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A Historical Perspective of the Notta's Node in Trigger Fingers

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Abstract

Trigger finger is a condition that can result from a stenosed digital flexor tendon sheath. The condition was first described by French physician Alphonse Notta in 1850 in a report that consisted of four case studies of adult patients. Notta's and other reports written in the 19th century reveal the early views on this condition's etiology and management. The 20th century saw the development of the modern day treatment methods for the condition, namely corticosteroid injections and the surgical release of the A-1 pulley.

Keywords

Trigger Finger; Notta's node; Alphonse Notta; hand surgery; history

Trigger fingers and thumbs are "anatomically distinct disorders that vary according to location but generally represent one pattern of pathology¹." Pathologically speaking, most trigger digit cases stem from digital flexor tendon entrapment that is caused by a thickened A-1 pulley, often secondary to a stenosed tendon sheath. Interestingly, although both conditions have similar etiologies, they manifest differently in adults and children. In adults, the presentation is not particular to any digit and most clinicians report that the symptoms manifested are snapping, clicking, locking, and stiffness associated with extending a flexed digit¹. In contrast, in children, this condition occurs in the thumb in 93–97% of the cases^{2, 3} and, instead of any snapping symptoms, the thumb locks in flexion¹. Also mentioned by most reports on pediatric trigger thumbs is the presence of a tendon nodule located on the volar aspect of the base of the thumb¹.

In recognition of Alphonse Henri Notta, the 19th century French discoverer of this triggering condition, this tendon nodule is now commonly referred to as Notta's node. This paper will begin by examining a translation of Notta's original report on the *doigt à ressort*, or trigger finger⁴. By exposing the clinical and etiological descriptions of trigger finger from Notta and subsequent historically important reports, we aim to elucidate the evolution of knowledge concerning this condition and its management through the last 159 years.

The doigt à ressort according to Notta

Notta (1824–1914), while serving as a medical intern at *l'hôpital Saint-Louis*, began the pioneer report on the ailment⁴, published in the August, 1850 edition of the hospital gazette, by acknowledging that the inspiration for his study was purely incidental:

“The disease which is being considered here has not yet been described by the authors... however, for no apparent reason there has been an interest in this field over the past months. Consequently we have been able to observe and study a number of cases. This allows us to give a complete description of the salient symptoms pertaining to this medical condition⁴.”

The report described how Notta, guided by his mentor Dr. Auguste Nélaton, examined four patients who presented with digits that could not be easily, if at all, extended from the flexed position. For each case, Notta narrated a remarkably meticulous exposition of the mechanics of the condition; however, because of the similarities between the cases, Case 1 can serve as the representative presentation of this condition. Notta wrote about *Patient X*, a 28- year-old seamstress, who presented to Nélaton with a hindrance in the middle finger of her right hand that prevented her from working. He elaborated:

“When all the fingers of the right hand are flexed and the patient wants to straighten them, all the fingers straighten out comfortably except the middle finger which begins to straighten then it stops. The patient then forcefully straightens out her extensors, a brisk movement occurs. The middle finger then straightens out easily. However, very often this extra effort of the extensors is not enough and her finger remains flexed. The patient then uses the extremity of the right thumb and lifts the last phalanges of the middle finger a little, this then allows her finger to be extended⁴.”

Despite the relative simplicity of the trigger finger condition, it remains to Notta’s credit that during the 159 years since this first clinical description, little has added to the condition’s mechanical characterization as recorded in this first case study. It is in *Case 2*, however, where Notta presented in detail the manifestation of the principal symptom, the node. After a similar description of the movements of the patient’s fingers, Notta wrote:

“To straighten the ring finger the patient has to use her other hand. A cracking sound at the center of the hand can be heard as it opens up by itself. Upon this event is observed: Firstly, a nodosity on the span of the flexor tendons located slightly above the inferior palmar crease given that the ring finger is flexed. Secondly, the nodosity disappears when the finger is extended and relocates itself at the digital palmar crease. Thirdly, the movement takes place in two steps. First there is a total resistance which then gives way and secondly there is the aforementioned sound followed by an acute protrusion at the point of the nodosity which seems to have overcome an obstacle...This (the protrusion) can be felt. Once overcome (the resistance) the finger extends with ease⁴.”

After presenting the case studies, Notta began the discussion of his reports with the following observation: “The medical condition described in these case studies pertains to a nodosity on the span of the flexor tendons of some fingers. This nodosity encounters

obstacles when fingers are being bent and stretched...What is the cause of this nodosity? What obstacles does it encounter⁴?”

His questions served as a natural segue into a detailed review of the anatomy of the muscles, tendons, and ligaments of the hand, the information for which Notta cited as being from an 1839 text by Dr. Maslieurat Lagénard⁵. Though using mostly lay terminology to describe this anatomy, Notta nevertheless presented an accurate and readable review on the biomechanics of the hand. The movements of the flexor tendons into the sheaths, with respect to the locations of the fibrous pulleys, were described in great detail. Based on this anatomy, he was able to surmise that the obstacle to extension was encountered when the digital flexor tendon attempted to pass through the proximal end of tendon sheath, which he referred to as the “synovial fluid cavity.” While citing inflammation as the cause of this obstacle, he remained unsure about the specific location of the problem, and noted that the inflammation could be present in the tendon itself or within its sheath.

To this end, Notta offered two hypotheses explaining the etiology of the node: 1) the node could be a swollen segment of the “synovial fluid cavity” or 2) the origin of the node could be the tendon itself, in which case the node would consist of a thickened area of the tendon. He proposed that, citing an analogy to plaque deposits on arterial walls, “pseudomembranes,” which attached themselves to the tendon or to the interior of the tendon sheath, could be the root cause of the inflammation. In either case, he noted, that the obstacle to extension was encountered when the swollen region attempted to pass through the “fibrous arch⁴,” what we know as the first annular pulley. However, “Once the nodosity is under the fibrous arch it (the finger) moves easily into its place⁴.”

Unfortunately for their four patients, Notta and Nélaton were unable to prescribe any beneficial treatments to relieve their symptoms. The application of hot compresses, vesicatory creams, and even six leeches⁴ over the nodal region, all uniformly failed to provide the patients even slight relief. In one man (*Case 3*), however, the condition spontaneously cured and the node gradually disappeared within eight months after he presented with the condition⁴. Notta did briefly describe a method that resembled a subcutaneous release of the A-1 pulley, but admitted that he had only tried it on cadavers⁴.

Although his understanding of the condition and his treatment methodology were somewhat flawed, Notta was able to recognize the primary causes of the condition, which he described in the closing remarks of his report. Noting that the woman of *Case Study 2* suffered from rheumatism, Notta conjectured (correctly) that the swelling of the “synovial cavity,” which was causing the obstacle to the finger’s extension, was secondary to the rheumatism⁴. For the man of *Case Study 3*, a chemist whose symptoms eventually disappeared, Notta attributed the onset of the condition to “a traumatic lesion on the tendon producing inflammation,” a consequence of when the man accidentally stabbed himself in the palm with a piece of broken glass in his laboratory⁴. Notta wrote that the scar from the accident could be seen “on the trajectory of the flexor tendon of the ring finger.” For the seamstress presented in *Case Study 1*, Notta conjectured that “the lesion” that induced the swelling could be a result of the “repetitive occupational microtrauma to the tendon” from her constant sewing⁴. It is remarkable that to this day, these causes continue to be associated

with the onset of trigger finger, although many cases are cited as being of an idiopathic nature.

Influence of Notta's Concepts in Later Years

In the half of the 19th century following Notta's publication, European medical societies became more interested in the *doigt à ressort*. French and German physicians led the research efforts on the topic and published a myriad of case reports and etiological descriptions of the condition^{6, 7}. Most of these consisted of similar anecdotal accounts of trigger finger cases and ended with the author espousing unscientific opinions concerning the condition's etiology and the appropriate treatments. Several scientific studies and accounts of treatment methodology deserve special mention, because they provide historical perspective on the common views of the condition's etiology and management of a particular era.

In his article *On Springing Fingers*, written and also translated into English in 1874, German physician Arthur Menzel elucidated a precise theory concerning the origin of a triggered digit⁸. Using cadavers, Menzel scientifically tested Notta's hypothesis that "a small, round, moveable body" on the flexor tendon blocks the passage through the fibrous pulley, causing the finger to be, either permanently or temporarily, locked in flexion. In his first experiment, Menzel artificially constructed a Notta's node on one of the cadaver's fingers by looping thread around an exposed region of the flexor tendon. Upon manipulation of the finger, however, Menzel discovered that this alone was not sufficient to achieve the onset of the triggering condition. It was only after additionally imposing an artificial constriction of the tendon sheath, by tying it down with thread, that he observed the characteristic "springing" motion of trigger finger⁸.

In a separate experiment using cadavers, Menzel tested whether small, internally-trapped "free bodies of foreign origin," which Notta referred to as "pseudomembranes," could sufficiently clog the sheath to a level that would impede passage of the flexor tendon and consequently cause the "springing" effect. This theory had previously been advanced by physicians writing on the topic⁶. However, after manually inserting into the sheath such things as hemp seeds and grains of rice, Menzel discovered that these "bodies" generally were forced to the side of the sheath and did little to impede passage of the tendon, thereby casting doubt on this particular hypothesis⁸.

Concluding the reports on his experiments, Menzel wrote that both the "contraction of the sheath" and the "circumscribed tumor of the tendon," which he hypothesized are "products of inflammation," act mutually as the causative agents of the ailment⁸.

Aside from a translation of Menzel's work, which ran in the 1874 volume of the Boston Medical and Surgical Journal, widely distributed original publications on trigger digits did not appear in the English language until over three decades after Notta's study, and not in American medical journals until around the turn of the 19th century. When English-speaking practitioners began to write about the topic, many of their articles simply relayed and remarked on the findings of French and German doctors while adding a few accounts of trigger finger cases as encountered in their personal practice. However, several British

orthopedic surgery textbooks published during this era offer insight regarding how physicians of that day proceeded to treat patients that presented with trigger finger^{9, 10}.

These texts present various recommendations regarding the treatment of trigger finger, including: the application of cold packs, “elastic compression and massage therapy,” puncturing and draining Notta’s nodule¹¹, and even one of the earliest English language reports of a procedure resembling the open A-1 pulley release¹². Despite the successful report of the resection of the A-1 pulley in several cases¹², it remained commonplace in surgery textbooks to recommend excision of any tendon node in the vicinity of the MCP joint^{9, 10}. One doctor, Charles Dana, even went as far as to prescribe the repeated (and unsuccessful) “application of the galvanic current” to the triggered digit of one patient¹³. Perhaps due to a universally poor understanding of the histopathological nature of trigger finger, procedures like node excision and galvanic current application were not deemed detrimental until a greater part of a century had transpired following Notta’s characterization.

Not until the advent of the second half of the 20th century were cogent explanations of the histopathology of Notta’s node and the etiology of the triggering condition presented. Though a basic understanding that the condition is secondary to a stenosis of the tendon sheath was agreed upon, conflicting hypotheses about the exact etiology emerged. A precise consensus on the topic is still yet to be reached.

In 1954, Chicago physicians J.J. Fahey and J.A. Bollinger wrote that, upon examining the A-1 pulleys of trigger finger patients under a microscope, they discovered that Notta’s nodule was actually “the markedly thickened fibrous sheath, sometimes 2–3 times the normal thickness².” They also discovered marked proliferation of irregular fibrous tissue in the pulley’s inner layer, a sign of fibrocartilaginous metaplasia. Even today, this finding continues to be cited by scientists examining affected tendon sheaths¹⁴. In children, however, Fahey and Bollinger wrote that the “nodular formation” was primarily due to degenerative changes within the tendon itself, notably the “splitting or fraying of the fibers².”

” Then, in 1972, Australian Doctors J.T. Hueston and W.F. Wilson proposed what they called the “needle and thread analogy” to explain the formation of Notta’s nodule as the result of a stenosed tendon sheath¹⁵. Hueston and Wilson hypothesized that, regardless of the cause of the stenosis, the constricted proximal end of the tendon sheath delivered a “recurrent deforming force” upon the tendon during each act of flexion and extension, eventually causing a particular segment of fibers to “bunch up¹⁵” into a nodular formation.

Also in the 1950’s, at the time when recommendations of nodule excision began to diminish and the resection of the A-1 pulley was becoming the prominent trigger finger management², two new treatment methodologies emerged. In 1953, at a meeting of the American Society for Surgery of the Hand, Dr. Lot D. Howard presented the results of his successful clinical trial, where, through administering hydrocortisone injections into the tendon sheath, five out of six patients experienced complete relief of the triggering symptoms¹⁶. Five years later, the Belgian physician Dr. Jean Lorthioir Jr. published a description of correcting trigger digits through a subcutaneous resecting of the constricted

tendon sheath with a fine tenotome¹⁷. Lorthioir claimed to have achieved good results, with “no inflammatory or functional complications,” in fifty-two patients.

The works of Lorthioir and Howard effectively serve as the starting point for modern treatments of trigger finger, as both corticosteroid injections and subcutaneous release are treatments currently prescribed by physicians. Currently, more than a century and a half following Notta’s research, discord regarding appropriate treatment of trigger finger persists amongst hand specialists, as evidenced by the several hundred articles published post-2000 in journals pertaining to the topic. (Figure 1)

From a historical perspective, the history of this condition’s treatments, from the application of leeches⁴ and the galvanic current¹³ in the 19th century to the treatment methods used today, effectively serves as a microcosm of the evolution of the field of medicine over the past two centuries. Through the early observational experience to current emphasis on evidence based medicine, the etiology of this intriguing condition, the trigger digit, though still not completely evident, is slowly being revealed.

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Figure 1.
Timeline of Major Events in Trigger Finger Management