatellar and retinacular tissues, with some residual fat pad hypertrophy. Medial and lateral patellar glides are restricted but not as severely as in Stage II. Significant patellofemoral arthritis is demonstrated by patellofemoral crepitus, and decreased joint space is seen by radiograph. Developmental patella infera is common. Continued quadriceps atrophy and weakness are present, with restricted flexion and extension. If enough time has elapsed, many of the signs indicative of IPCS will have disappeared. Residual patella infera and patellofemoral arthritis may be the only remaining symptoms.

TREATMENT RECOMMENDATIONS

As our experience with patella entrapment grew, we were making earlier diagnoses and, on occasion, were able to treat the syndrome arthroscopically. However, in advanced cases it was apparent that surgery that intraarticular and extraarticular involvement was present, mandating open debridement; failure to recognize the extraarticular component in these cases would generally necessitate repeat surgery to achieve more motion.

In assessing our treatment, then, we noted that patients diagnosed in Stage I and treated aggressively did better than those diagnosed in Stage III. Also, those who had combined open debridement, with or without manipulation, appeared to do better.

Aggressive rehabilitation using early motion and muscle stimulation is, therefore, the key to prevention. Patellar mobilization techniques should be used by the therapist and taught to the patient. The liberal use of antinflammatory medications may also prove helpful. Those patients with a tendency to lateral patella compression, patella infera, and poor pain tolerance should be identified early and treated vigorously. Analgesics and TENS units should be used liberally early in the recovery phases. Detected early enough, patients with Stage I IPCS will generally respond to this program.
If patellar mobility and knee extension are not achieved quickly, a "drop-out cast" or extension board can be used to achieve extension (Fig. 8). However, caution must be exercised when using such forceful extension measures as drop-out casts, anesthetic manipulations, subluxation hinges, or traction, since acceleration of chondral lesions can occur. Therefore, these techniques should be applied early and only briefly (3 to 5 days), and if unsuccessful, discontinued until after surgery, when they can again be used with caution. Strengthening of the quadriceps musculature must accompany the use of these techniques and/or surgery. If extension is achieved without associated increased quadriceps strength, flexion deformity may return. Therefore, it is imperative that a voluntary quadriceps contraction exist, with little or no quadriceps lag, prior to using extension drop-out casts, traction, or surgery. Neuromuscular stimulators have proved valuable in this regard.

Approximately 5% of patients will go on to active Stage II IPCS (primary IPCS). However, the majority of patients will be particularly susceptible to the treatment selected by the physician, who will thus determine whether they heal normally or progress to active Stage II IPCS (secondary IPCS).

Patients with Stage II, or active, IPCS generally require surgery. Continued manual manipulation, forced passive motion, or traction should be avoided since any maneuvers causing recurrent inflammation and pain will only worsen the severity of the disease process at this stage. The timing of surgery is critical; delays for months may be necessary to reduce inflammation and increase quadriceps strength. Patients in Stage II generally have both restricted extension and flexion. Attempts at mobilizing the knee usually prove futile, and it is better to work on muscle strength through the range of motion present. Also, when both flexion and extension contractions exist, it is preferable to "divide and conquer" than to attempt to correct both simultaneously. Achieving extension as soon as possible should be the priority.

Surgery for Stage II IPCS usually includes both intraarticular and extraarticular debridement and release. Arthroscopy is of little help. Lateral arthroscopy should include lateral retinacular release (excluding the vastus lateralis tendon), along with debridement of hypertrophied and dysplastic fat pad tissues and the lateral patellomeniscal ligament. A limited medial arthrotomy to further debride the fat pad and excise the medial patellomeniscal ligament is usually necessary. Attention must be paid to the fat pad attachments near the anterior horns of the menisci and the intercondylar extensions to the notch. If an ACL repair or reconstruction has been performed, attention must also be paid to the ligament in relationship to the femur, tibia, and intercondylar notch. Nonisometric placement of the ligament necessitates removal. At the time of arthroscopy, normal patellar mobility must be restored with passive patellar tilt exceeding 45°; peripatellar tendon adhesions, as well as suprapatellar adhesions, must also be released. One must also be prepared to advance the tibial tubercle proximally if patella infera is present. All incisions and portals must be meticulously closed and prophylactic antibiotics used. Nonsteroidal antiinflammatories should be given for an extended postoperative period and, if steroids are used, they should not be used until all wounds have healed (2 to 3 days). Continuous passive motion, particularly beneficial early in the postoperative recovery stages, can be combined with epidural morphine injections to aid pain control. Postoperative use of a drop-out cast, particularly at night, is usually necessary for a brief period.

Postoperatively, physical therapy is required on a daily basis. Nighttime extension splints are encouraged until active extension is demonstrated by the patient. Although it is desirable to preserve the knee flexion obtained at the time of surgery, it is far more important that knee extension be maintained; a loss of flexion can usually be rectified later with arthroscopy and manipulation. On occasion, it is necessary to include experienced psychological support because of the prolonged and associated morbidity with IPCS.

Patients presenting with Stage III ("burned out") IPCS have usually had multiple surgeries yet have persistent motion loss and/or residual knee arthritis resulting in moderate to severe impairment. Vigorous physical therapy fails to make substantial gains in these patients because of the associated arthritis. Likewise, corrective surgery, such as debridement and retinacular release, is of little benefit due to marked articular degeneration. Surgery should be reserved to control pain and be confined to "salvage" procedures such as tibial tubercle advancement, patellectomy, Maquet osteotomy, or total knee replacement.

**DISCUSSION**

In our opinion, immobility alone cannot account for the connective tissue changes we have observed. Rather, we have concluded that changes in the fat pad and retinacular tissues are a result of both injury and immobilization. Although it has long been observed that immobilization of joints surrounded by edematous and inflamed soft tissue often produces joint stiffness more quickly and severely than immobilization of nonedematous joints, the mechanism for this is
still controversial. In 1965, E. E. Peacock studied the biomechanical and biophysical aspects of joint stiffness. His intent was to define the role of collagen synthesis. Peacock's hypothesis was that molecular cross-linking would be prohibited by the physical distance between collagen fibrils and that the cause of intraarticular joint fibrosis was shortening of capsular and collateral tissues secondary to new collagen synthesis, to collagen reabsorption, or to both. Performing a series of animal studies in which joints were immobilized and cross-linking of collagen tissues was measured, he concluded that joint stiffness was secondary to changes affecting the joint capsular tissues, and that new collagen synthesis could be measured in immobilized joints. Peacock hypothesized that the prevention of new collagen synthesis or selected removal of newly synthesized collagen would hold promise in the treatment and control of joint stiffness.

Contrasting conclusions were presented by Akeson and Woo. They observed that while collagen synthesis continued, it did not accelerate, and the quantitative changes in collagen matrix components did not readily explain the physical changes observed following immobilization. They did note, however, a reduction in the concentration of glycosaminoglycans and water. They proposed that this decreased concentration could explain the alteration in the plasticity and pliability of connective tissue noted by Peacock. To confirm this hypothesis, they used the rabbit knee joint as a model and observed, as did we in our patients, significant fibrofatty proliferation of tissues in the intercondylar notch of these specimens. As postulated, water and glycosaminoglycans content were significantly reduced. Since they observed no change in total collagen content, they concluded that "the mechanisms which changed the physical characteristics of periarticular connective tissue, seemed to be cross linking of existing fibers or deposition of a small amount of new collagen, disposed randomly in the connective tissue weave without regard to the usual constraints imposed by physical forces." If the changes we observed in IPCS were secondary to immobility alone, one would expect reorganization of existing collagen but little new collagen formation, as noted by Akeson and Woo. If, however, inflammation combined with immobility results in collagenation (new collagen formation), as stated by Peacock, one could explain the process of IPCS. The clinical presentation of our patients is very similar to findings seen in the animal model studies and represents changes consistent with both Peacock's and Akeson's conclusions, which we believe are not mutually exclusive. Periarticular connective tissue, inflamed and edematous from injury, results in rapidly developing joint stiffness when immobilized. Loss of motion which modulates the synthesis of proteoglycans and collagen in the normal joint leads to decreased water content and glycosaminoglycans and increased collagen fiber cross-linking. New fiber formation occurs randomly throughout the periarticular connective tissue and fat pad, fueled by inflammation. In primary IPCS this process occurs rapidly and is enhanced by unknown (genetic?) factors. In secondary IPCS, however, additional factors must be presented.

**Patella infera**

The extensor mechanism is particularly prone to patella entrapment and patellar tendon contracture. The variable extent of ligamentous attachments, particularly the patellomeniscal ligaments, suggests that some knees may be more likely to develop patella infera than others. Patella infera occurred in 16% of our cases, the majority of our Stage III patients. Progression from a normal patella height into infera was documented in several of these patients, but not all. It is unknown whether untreated IPCS would inevitably result in patella infera. We found that a very specific set of conditions must occur simultaneously for patella infera to develop. The first condition is a peritendinous inflammatory reaction which creates a constricting scarification in the surrounding tissues of the patellar tendon (IPCS). Second, the patella must become entrapped in the lower femoral sulcus, secondary not only to infrapatellar contracture but also to patellofemoral adhesions in the medial, lateral, and suprapatellar areas. This latter situation is facilitated by quadriceps weakness and an associated quadriceps lag such that normal patella excursion is not maintained, thereby permitting suprapatellar pouch contracture. As a patient progresses from Stage II IPCS to Stage III, the infrapatellar tissues soften and extension gradually increases. Meanwhile, the patella remains entrapped in the lower femoral sulcus (from suprapatellar pouch scarring), and then patellar tendon contracture occurs.

In the past, IPCS as a cause of patella infera has been largely unrecognized. In an as yet unpublished paper entitled "Patella Inferra Syndrome," Wojtys et al. reported 11 patients with patella infera (presented at AAOS annual meeting, New Orleans, 1986). The presentation of these patients, their physical findings and tissue biopsies are consistent with our Stage III IPCS. They noted that patellar tendon length was as much as 83% shorter and that this could occur within months of the onset of "quadriceps muscle hypotonia." Although quadriceps hypotonia is certainly an important aspect of IPCS, we believe that developmental patella infera is a consequence of IPCS and patella entrapment. Our opinion is supported by the fact that patients with quadriceps hypotonia are extremely common, yet developmental patella infera is very rare.

In an extensive review of 128 cases of patella infera, Caton, Dejour et al. document the origin of their cases as iatrogenic, mechanical, natural, or algodystrophic (inflammatory). It is probably within this latter group that the syndrome of Infrapatellar Contracture Syndrome existed. Dejour, on an AOSSM Traveling Fellowship in 1988, commented on the causes of patella infera: "Bad indications, bad surgery, bad surgeon, bad patient." Perhaps a fifth category should be added: "bad healing."
SUMMARY

IPCS is an infrequently recognized cause of patella entrapment with restricted knee motion. IPCS results in significant morbidity to patients with a common history of knee inflammation and swelling secondary to surgery or trauma associated with quadriceps weakness and knee immobility. Through clinical, anatomical, and histological studies, we clarify this syndrome and distinguish it within the more general entity of knee arthrofibrosis. Patients suffering from IPCS progress through three stages of the disease with increasing symptoms, arthrosis, and morbidity. Early detection is the only way to avoid significant complications. When surgery is indicated, we recommend a combined intraarticular and extraarticular anterior approach, in combination with extended postoperative rehabilitation.

REFERENCES

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DISCUSSION

Kenneth E. DeHaven, MD, Rochester, New York: I think this is a very important paper for two reasons. It first of all focuses attention on limited motion following major knee surgery and injury which can be a very serious problem and which does not receive enough attention, although it leads to severe consequences, with both functional symptoms and degenerative changes. This paper focuses on that problem in general and very specifically on a particular group of patients in that category.

The second important thing about the paper is that it clearly identifies a group of patients who have a more or less limited area of arthrofibrosis. The authors have defined the clinical presentation and the physical and surgical findings of these cases, outlined treatment recommendations based on their results and observations, and have proposed both etiology and pathophysiology. They are to be congratulated for this effort.

I must say that I have not encountered, or at least I have not recognized this exact syndrome, but I have treated several cases that have many features in common with these, except that the arthrofibrosis also obliterated the suprapatellar pouch, and if the patella infera was present in these cases, it was not recognized. Their patients and ours have many features in common, the problem being postoperative and often after ligamentous surgery, although our cases are heavily weighted toward acute ligament surgery versus chronic. I would ask the authors whether theirs is similar or not? In our experience, it tends to be an older patient, over the age of 25, with limited flexion and extension. The patellar mobility is greatly limited, which is very similar, as are the results of lack of improvement with time and conservative treatment and frequently articular changes. So, all of those characteristics are very similar, but the difference is that in this series the suprapatellar pouch obliteration was not a significant part of the spectrum. With these cases that I have treated, there has been improvement with percutaneous release and resection of the arthrofibrosis with very excellent return of flexion and a limited improvement of extension, averaging about 8°, so the cases were not as severely limited in extension as some of the cases in this series. And also, the ultimate functional results have not been as discouraging in the group of patients that I have treated. In short, it seems likely to me that the basic process in our cases and the present series is similar and related but certainly further study is necessary to clarify this.

The authors have identified the extraarticular as well as
intraarticular nature of this arthrofibrosis in their cases, and if the flexion contracture cannot be corrected by percutaneous release and resection, especially with the patella infera presentation, their recommendations for open intraarticular and extraarticular surgery appear very sound, and I am going to add this to my approach to these difficult patients.

I appreciate the authors bringing this to our attention, and I encourage them to continue their efforts to understand the process. I echo their plea to prevent this complication by meticulous attention to detail in the original surgery and by early mobilization, and if you cannot prevent it, to recognize and treat it early, when it is easier to deal with, rather than late.

Authors' Reply: Thank you, Ken. We could not present everything about the syndrome today; there just was not sufficient time. I think I would like to stress that the number one cause of IPCS in our series was intraarticular ACL surgery for two reasons: first of all, about 5% to 10% of those patients will develop primary fibrosis or that fibrotic healing that Jim Andrews refers to. Another 10% develop it on a secondary basis, secondary to either a nonisometric graft, purposely keeping them in a cast or keeping them away from extension so that the fat pad fills in with this fibrotic reaction. ACL surgeries, acutes and chronics, were equally divided in the group, extraarticulars and intraarticulars were both represented, and arthroscopic and nonarthroscopic were equally represented. We did not find a trend, other than the ACL surgery being the number one cause.