

Swan Neck Deformity: Pathophysiology, Classification, and Contemporary Management Strategies — A Narrative Review

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ABSTRACT

Swan neck deformity (SND) is a complex digital deformity characterized by hyperextension of the proximal interphalangeal (PIP) joint associated with flexion of the distal interphalangeal (DIP) joint. Although classically associated with rheumatoid arthritis, SND may also arise from traumatic injuries, neurological disorders, connective tissue diseases, congenital abnormalities, and chronic tendon imbalance. The deformity represents a multifactorial disruption of the intricate biomechanical equilibrium between intrinsic and extrinsic tendon systems, ligamentous stabilizers, and articular structures of the finger. Progressive alteration of these relationships may lead to pain, stiffness, impaired dexterity, diminished grip strength, and substantial functional disability.

The pathogenesis of SND involves several mechanisms, including volar plate laxity, intrinsic muscle tightness, terminal extensor tendon dysfunction, dorsal migration of the lateral bands, and chronic synovitis. Understanding the underlying etiology and stage of deformity is essential for establishing an appropriate treatment strategy. Multiple classification systems have been proposed, with the Nalebuff classification remaining the most widely used in clinical practice because of its therapeutic implications.

Management ranges from conservative interventions, such as splinting and hand therapy, to a broad spectrum of surgical procedures aimed at restoring extensor balance and joint stability. Surgical options include flexor digitorum superficialis tenodesis, lateral band translocation, central slip reconstruction, volar plate advancement, intrinsic release, arthroplasty, and arthrodesis. Selection of treatment depends on deformity flexibility, joint integrity, patient functional demands, and associated systemic disease.

Recent advances in hand surgery have improved understanding of extensor mechanism biomechanics and facilitated the development of individualized reconstructive approaches. Nevertheless, treatment outcomes remain variable, particularly in advanced deformities associated with inflammatory arthropathies. This narrative review summarizes current evidence regarding the anatomy, biomechanics, pathophysiology, classification, clinical evaluation, and modern management of swan neck deformity, with emphasis on surgical decision-making and contemporary reconstructive techniques.

KEYWORDS: Swan neck deformity; proximal interphalangeal joint; extensor mechanism; rheumatoid arthritis; hand surgery; tendon imbalance; reconstructive surgery..

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INTRODUCTION

Swan neck deformity (SND) represents one of the most recognizable and functionally significant deformities affecting the hand. The condition is characterized by hyperextension of the proximal interphalangeal (PIP) joint combined with flexion of the distal interphalangeal (DIP) joint, resulting in a characteristic zig-zag deformity that compromises coordinated finger motion and fine motor function. Although traditionally associated with rheumatoid arthritis (RA), SND encompasses a heterogeneous spectrum of pathological processes involving tendon imbalance, ligamentous insufficiency, joint destruction, and neuromuscular dysfunction.

The biomechanical complexity of the finger extensor apparatus explains the diverse etiological mechanisms capable of producing this deformity. Physiological digital motion depends on a precise equilibrium between intrinsic muscles, extrinsic flexors and extensors, retinacular ligaments, volar plate integrity, and articular congruity. Disruption at any level of this interconnected system may progressively alter force transmission across the PIP and DIP joints, ultimately generating the characteristic posture of SND.

Historically, the understanding of SND evolved substantially during the twentieth century alongside advances in hand surgery and rheumatology. Early descriptions focused predominantly on rheumatoid deformities; however, subsequent studies demonstrated that traumatic injuries, chronic mallet finger deformity, cerebral palsy, systemic lupus erythematosus, psoriatic arthritis, ligamentous laxity syndromes, and congenital conditions may all precipitate similar biomechanical alterations.

Clinically, patients may present with pain, stiffness, diminished pinch strength, impaired grasping ability, snapping phenomena, and progressive loss of dexterity. Functional impairment varies according to deformity rigidity, chronicity, associated joint degeneration, and

systemic disease activity. Early deformities may remain flexible and passively correctable, whereas longstanding cases frequently progress toward fixed contractures and irreversible articular destruction.

The management of SND remains challenging because no single reconstructive procedure adequately addresses all etiologies and stages of deformity. Treatment strategies must therefore be individualized according to patient age, functional demands, disease chronicity, degree of joint degeneration, and flexibility of the deformity.

This narrative review provides a comprehensive overview of the current understanding of swan neck deformity, focusing on anatomy, biomechanics, pathophysiology, classification systems, diagnostic evaluation, and modern treatment strategies.

METHODS

A narrative review of the literature was conducted using PubMed/MEDLINE, Scopus, Google Scholar, and Web of Science databases. Articles published between January 2000 and May 2026 were reviewed, with priority given to studies published between 2020 and 2026. Search terms included “swan neck deformity,” “proximal interphalangeal hyperextension,” “extensor mechanism,” “rheumatoid hand deformities,” “flexor digitorum superficialis tenodesis,” “lateral band translocation,” and “hand reconstruction.” Additional references were identified through manual review of bibliographies from relevant articles.

Included publications comprised narrative reviews, systematic reviews, clinical studies, biomechanical analyses, surgical technique articles, and classic landmark publications relevant to the pathophysiology and management of SND. Non-English studies without available translation and studies lacking clinical relevance were excluded.

SURGICAL ANATOMY OF THE FINGER

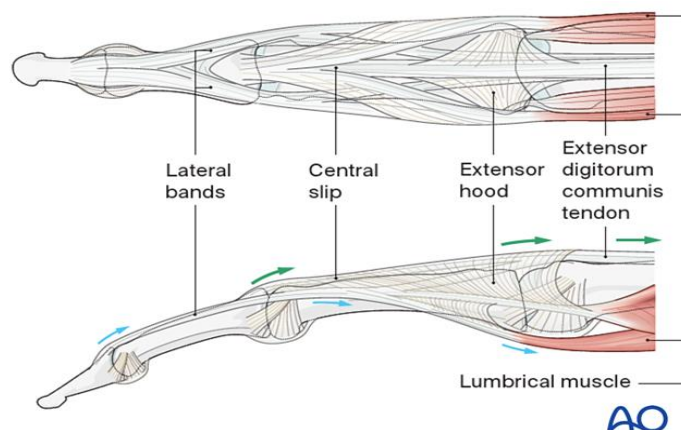


Figure1. Anatomy of the Finger Extensor Mechanism. Anatomical illustration of the finger extensor apparatus demonstrating the extensor digitorum communis tendon, central slip, lateral bands, extensor hood, and lumbrical contribution to coordinated interphalangeal motion.

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A detailed understanding of finger anatomy is essential for comprehending the pathogenesis and treatment of swan neck deformity. The coordinated movement of the finger depends on a sophisticated interaction between osseous structures, capsuloligamentous stabilizers, intrinsic musculature, and the extensor-flexor tendon systems.

The volar plate is a fibrocartilaginous structure located along the palmar aspect of the PIP joint. It functions as a primary restraint against hyperextension while simultaneously increasing joint stability during flexion. Attenuation or rupture of the volar plate is a critical pathological event in many forms of swan neck deformity.

The extensor mechanism of the finger is among the most intricate tendon systems in the human body. The extensor digitorum communis tendon divides into a central slip and two lateral bands at the level of the proximal phalanx. The central slip inserts onto the base of the middle phalanx and extends the PIP joint, while the lateral bands converge distally to form the terminal tendon.

Dynamic stabilization of the extensor mechanism is reinforced by the triangular ligament and transverse retinacular ligament. Imbalance between these structures may alter lateral band positioning and significantly modify force transmission across the interphalangeal joints.

The intrinsic muscles of the hand, including the lumbricals and interossei, contribute simultaneously to MCP flexion and IP extension. Intrinsic tightness, commonly observed in rheumatoid arthritis and neurological disorders, may increase extension forces across the PIP joint.

BIOMECHANICS OF THE EXTENSOR MECHANISM

Finger motion depends on a highly coordinated interaction between intrinsic and extrinsic musculotendinous units, retinacular stabilizers, and joint congruity. Swan neck deformity develops when this equilibrium becomes disrupted.

Several biomechanical pathways may initiate the deformity:

- Volar plate insufficiency permitting PIP hyperextension
- Intrinsic muscle tightness increasing dorsal extension forces
- Terminal tendon disruption shifting extensor force proximally
- Dorsal migration of lateral bands
- Chronic synovitis weakening capsuloligamentous stabilizers

One of the most important biomechanical concepts is the role of lateral band displacement. Under normal conditions, the lateral bands remain slightly volar to the axis of PIP rotation during flexion. Dorsal migration converts them into hyperextensors of the PIP joint.

Chronic mallet finger illustrates the interdependence of the extensor apparatus. Loss of terminal tendon continuity causes persistent DIP flexion and redistribution of extensor forces proximally, ultimately producing PIP hyperextension.

DEFINITION AND PATHOPHYSIOLOGY OF SWAN NECK DEFORMITY

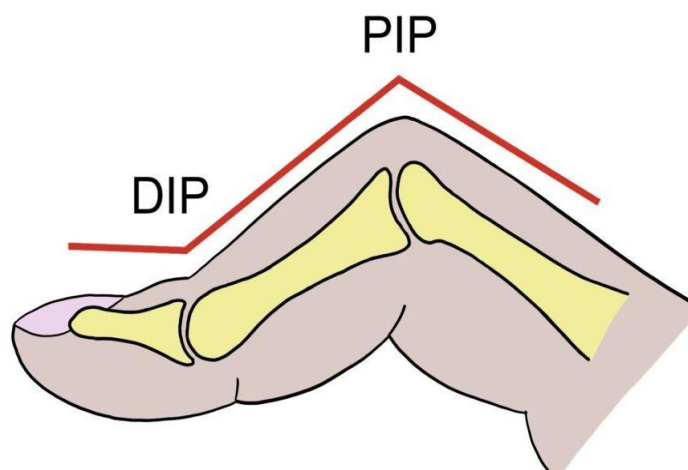


Figure 2. Schematic Representation of Swan Neck Deformity. Simplified schematic illustration demonstrating proximal interphalangeal joint hyperextension associated with distal interphalangeal flexion characteristic of swan neck deformity.

Swan neck deformity is defined as hyperextension of the PIP joint associated with flexion of the DIP joint. Although the clinical appearance is relatively uniform, the underlying pathophysiology is heterogeneous.

The deformity may originate through several mechanisms:

1. Primary PIP joint pathology

2. Primary DIP joint pathology
3. MCP joint abnormalities
4. Intrinsic muscle imbalance
5. Neurological dysfunction

Primary pathology at the PIP joint commonly involves attenuation or rupture of the volar plate. Distal pathology

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such as chronic mallet finger may also generate secondary SND through compensatory extensor redistribution.

Inflammatory arthropathies produce progressive weakening of ligaments, tendon attenuation, cartilage destruction, and capsular distention. Neurological disorders may generate deformity through intrinsic muscle spasticity and abnormal motor control.

As the deformity progresses, secondary structural changes develop throughout the finger. Chronic hyperextension stretches the volar plate and collateral ligaments, leading to worsening instability and eventual fixed deformity.

ETIOLOGY



Figure 3. Clinical Appearance of Swan Neck Deformity in Rheumatoid Arthritis. Clinical photograph demonstrating characteristic hyperextension of the proximal interphalangeal joint associated with distal interphalangeal flexion in a patient with advanced rheumatoid arthritis.

Rheumatoid Arthritis

Rheumatoid arthritis remains the condition most classically associated with SND. Chronic inflammatory synovitis progressively weakens periarticular ligaments, attenuates the volar plate, and damages tendon structures.

Traumatic Causes

Trauma constitutes another important cause of SND. Volar plate injuries, untreated mallet finger deformities, flexor tendon injuries, and intra-articular fractures may disrupt normal tendon balance.

Mallet Finger

Chronic mallet finger represents one of the most common pathways toward secondary SND. Terminal extensor tendon disruption prevents active DIP extension and causes persistent distal flexion.

Neurological Disorders

Neurological disorders such as cerebral palsy may produce SND through intrinsic muscle spasticity and abnormal motor control.

Connective Tissue Disorders

Ligamentous laxity syndromes such as Ehlers-Danlos syndrome predispose patients to digital instability and progressive hyperextension deformity.

Psoriatic Arthritis

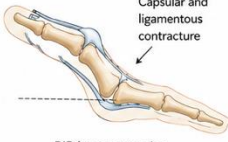
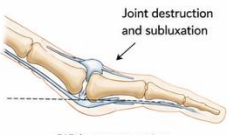
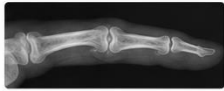
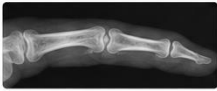
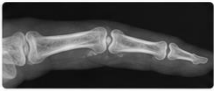
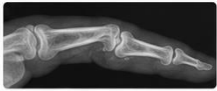
Psoriatic arthritis may generate SND through chronic synovitis, tendon imbalance, and joint destruction.

Congenital Causes

Congenital SND is relatively uncommon but may occur secondary to intrinsic muscle imbalance or generalized ligamentous laxity.

CLASSIFICATION SYSTEMS

Figure 4. Nalebuff Classification of Swan Neck Deformity

	STAGE I Flexible deformity	STAGE II Intrinsic tightness	STAGE III Fixed deformity	STAGE IV Severe fixed deformity with joint destruction
Anatomic changes	 Lateral band dorsal migration Volar plate attenuation PIP hyperextension (reducible) DIP flexion	 Intrinsic tightness (shortening) PIP hyperextension (partially reducible) DIP flexion	 Capsular and ligamentous contracture PIP hyperextension (not passively correctable) DIP flexion	 Joint destruction and subluxation PIP hyperextension (fixed) DIP flexion (fixed)
Clinical appearance				
Passive correction	✓ Full correction possible	☾ Partial correction possible	✗ No correction possible	✗ No correction possible
Radiographic appearance (Key findings)	 Normal joint alignment Preserved joint spaces	 Early osteopenia Preserved joint spaces	 Joint space narrowing Marginal erosions	 Joint destruction Subluxation/deformity Erosions and osteophytes
Key clinical features	• Deformity is supple • No intrinsic tightness • Minimal or no joint changes • Usually early and painless	• Intrinsic tightness present • Deformity partially correctable • Minimal joint changes • May have mild symptoms	• Fixed PIP hyperextension • Capsular/ligament contracture • Pain or instability may be present • Functional limitation	• Fixed deformity • Pain and instability common • Significant joint destruction • Marked functional impairment

PIP: proximal interphalangeal joint; DIP: distal interphalangeal joint.

Figure 4. Nalebuff Classification of Swan Neck Deformity. Diagrammatic representation of the four stages of the Nalebuff classification demonstrating progression from flexible proximal interphalangeal hyperextension to advanced fixed deformity with severe joint destruction and functional impairment.

Nalebuff Classification

Type I

Flexible deformity with preserved joint mobility.

Type II

Intrinsic tightness with limited PIP flexion.

Type III

Fixed contracture with early articular degeneration.

Type IV

Severe deformity with advanced joint destruction.

The Nalebuff classification remains highly valuable because it directly correlates with treatment planning and prognosis.

CLINICAL EVALUATION

Clinical evaluation should identify the primary pathological mechanism, determine deformity flexibility, evaluate associated joint degeneration, and quantify functional impairment.

Important elements include:

- Duration of deformity
- History of trauma
- Presence of inflammatory disease
- Neurological disorders
- Previous surgery
- Functional limitations

Physical examination typically reveals hyperextension of the PIP joint and flexion of the DIP joint.

The deformity should be evaluated for:

- Passive correctability
- Intrinsic tightness
- Tendon integrity
- MCP instability
- Synovitis
- Grip strength

The Bunnell test may help identify intrinsic muscle tightness.

IMAGING ASSESSMENT

Plain radiographs remain the initial imaging modality of choice. Imaging may reveal:

- Joint degeneration
- PIP hyperextension
- DIP flexion
- Subluxation
- Erosive changes
- Chronic instability

Ultrasound and MRI may provide additional information regarding tendon integrity, volar plate injuries, and synovitis.

CONSERVATIVE TREATMENT

Conservative management remains an important component of treatment, particularly in early-stage flexible deformities.

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Splinting

Splinting represents the cornerstone of nonoperative treatment. Common options include:

- Figure-of-eight splints
- Silver ring splints
- Extension-block splints

Hand Therapy

Specialized hand therapy focuses on:

- Range-of-motion exercises
- Intrinsic stretching
- Tendon gliding
- Strengthening exercises

Pharmacological Treatment

In inflammatory conditions such as rheumatoid arthritis, systemic disease control is essential.

Surgical Treatment

Surgical management is indicated when conservative treatment fails or when progressive deformity compromises hand function.

Flexor Digitorum Superficialis Tenodesis

FDS tenodesis remains one of the most commonly performed procedures for flexible SND. One slip of the FDS tendon is used to create a volar restraint against PIP hyperextension.

Lateral Band Translocation

Lateral band procedures reposition the lateral bands volarly to restore balanced extension forces.

Volar Plate Advancement

This procedure restores static restraint against hyperextension and improves joint stability.

Intrinsic Release

Intrinsic release decreases excessive extension forces transmitted through the extensor hood.

Central Slip Reconstruction

Central slip reconstruction may be indicated in selected cases involving extensor mechanism disruption.

Arthroplasty

PIP arthroplasty aims to relieve pain, preserve motion, and improve alignment.

Arthrodesis

Arthrodesis remains the most reliable salvage option for severe fixed deformities with advanced joint destruction.

OUTCOMES AND COMPLICATIONS

Outcomes vary according to etiology, deformity severity, surgical technique, and patient-related factors.

Common postoperative complications include:

- Recurrence
- Stiffness
- Residual deformity
- Implant failure
- Nonunion

Patients with early flexible deformities generally achieve superior functional recovery compared with those presenting with chronic rigid deformities.

EMERGING CONCEPTS AND FUTURE DIRECTIONS

Despite substantial progress in the understanding and treatment of swan neck deformity, several controversies remain unresolved. One of the most debated aspects concerns the optimal timing of surgical intervention in inflammatory arthropathies. Some authors advocate early reconstructive procedures before irreversible joint destruction develops, whereas others recommend delaying surgery until systemic disease activity is adequately controlled. Contemporary biologic therapies have significantly altered the natural history of rheumatoid hand deformities, potentially allowing earlier intervention with improved preservation of soft tissue quality and joint integrity.

Another major controversy involves selection between motion-preserving procedures and salvage operations in advanced deformities. Although arthroplasty may preserve functional mobility, implant durability and postoperative instability remain significant concerns, particularly in younger or highly active patients. Conversely, arthrodesis provides predictable pain relief and stability but sacrifices interphalangeal motion. Comparative long-term studies evaluating functional outcomes and patient satisfaction between these approaches remain limited.

Modern biomechanical studies have improved understanding of extensor tendon dynamics, particularly regarding lateral band migration and intrinsic muscle imbalance. Nevertheless, the precise contribution of each pathological component likely varies among patients and disease etiologies. This heterogeneity explains why no single reconstructive procedure has demonstrated universal superiority.

Recent surgical trends increasingly emphasize individualized reconstruction based on dynamic tendon analysis, degree of articular degeneration, and patient-specific functional demands. Combined procedures involving tendon balancing, volar stabilization, and intrinsic release are becoming more common because isolated correction frequently fails to address the multifactorial nature of the deformity.

Minimally invasive and soft tissue-preserving approaches have also gained attention. Potential advantages include reduced postoperative stiffness, faster rehabilitation, and lower complication rates. However, current evidence remains largely limited to small retrospective series, and robust prospective comparative studies are lacking.

Advances in imaging technology may further improve preoperative planning. High-resolution ultrasound and dynamic magnetic resonance imaging allow increasingly

detailed assessment of tendon imbalance, synovitis, and capsuloligamentous integrity. These modalities may facilitate earlier diagnosis and more precise surgical indication.

Future reconstructive strategies may incorporate three-dimensional surgical planning, patient-specific implants, and biologic augmentation techniques. Tissue engineering and regenerative medicine approaches involving tendon scaffolds, growth factors, and cartilage regeneration may eventually expand treatment options for severe deformities associated with advanced joint destruction.

Importantly, the current literature on swan neck deformity remains limited by significant methodological heterogeneity. Many available studies consist of retrospective case series with small sample sizes, variable outcome measures, and short-term follow-up.

Standardization of clinical outcome reporting and development of higher-quality comparative studies are necessary to establish stronger evidence-based treatment algorithms.

Conclusion

Swan neck deformity represents a complex pathological condition arising from disruption of the intricate biomechanical equilibrium governing finger motion. Although classically associated with rheumatoid arthritis, the deformity may result from numerous traumatic, inflammatory, neurological, congenital, and degenerative processes.

Successful treatment requires comprehensive understanding of finger anatomy, dynamic tendon balance, and disease-specific pathology combined with individualized surgical planning and multidisciplinary rehabilitation.

TABLES

Table 1. Major Etiologies of Swan Neck Deformity

Etiology	Main Pathophysiological Mechanism	Typical Clinical Features
Rheumatoid arthritis	Synovitis and intrinsic tightness	Multiple finger involvement
Chronic mallet finger	Terminal tendon disruption	DIP flexion preceding PIP hyperextension
Volar plate injury	PIP instability	Posttraumatic hyperextension
Cerebral palsy	Intrinsic spasticity	Dynamic deformity
Ehlers-Danlos syndrome	Ligamentous laxity	Hypermobility
Psoriatic arthritis	Synovitis and joint destruction	Asymmetric involvement

Table 2. Surgical Procedures and Main Indications

Procedure	Primary Indication	Main Limitation
FDS tenodesis	Flexible deformity	Recurrence
Lateral band translocation	Dorsal migration	Technical complexity
Volar plate advancement	Volar plate insufficiency	Limited in rigid deformity
Arthroplasty	Degenerative disease	Implant failure
Arthrodesis	Severe degeneration	Loss of motion

DECLARATIONS

Conflicts of Interest

The authors declare no conflicts of interest.

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Author Contributions

All authors contributed substantially to the conception, literature review, manuscript drafting, critical revision, and final approval of the manuscript.

Ethical Approval

Not applicable.

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