A swan-neck or boutonniere deformity occurs in approximately half of patients with rheumatoid arthritis. The cause of boutonniere deformity is chronic synovitis of the proximal interphalangeal joint. Swan-neck deformity may be caused by synovitis of the metacarpophalangeal, proximal interphalangeal, or distal interphalangeal joints. Numerous procedures are available for the operative correction of these finger deformities. The choice of surgical procedure is dependent on accurate staging of the deformity, which is based on the flexibility of the proximal interphalangeal joint and the state of the articular cartilage. The patient’s overall medical status and corticosteroid use, the condition of the cervical spine, the need for operative treatment of large joints, and the presence of deformities of the wrist and metacarpophalangeal joints must also be considered when planning treatment. In the later stages of both deformities, soft-tissue procedures alone may not result in lasting operative correction.

The hip, knee, shoulder, and elbow joints are commonly involved in rheumatoid arthritis. The effect of arthritis on large joints influences the ability of a patient to care for himself or herself, to carry out activities of daily living, and to participate in postoperative hand rehabilitation and therefore may necessitate operative correction of large-joint arthritis before operative correction of finger deformities. For instance, the hand and wrist must be able to withstand the added upper-extremity forces required to use walking aids during rehabilitation of the lower extremity. Stabilization of the wrist or thumb metacarpophalangeal (MCP) or interphalangeal joints by arthroplasty facilitates the patient’s rehabilitation after hip or knee arthroplasty by allowing pain-free use of walking supports, such as walkers, crutches, and canes.

Oral corticosteroid administration presents special concerns, including the risk of postoperative wound infection and an increase in overall healing time of soft tissues. If a general anesthetic is required for a patient taking oral corticosteroid medication, the preoperative administration of intravenous corticosteroids is indicated, followed by a tapering-dose regimen postoperatively.

**Clinical Evaluation of the Wrist and Hand**

Because collapse of the carpus into supination, volar translation, and ulnar translocation occurs often and is most easily noted on visual inspection, examination for active synovitis and deformity of the radiocarpal joints and the distal radioulnar joints (DRUJs) should be carried out routinely. Inspection for dorsal subluxation of the distal ulna, rupture of the extensor carpi ulnaris tendon and the extensor tendons of the small and ring fingers (the caput ulnae syndrome), and rupture of the extensor pollicis longus is essential. Evaluation of the MCP joints for active synovitis and the presence of ulnar drift and volar subluxation is also critical. Wrist, DRUJ, and MCP joint abnormalities are important not only because they may be of greater functional significance to the patient than the finger deformities, but also because they may directly contribute to the development of those deformities by altering the extent and direction of in vivo forces generated by flexor and extensor muscle contraction in the fingers. In addition, wrist and MCP joint deformities may influence the longevity of corrections of finger deformities.

**Basic Assessment of Finger Deformities**

Once the physical examination of the large joints, wrist, and hand has been completed, clinical examination of the fingers is carried out. The resting positions of the proximal interphalangeal (PIP) and distal interphalangeal (DIP) joints are noted, and the active and passive ranges of motion of each joint are recorded. The initiation of flexion is observed while examining the active ranges of motion, because difficulty in initiating PIP joint flexion is associated with early swan-neck deformity.

Involvement of the PIP and DIP joints in the rheumatoid process is assessed. Bulging of the joint indicates the presence of an effusion and implies possible capsular attenuation and laxity. Crepitus with joint motion indicates articular cartilage damage. In cases of boutonniere deformity, the skin is assessed for tightness volarly over the PIP joint and attenuation over the dorsum of the PIP joint. In cases of swan-neck deformity, the skin is examined for tightness over the PIP joint dorsally and volar skin attenuation.

Flexor tenosynovitis is indicated by palpable mobile fullness in the distal volar forearm. Flexor tenosynovitis of the finger is identified on the basis of three findings: swelling, discrepancy between active and passive motion of the finger, and palpable crepitus along the course of the flexor tendon on active and passive flexion of the digit. Extensor tenosynovitis is signaled by the presence of persistent swelling and crepitus along the course of the long extensor tendons both proximal and distal to the extensor retinaculum.

Flexor or extensor tendon tenosynovitis or rupture at the wrist level must be identified, as correction of the finger deformity without concomitant operative correction of wrist synovitis and tendon rupture does not increase active range of motion. Proximal tendon ruptures are diagnosed by visual inspection (i.e., if proximal muscle contraction is seen without finger motion) or palpation (i.e., if tendon excursion is absent distal to the site of a rupture). The tenodesis effect of wrist flexion and extension on digital motion is assessed to rule out tendon ruptures at the wrist level. If passive wrist flexion fails to elicit extension of the fingers, rupture of the long extensor tendons should be suspected. Adhesion of a ruptured tendon to the surrounding tissue proximal to the wrist may decrease the accuracy of this test.

The Bunnell test of intrinsic tendon tightness is performed in all fingers of patients with rheumatoid arthritis. It has special relevance in patients with early swan-neck deformities, as it assesses the relative contribution of tight intrinsics to the genesis of the deformity. While the MCP joint is held in the extended position, the degree of resistance to passive flexion of the PIP joint is determined (Fig. 1, A). Increased resistance to passive PIP joint flexion signifies relative shortening of the intrinsic muscle-tendon unit.
Tightness of the oblique retinacular ligament in digits with early boutonniere deformity is also evaluated with another type of intrinsic tightness test. The PIP joint is held in maximum extension by the examiner, and the degree of resistance to passive flexion of the DIP joint is evaluated (Fig. 1, B).

Etiology and Classification of Swan-Neck Deformity

Swan-neck deformity is characterized by hyperextension at the PIP joint and flexion of the DIP joint (Fig. 2). The deformity, which may be flexible or fixed, is associated with varying degrees of destruction of the PIP joint.

Synovitis of the PIP joint with a variable degree of tenosynovitis within the finger flexor sheath is the most common cause of swan-neck deformity. Attenuation of the volar plate, collateral ligaments, and flexor digitorum superficialis insertion allows the PIP joint to hyperextend due to extensor mechanism forces. These forces are accentuated by intrinsic muscle contractures and intrinsic tightness due to MCP joint flexion and volar subluxation. Synovitis in the DIP joint with attenuation or rupture of the terminal tendon of the extensor mechanism concentrates the extensor forces at the PIP joint, which can initiate or accentuate a swan-neck deformity. Synovitis in the PIP joint causes attenuation of the volar plate, which acts as a check-rein to PIP joint hyperextension. Synovitis also produces attenuation of the transverse retinacular ligaments, which are responsible for maintaining the normal position of the lateral bands. When the transverse retinacular ligaments become attenuated, the lateral bands can subluxate dorsal to the axis of PIP joint rotation and produce a constant and accentuated PIP joint extension force. Eventually, the extensor mechanism over the PIP joint, especially the triangular ligament, and the dorsal skin become contracted, resulting in a fixed hyperextension contracture of the joint. Articular destruction secondary to persistent synovitis, as well as altered joint-contact mechanics, contributes to a fixed and occasionally painful deformity.

Nalebuff and Zancolli have each devised comprehensive classification schemes for swan-neck deformity. The classification devised by Nalebuff is very helpful in selecting a treatment option for operative correction of the deformity. Zancolli’s classification is instructive in determining the underlying cause of the deformity.

Nalebuff described four types. Type I is characterized by a flexible hyperextension deformity of the PIP joint. In type II deformity, tightness of the intrinsic muscles causes limitation of PIP joint flexion when the MCP joint is maintained in extension. Type III deformity is characterized by limited PIP joint motion in all MCP joint positions due to the inability of the lateral bands to translate volar to the axis of rotation of the PIP joint. Type IV deformity is characterized by limited PIP joint flexion in all MCP joint positions due to the inability of the lateral bands to translate volar to the axis of rotation of the PIP joint. In type IV deformity, there is destruction of the articular surface of the PIP joint.

Zancolli’s classification scheme focuses on three potential causes of deformity. The first involves rela-
tive shortening of the long digital extensor tendons secondary to MCP joint subluxation or dislocation, wrist flexion deformity, or primary mallet-finger deformity, which causes extrinsic extensor tightness, an increased extension moment at the PIP joint, and PIP joint hyperextension. The second involves relative shortening of the digital intrinsic muscles (intrinsic tightness) secondary to MCP joint subluxation or dislocation, which results in translocation of the lateral bands dorsal to the axis of rotation of the PIP joint and the development of a hyperextension deformity. The third cause involves weakening and attenuation of the volar plate and collateral ligaments of the PIP joint and destruction of the articular cartilage, which leads to the development of a swan-neck deformity of the PIP joint.

Treatment of Swan-Neck Deformity

The treatment of swan-neck deformities begins with simple methods for the early stages (as classified according to Nalebuff) and proceeds to more complex procedures.

Type I

The goals of treatment of the flexible swan-neck deformity are to prevent PIP joint hyperextension and to improve the arc of PIP joint flexion. In its early stages, when a correctable swan-neck deformity is associated with minimal synovitis of the PIP joint, nonoperative treatment with the use of external splints, such as a figure-of-eight ring splint, is advocated to prevent PIP joint hyperextension. This technique is appropriate if inability to initiate PIP joint flexion from the position of hyperextension is the difficulty or if the deformity is not disabling.

An extensor tendon subluxation at the level of the MCP joint or a flexion contracture of the MCP joint should be corrected concurrent with or prior to surgical correction of the swan-neck deformity. A wrist flexion contracture may also contribute to the swan-neck deformity, and its correction may be required to balance the hand.

The primary cause of the flexible swan-neck deformity must be established before initiating operative treatment. Although PIP joint synovitis and weakening of the volar PIP structures are the most common findings, DIP joint synovitis can also be a primary cause of swan-neck deformity, due to the transfer and concentration of extension forces at the PIP joint.

DIP Fusion

If full PIP joint flexibility is preserved in a primary rheumatoid mallet finger, fusion of the DIP joint is an option. Arthrodesis of the DIP joint can be performed with a low-profile cannulated screw or other headless screw for secure fixation. A headless screw can be buried beneath the osseous surface of the tip of the distal phalanx, which helps to avoid irritation of the fingertip on fine object grasping. Alternatively, Kirschner-wire fixation, with or without the addition of a tension-band wire, or lag-screw fixation with a 2.0- or 2.4-mm-diameter screw may be used.

The DIP joint is approached dorsally through an incision centered over the joint. The germinal matrix of the nail proximal to the eponychium must be avoided. If headless-screw fixation is chosen, the articular cartilage is excised with a rongeur, and the medullary cavities of the middle and distal phalanges are manually drilled. After the size of the screw has been determined, a transarticular headless screw is inserted under fluoroscopic guidance through a stab wound in the tip of the finger 1 to 2 mm volar to the nail plate with the joint in full extension (Fig. 3). A lag effect is achieved across the arthrodesis site. A mallet-finger splint is worn until fusion is noted radiographically. The PIP joint is not immobilized, and the patient is encouraged to actively flex and extend.

Fig. 2 Swan-neck deformity. Left, Terminal tendon rupture may be associated with synovitis of the DIP joint, leading to DIP joint flexion and subsequent PIP joint hyperextension (A). Rupture of the flexor digitorum superficialis tendon may occur due to infiltrative synovitis, which may lead to decreased volar support of the PIP joint and subsequent hyperextension deformity (B). Right, Lateral-band subluxation dorsal to the axis of rotation of the PIP joint (C), contraction of the triangular ligament (D), and attenuation of the transverse retinacular ligament (E) are depicted.
extend the PIP joint during the postoperative period.

If a box-wire technique is preferred, 26- or 28-gauge cerclage wires are passed through the distal aspect of the middle phalanx and the proximal aspect of the distal phalanx. One wire is oriented in the frontal plane; the second, in the sagittal plane. The wires are tightened simultaneously, and fixation may be augmented by a transarticular Kirschner wire to control translation. Some surgeons prefer transarticular pinning of the DIP joint with two Kirschner wires as the sole method of fixation, in order to control both translation and rotation.

**PIP-Joint Flexor Tenodesis**

Often, the major difficulty noted by the patient with a flexible swan-neck deformity is an inability to initiate active PIP joint flexion due to a hyperextended resting posture. The loss of volar support at the PIP joint is addressed by means of a flexor tenodesis. Oblique retinacular ligament reconstruction or volar-skin dermodesis are both options. Alternatively, flexor tenodesis can reliably establish a sturdy checkrein against PIP joint hyperextension that does not attenuate over time. While not necessarily improving the total arc of flexion of the PIP joint, tenodesis of the PIP joint in 20 degrees of flexion eliminates locking and ensures that the initiation of digital flexion at the PIP joint is improved and that the arc of PIP joint motion remains functional.

Our preferred operative approach is a Bruner incision extending from the proximal digital crease to the distal digital crease. A triangular skin flap is raised to identify the digital sheath, and the neurovascular bundles are protected. The flexor sheath from the distal end of the A2 pulley to the proximal end of the A4 pulley is identified and opened as a flap with the base along the bone attachment of the flexor sheath. The flexor digitorum superficialis tendon is identified within the digital sheath, and one slip of the tendon is sectioned proximally at the level of the decussation. The sectioned tendon end is passed through a transverse incision in the distal aspect of the A2 pulley and sutured back on itself. Immobilization with a dorsal block splint in 20 degrees of PIP joint flexion is maintained for 6 weeks. Passive and active PIP- and DIP-joint flexion exercises are begun at 3 weeks.

A less extensive approach may be undertaken as well (Brian D. Adams, MD, written communication, November 1998). A midlateral incision is made over the PIP joint. The sheath is opened at the proximal edge of the A4 pulley. With the finger in the flexed position, the slip of the flexor digitorum superficialis tendon is extracted and sectioned proximally at the level of the decussation. The slip is then sutured to the outside of the flexor sheath along its volar-midlateral aspect. The postoperative protocol is similar to that previously described.

**Lateral-Band Tenodesis**

Rerouting of the lateral band volar to the axis of rotation of the PIP joint, as described by Littler (volar to Cleland’s ligament) or Zancolli (through the flexor tendon sheath), may be used to treat the early flexible swan-neck deformity caused by PIP joint synovitis. The effect is similar to that achieved with tenodesis of the flexor digitorum superficialis, in that a volar checkrein against PIP joint hyperextension is provided. The choice between lateral-band tenodesis and flexor tenodesis is a matter of personal preference.

With this technique, the extensor tendon apparatus overlying the PIP joint is approached dorsally through a longitudinal curvilinear incision. Dissection is carried volarily, and Cleland’s ligament is divided to expose the flexor sheath. The neurovascular bundle is protected, and the dorsally subluxated lateral band is dissected free of its dorsal attachments to the central extensor tendon and the proximal aspect of the triangular ligament. The lateral band is carried volar to the axis of rotation of the PIP joint with passive flexion of the PIP joint. It is not detached from either its proximal or its distal attachments.

A wide dorsally based flap of flexor sheath is created at the level of the PIP joint. The incision in the sheath is made under direct vision close to the midline of the digit.
The volarly displaced lateral band is placed under the raised flap of flexor tendon sheath. The flap is replaced in its anatomic position and then repaired with nonabsorbable sutures.

Free gliding of the lateral band is confirmed by gentle proximally and distally directed traction on the translocated lateral band. The joint is maintained in flexion by an assistant during closure, and the dressing is applied. A dorsal blocking splint is used to prevent PIP joint extension beyond 30 degrees of flexion for 4 weeks. Digital flexion is encouraged in the postoperative period, but full active PIP joint extension is not allowed until 6 weeks postoperatively.

**Type II**

In type II swan-neck deformity, passive and active flexion of the PIP joint are limited when the MCP joint is in extension due to tightness of the intrinsic muscles. Arthritis in the MCP joint is frequently associated with intrinsic muscle tightness. Thus, to obtain and maintain correction of a swan-neck deformity, MCP joint arthroplasty with or without intrinsic release may be required.

In the absence of deforming MCP joint arthritis, isolated intrinsic release may be indicated. The intrinsic tendon, which inserts into the lateral band at the level of the base of the proximal phalanx, is approached through a dorsal incision centered over the MCP joint and the proximal phalanx. The central extensor tendon, ulnar sagittal band, and ulnar intrinsic tendons are exposed. The lateral band is isolated, and a 1-cm segment of lateral band with attached sagittal band fibers is excised.4 Release of the ulnar intrinsic tendon may diminish ulnar drift at the MCP joint and may correct the deforming forces that cause swan-neck deformity.

**Type III**

Type III MCP swan-neck deformity is characterized by decreased PIP joint flexion in all positions of MCP joint flexion and extension. This type of swan-neck deformity cannot be addressed solely by any one of the procedures described previously. The lateral bands have become fixed dorsal to the axis of rotation of the PIP joint, and a PIP-joint soft-tissue contracture is present. The authors prefer a technique that includes release of the lateral bands from their dorsal attachments to the central tendon and to the triangular ligament, with subsequent translocation of the lateral bands volar to the axis of rotation of the PIP joint. This is combined with a dorsal PIP joint capsulectomy, collateral ligament release, and tenolysis of the extensor tendon over the dorsum of the proximal phalanx.8,9

An oblique incision over the dorsum of the middle phalanx is continued as a curvilinear longitudinal dorsal incision over the PIP joint. Skin flaps are raised to expose the dorsally subluxated lateral bands, the central extensor tendon over the dorsum of the proximal phalanx, and the lateral aspect of the PIP joint. Both lateral bands are then released along their dorsal margins throughout the length of the incision from over the middle of the proximal phalanx to their confluence over the dorsum of the middle phalanx. The central extensor tendon is dissected free of the underlying dorsum of the proximal phalanx as well as the dorsal PIP joint capsule.

The dorsal PIP joint capsule is then resected, and the radial and ulnar collateral ligaments are released from the proximal phalanx beginning dorsally. Sufficient collateral ligament release is carried out to passively flex the joint to 90 degrees. The lateral bands will come to lie volar to the axis of PIP joint rotation with passive flexion; therefore, fixation of the lateral bands volar to the axis of rotation of the PIP joint is not required.

Palmar traction lysis of the flexor tendons is carried out in addition to the dorsal releases if flexor tendon tenosynovitis is present. This is done through an incision over the distal volar forearm. The desired tendon is isolated and pulled in a proximal direction to lyse any restricting adhesions proximal to the finger. Transarticular PIP pinning with the joint flexed to 20 degrees is maintained for 2 weeks postoperatively for severe deformities.

**Type IV**

In type IV swan-neck deformity, there is a fixed hyperextension deformity of the PIP joint with advanced destruction of the articular cartilage of the PIP joint. Soft-tissue procedures alone neither relieve pain reliably nor restore digital motion and function. Arthrodesis or flexible implant arthroplasty are reliable options when joint destruction has occurred.

**Arthrodesis**

Arthrodesis of the PIP joint in a position of flexion is the authors’ preferred technique for type IV deformities.10 Fusion of the PIP joint provides relief of pain and allows digits to readily conform when medium- and large-size objects are grasped. The PIP joint is approached through a dorsal curvilinear incision, and the central slip is detached from the base of the middle phalanx. Sufficient collateral ligament is resected to allow delivery of the head of the proximal phalanx and the base of the middle phalanx into the wound. The remaining articular surfaces and subchondral bone are resected with use of a rongeur, and the bone ends are apposed in 30 degrees of flexion.
A tension band over two Kirschner wires provides adequate fixation for the PIP joint fusion. A transverse hole is drilled through the dorsal aspect of the middle portion of the diaphysis of the middle phalanx. A 26-gauge flexible wire is passed through. Two 0.035-inch Kirschner wires are drilled obliquely in a retrograde fashion through the distal end of the proximal phalanx and then antegrade into the middle phalanx with the PIP joint held in 25 to 40 degrees of flexion (index finger at 25 degrees, increasing at 5-degree increments for the long, ring, and small fingers). The wires are inserted into the medullary cavity of the middle phalanx, parallel to each other in both the frontal and the sagittal plane. The flexible wire is bent in a figure-of-eight fashion and then passed around the proximal Kirschner wires. The wire is then tightened in a single knot.

Intraoperative fluoroscopy is used to confirm proper positioning of the arthrodesis and the internal fixation. The Kirschner wires are cut, the extensor mechanism is repaired, and the skin is closed. An external splint is applied to the PIP joint and is worn until the fusion is solid. Active- and passive-motion rehabilitation of the DIP joint is begun at the time of suture removal.

**PIP Arthroplasty With a Silicone Implant**

A volar approach is made through a Bruner incision centered over the PIP joint flexion crease. The digital neurovascular bundles are retracted, and the flexor sheath is opened from immediately distal to the A2 pulley to immediately proximal to the A4 pulley. The flexor digitorum superficialis and profundus tendons are retracted, and the volar plate is released from its insertion into the proximal phalanx. The joint surfaces are resected, taking care to preserve the attachments of the collateral ligaments and the central slip, and the medullary cavities are prepared (with the use of curettes, rasps, or a burr) to accept the prosthesis. Sufficient bone is removed to prevent the prosthesis from assuming a hyperextended position, and the flexor sheath is closed. Protected range-of-motion exercises commence on suture removal.

**Etiology and Classification of Boutonniere Deformity**

The boutonniere deformity (Fig. 4) is characterized by a flexion posture of the PIP joint and a hyperextension posture of the DIP joint. The deformity may be flexible or fixed. Unlike the swan-neck deformity, which may be caused by abnormalities at the MCP, PIP, and (occasionally) DIP joints, the inciting event is persistent synovitis of the PIP joint, which causes attenuation of the central slip, transverse retinacular ligaments, and triangular ligament. Due to weakening of these structures, the lateral bands subluxate volar to the axis of rotation of the PIP joint and become PIP joint flexors rather than extensors. With persistence of PIP joint flexion, the volar plate, collateral ligaments, and oblique retinacular ligaments become contracted, leading to a fixed contracture of the joint.

Both Nalebuff and Zancolli have described classification schemes that characterize the deformity and guide treatment. Nalebuff's system has three stages, based on the passive correctability of the PIP joint flexion deformity and the condition of the articular surfaces of the PIP joint. Stage 1 boutonniere deformity is characterized by synovitis of the PIP joint and a slight, fully correctable extensor lag. Stage 2 deformity consists of a marked flexion deformity of the
PIP joint that is either flexible or fixed. Stage 3 deformity is characterized by destruction of the PIP joint.

Zancolli\textsuperscript{5} distinguished boutonniere deformity due to rheumatoid arthritis from traumatic boutonniere deformity. In his system, there are two types of boutonniere deformities: those with passively correctable PIP joint flexion and DIP joint extension deformities and those with fixed deformities. As deformities progress from flexible to fixed, they become far less likely to respond to splinting or soft-tissue reconstructions.

**Treatment of Boutonniere Deformity**

Early boutonniere deformity may be treated nonoperatively with low-profile extension splinting of the PIP joint. A mild boutonniere deformity does not usually cause functional impairment, because it does not limit grasp of moderate- and large-size objects. Synovectomy of the PIP joint may be indicated in a patient with a mild, progressive boutonniere deformity and persistent synovitis that is unresponsive to oral medication or local injection of corticosteroids (Fig. 5).

Hyperextension of the DIP joint causes functional limitation when it prevents the volar aspect of the fingertip from coming into contact with an object on digital flexion. Operative correction of a DIP-joint hyperextension deformity, a straightforward solution to this problem, is most easily accomplished by section of the terminal tendon over the middle phalanx.\textsuperscript{13}

**Terminal Tendon Release**

Release of the terminal tendon\textsuperscript{13} is carried out through a longitudinal incision over the middle phalanx and DIP joint. The terminal extensor tendon is exposed and sharply divided over the distal aspect of the middle phalanx at a level that is proximal to the insertion of the fibers of the oblique retinacular ligament. Although a slight mallet deformity may occur, the deformity is well tolerated, as the new position improves the patient’s ability to grasp.

**Central-Slip Reconstruction**

For more severe boutonniere deformities in which there is a well-preserved PIP joint cartilage (Nalebuff stage 2 deformities), central-slip reconstruction by means of an anatomic technique, as advocated by Urbaniak\textsuperscript{14} and Flatt,\textsuperscript{15} is indicated. A curvilinear incision is made over the dorsum of the PIP joint, and the central slip and lateral bands are identified. The redundant portion of the central slip over the PIP joint is excised, and the tendon is advanced and reattached to the base of the middle phalanx. The lateral bands are mobilized from their volarly displaced position by releasing the transverse retinacular ligament attachments along the volar margin of the lateral bands. The lateral bands are repositioned dorsally over the PIP joint and sutured to each other. A terminal tenotomy, as previously described, is done not only to correct a preexisting DIP-joint hyperextension deformity, but also to prevent a secondary deformity due to the force from the repositioned bands.

**Arthrodesis**

If articular destruction is evident or if a severe fixed flexion contracture is present, even without severe articular changes, arthrodesis of the PIP joint in 25 to 40 degrees of flexion, as described for a swan-neck deformity, is a reliable option for managing pain and treating deformity. Flexible implant arthroplasty of the PIP joint combined with release of the terminal extensor tendon is a less reliable option, due to marked attenuation of the dorsal extensor mechanism.

**Results**

There is a relative lack of clinical studies evaluating the results of operative treatment of swan-neck and boutonniere deformities in patients with rheumatoid arthritis. In addition, the number of surgical options for the correction of rheumatoid finger deformities is relatively limited. In a report on the results of operative treatment of Nalebuff type III swan-neck and stage 2 boutonniere deformities, Kiefhaber and Strickland\textsuperscript{9} concluded that the results of soft-tissue procedures for swan-neck deformity are more consistent than those for boutonniere deformity. The initial postoperative gain in arc of flexion after lateral band release, extensor tenolysis, and PIP-joint dorsal capsulectomy for type III swan-neck deformity decreased by 17 degrees during the first year postoperatively. Recurrence of deformity was noted, signifying the transient nature of correction of
the PIP-joint hyperextension deformity in some fingers. Despite deterioration in results over time, the total arc of PIP joint motion was shifted into flexion, thus improving grasp of small and large objects.

Those authors also found that operative correction of boutonniere deformity by reconstruction of the extensor mechanism was less reliable than operative correction of swan-neck deformities, as the deterioration over time was greater. Only 4 of 19 patients could extend their PIP joints beyond 20 degrees of flexion, and 11 of 19 patients had extension deficits of 45 degrees or more at the PIP joint. Currently, the authors recommend arthrodesis of the PIP joint in digits with severe rheumatoid boutonniere deformities.

Tonkin et al published two reviews of patients treated for swan-neck deformity due to various causes and in various stages.[6,17] The procedure of choice for swan-neck deformity was lateral-band translocation, as described by Zancolli. As the patient population from which the first study was derived was small, the causes were diverse, and the follow-up was short, the insight provided by this study is limited.

In the other clinical study, 31 stiff swan-neck deformities, with or without PIP joint destruction, were treated by synovectomy, lateral-band release and translocation, and dorsal capsulectomy. The improvement in the position of the arc of motion into flexion was similar to that reported by Kiefhaber and Strickland for digits without joint destruction.

### Summary

A successful operative result in the correction of a swan-neck or boutonniere deformity in a rheumatoid hand depends on a complete preoperative examination, correct staging of the deformity and proper timing of operative treatment. Although operative correction frequently reduces pain and increases function, the surgeon must remain realistically reserved as to expectations regarding the long-term results of procedures used to correct these deformities.

### References


