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Lateral band translocation for swan-neck deformity: Outcomes of 41 digits after a mean follow-up of eight years

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ABSTRACT

Background: Swan-neck deformity (SND) of the fingers can cause major functional impairment. The Zancolli–Tonkin procedure is a crossed dynamic tenodesis that prevents overextension of the proximal interphalangeal (PIP) joint and promotes extension of the distal interphalangeal (DIP) joint. We assessed the outcomes of this procedure in patients with SND due to various causes.
Hypothesis: The Zancolli–Tonkin procedure provides effective and stable correction of SND due not only to RA, but also to other conditions.
Patients and methods: Consecutive patients managed at two centres between 2000 and 2013 were included. The causes of SND were inflammatory joint disease, trauma, iatrogenic events, and neurological disorders. The same operative technique was used in all patients.
Results: Forty-one fingers in 14 patients were evaluated. After a mean follow-up of 8 years, all patients could harmoniously flex the operated fingers and none had recurrence of the deformity. At the PIP joints, mean active flexion was 86° (range: 40°–90°) and mean loss of extension was 15° (range: 0°–40°). At the DIP joints, mean active flexion was 65° (range: 0°–70°) and mean extension lag was 4° (range: 0°–30°). The mean visual analogue scale pain score was 1/10 (range: 0/10–8/10) and the mean patient satisfaction score was 7.5/10 (range: 4/10–10/10).
Discussion: The SND was corrected and the results were stable after 8 years in all cases. Advantages of the Zancolli–Tonkin procedure include limited invasiveness, with no need to harvest a distant tendon, and rapid active postoperative rehabilitation. The moderate excessive PIP joint flexion has no adverse impact on the overall functional outcome. The high level of patient satisfaction reflects the improvements in function.
Conclusion: The Zancolli–Tonkin procedure is a simple and reliable technique that provides lasting correction of an incapacitating deformity associated with impaired overall hand function.
Level of evidence: IV.

1. Introduction

Swan-neck deformity (SND) of the fingers is produced by relative overactivity of the extensor mechanism at the proximal interphalangeal joint (PIPJ) combined with flexion of the distal interphalangeal joint (DIPJ). SND may impair global hand function. Causes of SND include inflammatory joint disease, chiefly rheumatoid arthritis (RA); post-traumatic or neurological imbalance (e.g., lesions of the PIP palmar plate, failure of the terminal extensor tendon, compartment syndrome of the hand, spasticity, and Parkinson's disease); constitutional joint laxity (e.g., hypermobility syndrome, Ehler-Danlos syndrome, and Marfan's disease); and iatrogenic events. SND may be a cosmetic concern, but surgery is usually considered only when it impairs function [1].

The digital chain is a functional unit whose harmonious motion depends on both the balance of forces and good synchronisation of the three phalanges [2,3]. The oblique retinacular ligaments (ORL) have a controversial role but may contribute to ensure good coordination among the components of the digital chain. SND is due to a combination of excessive extension forces applied to the base of the middle phalanx and of primary or secondary failure of the palmar PIPJ structures [3]. This imbalance leads to excessive PIPJ extension with primary or secondary DIPJ flexion. Dorsal subluxation of the lateral bands impairs the initiation of active PIPJ flexion, which is accompanied with snapping of the joint. Over time, the PIPJ becomes fixed in extension and the DIPJ extension lag increases.

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Zancolli [4] described three pathophysiological mechanisms causing SND:

- an intrinsic mechanism, involving overactivity or contracture of the interosseous muscles;
- an extrinsic mechanism, involving mallet finger deformity or subluxation of the extensor tendon at the metacarpophalangeal joint;
- an articular mechanism, consisting in palmar plate failure or a dorsal fracture-subluxation of the PIP.

Several mechanisms may be combined, as seen in RA [5]. The extrinsic and articular mechanisms are responsible for most cases of post-traumatic SND.

Many surgical techniques for the treatment of SND have been described [6–8]. They fall into three main groups [9]. For SND secondary to mallet finger, release of the central slip balances the extensor mechanism forces [8]. Techniques that counteract the PIPJ hyperextension without directly acting on the DIPJ include Bate's capsulorrhaphy and tenodesis of the flexor digitorum superficialis (FDS) [1,10–12], which shares similarities with the palmar plate arthroplasty described by Eaton and Malerich for the treatment of dorsal fracture-subluxation [13]. The other techniques are designed to act on both interphalangeal joints by fashioning an ORL-like structure via either mobilisation of one of the extensor mechanism lateral bands [6,14,15] or a spiral tendinous graft crossing behind the DIPJ and in front of the PIPJ [16].

The first technique described by Littler [15] involved severing the lateral band proximally then transferring it volar to over the Cleland's ligament and stitching it to the flexor tendon sheath. For spastic hands, Zancolli [4] advocated translocation of the middle part of the lateral band volar to the centre of the PIPJ, while preserving lateral band continuity. Subsequently, Zancolli introduced a modification in which the translocated lateral band is stabilised by creating a sling between the palmar plate and FDS, which are secured to each other by two stitches [6]. Tonkin et al. extended the indications of this procedure to the rheumatoid hand, provided the PIPJ alterations were moderate.

The simplicity of the Zancolli–Tonkin procedure and the outcomes reported by Tonkin and al. [14] in SND due to RA and other conditions led us to adopt this method for our patients. Our hypothesis was that the Zancolli–Tonkin procedure provided effective and stable correction of SND due not only to RA, but also to other conditions. The objectives of this study were to evaluate whether this procedure restored phalangeal synchronisation and digital chain balance and to assess long-term outcomes in patients with SND due to various conditions.

2. Patients and methods

This retrospective study conducted in two centres included 20 consecutive patients with SND of one or more fingers, due to various causes, and treated by the Zancolli–Tonkin procedure between 2000 and 2013. Surgery was performed because of functional impairment. Fingers with degenerative PIPJ disease were excluded. A follow-up of at least 1 year after surgery was required for study inclusion.

The surgical procedures were performed by two senior surgeons, one in each of two hand surgery units, as described by Tonkin et al. [14]. The mid-portion of one lateral band was translocated to the palmar aspect then maintained over the PIPJ by suturing to the ipsilateral FDS slip and lateral aspect of the palmar plate (Fig. 1). Postoperatively, a dorsal extension-block splint maintaining 20° of PIPJ flexion was worn for 3 to 4 weeks, during which gentle flexion protected by the splint was allowed. After splint removal, rehabilitation was conducted under the supervision of a physiotherapist.

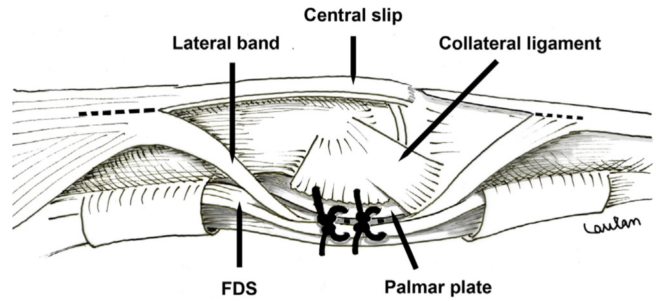

Fig. 1. The Zancolli–Tonkin procedure. The mid-portion of one of the lateral bands is translocated to the palmar aspect, where it is secured by suturing the ipsilateral FDS slip to the lateral aspect of the palmar plate.

Demographic data, the number of treated fingers, and the SND stage according to Nalebuff and Millender were recorded [17]. At last follow-up, an independent examiner evaluated the active range of motion, scars, and global hand function. When flexion was incomplete, the pulp-to-palm distance was measured in centimetres. Active extension of both interphalangeal joints was measured with a goniometer in patients who were assessed in person and self-evaluated by those who were assessed over the telephone. The QuickDASH questionnaire was completed by each patient to assess global upper-limb function [18]. Patients were asked to rate their functional impairment as absent, moderate (only during unusual gestures or significant effort), marked (during activities of daily living), or severe (preventing normal use of the hand). They also specified whether they had resumed their daily activities at a higher, equal, or lower level than before surgery.

Residual pain was self-evaluated using a 10-centimetre visual analogue scale (VAS). Patients were asked to assess their satisfaction by rating their functional outcome on a scale from 0 to 10; scores ≥ 9 indicated an excellent outcome, 7 to 8.9 a good outcome, 5 to 6.9 a fair outcome, and < 5 a poor outcome.

3. Results

Among the 20 patients who had surgery, 4 were lost to follow-up, one was too severely ill to be evaluated, and one died. This left 14 patients for the study, of whom 12 were evaluated in person and 2 over the telephone. There were 8 women and 6 men with a mean age at surgery of 51 years (range: 31–77 years). Of the 14 patients, 7 were sedentary, 2 were heavy manual workers, 4 were light manual workers, and 1 was unemployed. At surgery, 2 patients were on disability leave because of their hand function impairments.

Surgery was performed on both hands in 3 patients (all right-handed), on the right hand in 7 patients (4 right-handed, 2 left-handed, and 1 ambidextrous), and on the left hand in 4 patients (all right-handed). The number of fingers with SND was 1 in 5 patients, 2 in 5 patients, 3 in 2 patients, and 4 in 5 patients, yielding a total of 41 operated fingers, in 17 hands. Of the 41 fingers, 10 were forefingers, 14 middle fingers, 11 ring fingers, and 6 little fingers.

The cause of SND was RA in 33 fingers, trauma (mallet finger) in 2 fingers, cerebral palsy in 2 fingers, constitutional hypermobility in 1 finger, and surgical fourth-finger FDS removal in 3 fingers (transfer for radial nerve palsy in 1 finger and lasso procedure to treat ulnar claw finger deformity in 2 fingers). In the Nalebuff and Millender classification, 12 fingers were stage I, 11 were stage II, 9 were stage III, and 9 were stage IV. All patients had major disability, usually with markedly impaired grip function. The unsightly nature of the deformities was a concern for all patients.