HALLUX RIGIDUS


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Patients with hallux rigidus are customarily classed into adolescent and adult groups according to the age at which symptoms first appear. Radiologists have called attention in adolescent cases to an appearance of increased density, sometimes of fragmentation, of the epiphysis of the proximal phalanx, and it has been claimed that these changes may have etiological significance. This paper describes a series of clinico-pathological studies undertaken by the authors to determine the nature of the alleged radiographic changes and to investigate the detailed pathology and etiology of hallux rigidus.

Clinical material—Thirty-three patients were studied. Eighteen were under twenty-five years of age (adolescent group) and fifteen were over twenty-five years of age (adult group). Twelve operation specimens of bone and joint tissue were examined histologically. A control series of fifty normal feet was studied radiologically; and specimens of healthy great-toe joints obtained at autopsy were sectioned.

Analysis of Important Observations

Clinical features—The youngest patient was ten years of age, the oldest sixty-two; the other cases were distributed fairly evenly between these extremes. In the adult group the two sexes were affected equally, but in the adolescent group seventeen of the eighteen patients were females. This great preponderance of females in the adolescent group is not easy to explain but may reflect the difficulty of obtaining satisfactory shoes for growing girls.

Symptoms—Pain of gradual onset in the great toe had been present variably between six weeks and nineteen years. In a few cases the pain had been precipitated by injury, but usually no cause could be remembered. The pain was described as constant, burning, throbbing or aching. It was relieved by rest and made worse by walking or sometimes by the warmth of the bed.

Signs—The metatarsophalangeal joint was enlarged and tender, and often held in slight flexion. Active dorsiflexion was impossible and attempts to dorsiflex the toe passively were painful. Flexion was little impaired except in advanced cases in which the joint was completely rigid. Movements of the joint were often accompanied by crepitus. The skin beneath the metatarsophalangeal joint was smooth, but a callosity usually covered the medial half of the pad of the great toe. A much larger callosity occurred under the heads of the second, third and fourth metatarsals. A third callosity was often seen on the outer border of the heel. Occasionally a large exostosis on the dorsum of the first metatarsal head was covered by a bursa giving rise to a painful dorsal bunion.

The interphalangeal joint was always hypermobile in adolescents; as much as 60 to 70 degrees of passive dorsiflexion was often present. In adults the range of interphalangeal movement was sometimes diminished, and occasionally painful. In some patients spasm of the extensor hallucis longus raised a longitudinal fold of skin and pulled the terminal phalanx into hyperextension. The general shape of the feet was seldom normal. Hallux rigidus was usually associated with long, narrow, pronated feet with unstable arches. Other abnormalities included excessively long great toes, hypermobile or adducted first metatarsals, valgus heels due to shortening of the tendo Achillis, and short flexor hallucis longus tendons. There was usually some clawing of the outer four toes.
Shoes were seldom wide enough and were often too short. Shape and wear were so typical that the diagnosis of hallux rigidus could often be made simply on a glance at the patient's shoes (Figs. 1 to 5). There was excessive wear on the outer side of the heel and posterior half of the sole, and under the terminal phalanx of the great toe. The tread was much nearer the front of the shoe than normal. The toe-spring was correspondingly shortened, and the upper bulged over the outer side of the heel and the posterior half of the sole. There were at least two creases across the vamp. One, at the level of the metatarso-phalangeal joints, crossed it completely. In front of this crease, and on the medial side only, there were one, two or three more, furrowing the toe-cap; these had been caused by the hypermobile interphalangeal joint of the great toe.
PATHOLOGY

The proximal epiphysis of the phalanx—Increased radiographic density of the proximal epiphysis of the phalanx (Fig. 6) was present in all cases in our adolescent group. Sometimes an irregular blotchy density was seen (Fig. 7) and occasionally the radiographs suggested fragmentation of the epiphysis (Fig. 8). Exactly similar changes were, however, seen in the clinically normal foot in unilateral cases; and in a control series of fifty unselected symptomless feet in patients of similar age attending the clinic for other reasons. In these patients, areas of relative translucence often have the appearance of fragmentation (Fig. 9).

To study the matter further, four healthy great-toe joints were removed at autopsy from children between the ages of six and fifteen years. The specimens were radiographed and then sectioned. In every case the phalangeal epiphysis appeared relatively opaque in the radiographs (Fig. 10). Histologically the bone of the epiphysis consisted of very broad trabeculae with small marrow spaces. The trabeculae, in contrast with those of the phalangeal shaft, were disposed more transversely across the long axis of the bone. In some of the larger bone masses the small vascular canals were surrounded by concentric rings of laminated bone, thus showing a pattern resembling compact or cortical bone. Occasionally a raphe of cellular fibrous tissue extended between the articular cartilage and the growth plate (Fig. 11), dividing the epiphysial bone into two or more parts and clearly indicating that ossification had taken place simultaneously from two or more secondary centres. A similar appearance of coarsely trabeculated, almost compact, bone in the epiphysis was observed in several phalangeal epiphyses removed at operation for adolescent hallux rigidus (Fig. 12). In no instance was there any evidence of avascular necrosis, bone dystrophy, fracture or inflammation.

The joint—Where the onset had been recent, examination at operation showed an increase in the fluid content of the joint. The fluid was clear and viscous, but the quantities were too small for analysis. Erosions of the articular cartilage at the centre and at the dorsal margin of the base of the phalanx were always found. Erosion of the corresponding area of the metatarsal head was seldom seen. Histologically the synovial membrane showed moderate synovial proliferation with congestion of the villi and a light lymphocytic infiltration (Fig. 13). The cartilage erosion and synovial changes were consistent with the reaction of the joint to
Fig. 11
Section of epiphysis and articular cartilage of normal great-toe proximal phalanx. (Autopsy specimen from male, aged fifteen years.) Articular cartilage is normal. Epiphysial bone is formed of broad and strong trabeculae whose general disposition is across the long axis of the bone. There is a median raphe of fibrous tissue which divides the epiphysial bone and has arisen from two centres of ossification. (H. & E., .33 approx.)

Fig. 12
Coarsely trabeculated, almost compact, bone of the basal epiphysis. Operation specimen from girl aged ten years (cf. radiograph in Figure 6). This is healthy bone of the structure normally seen in this epiphysis. Section shows articular cartilage (above), and epiphysial bone, healthy growth cartilage and metaphysis (below). (H. and E., x18 approx.)
Figure 13—Synovial membrane in hallux rigidus. Moderate villous proliferation, vascular congestion and light lymphocytic infiltration of synovial tissues near reflection from metatarsal head. Traumatic synovitis. Early erosion of phalangeal cartilage was present. (Girl, aged twelve years.) (H. and E., × 40 approx.)

Figure 14—Earliest radiographic evidence of osteoarthritis. Left foot of girl, aged fifteen years, with bilateral hallux rigidus. The basal epiphysis is almost united but still appears dense. The metatarsal head is becoming flattened and shows osteophytic lipping of the dorso-lateral margin.

Figure 15—Section of base of proximal phalanx of great toe. Male, aged twenty-one years, with three years' history of hallux rigidus. Osteoarthritis with cartilage erosion and fibrillation near centre and towards dorsal margin (right), and dorsal osteophyte. The sclerotic subchondral bone is the normally dense bone of the epiphysis which persists after epiphysial union. (H. and E., × 40 approx.)
chronic trauma. In adolescent patients we found no evidence of pre-existing arthritis of rheumatoid or infective type.

In cases of longer standing there was evidence of well-marked osteoarthritis. The earliest age at which radiographic evidence of osteoarthritis was present was fifteen years; deformity of the joint facets and marginal lipping can be seen in Figure 14. Histologically the appearances were those characteristic of osteoarthritis, which have been described recently by Collins (1949). There was an unusual amount of dense subchondral bone (Fig. 15), representing a survival of the original dense epiphysis rather than subchondral bone sclerosis reactive to destruction of the overlying cartilage. Loss of superficial cartilage layers, clumping of chondrocytes in the deeper layers and irregularity of the calcified line at the base of the articular cartilage were often seen (as in Fig. 16). Fibrillation of cartilage and irregular advance of ossification into the articular cartilage were also seen (Fig. 17), and marginal osteophytic spurs and shelves took the usual form.

![Figure 16](image1.jpg)

**Figure 16**—Early osteoarthritis in hallux rigidus. Note loss of surface zones of articular cartilage, distorted calcified line in deep cartilage and density of subchondral bone. The marrow spaces, vascular canals and some bone canaliculi are outlined by the silver stain. (Gomori's silver nitrate method, ×31 approx.)

![Figure 17](image2.jpg)

**Figure 17**—Osteoarthritis in hallux rigidus. Section of base of proximal phalanx with typical osteoarthritic changes, destruction and fibrillation of cartilage and irregularity of chondro-osseous junction. Male, aged forty-four years, with symptoms arising in adult life. (H. and E., × 27 approx.)

**DISCUSSION**

An appearance of increased density of the basal epiphysis of the proximal phalanx in adolescent hallux rigidus has led to much speculation about its significance. Hauser (1939) and Glissan (1946) considered that the radiographic appearances might represent an affection of the bone of a similar nature to Legg-Perthes disease of the hip and other forms of osteochondritis juvenilis, and they believed that these changes were of etiological significance. Brailsford (1948) stated that "pain in the big-toe joint with limitation of movement in the young person may be associated with increased density of the basal epiphysis. In some cases this is the forerunner of chronic arthritis of the joint. The dense epiphysis may appear to be split into two parts by a fissure in the mid-line. This may be seen in girls about the age of twelve years who complain of pain." Our studies of the morbid anatomy of hallux rigidus do not support the view that the radiographic appearance of density of the basal epiphysis
of the proximal phalanx is etiologically significant. The fact that similar changes are present in normal feet in adolescents suggests that the increased density does not represent any abnormality of the bone. In this connection a study of the development of the basal phalangeal epiphysis is of interest. The epiphysis appears as two or three tiny dots at the age of two to three years in girls and three to four years in boys. These centres coalesce to form a single centre of ossification within a year. At six years of age the epiphysis becomes denser than the adjoining bone and remains dense throughout the period of growth. It fuses with the shaft at thirteen to fourteen years in girls and fifteen to seventeen years in boys. After fusion has occurred the base of the phalanx retains its increased density in some degree throughout life. Similar inequalities in radiographic density have been observed in the calcaneal epiphysis and have been misinterpreted as pathological abnormalities. But Hughes (1948) has determined that the same appearance is seen in normal feet as well as in feet with painful heels, and Bergmann (1926) has shown histologically that the apparent increase in radiographic density is due to the closely packed but healthy trabeculae, which in this epiphysis also are disposed at 90 degrees to the general disposition of the trabeculae in the main part of the bone.

As to the nature of the pathological changes in hallux rigidus, our observations have led us to conclude: 1) that the earliest detectable pathological changes are traumatic synovitis and cartilage erosion; and 2) that osteoarthritis develops sooner or later and has the same characteristics as osteoarthritis occurring elsewhere in the body.

![Fig. 18](image)

Diagram showing how a boat-shaped sole elevates the first metatarsal and how the curvature of the transverse anterior arch of the foot becomes reversed.

The fact that gross osteoarthritis of the metatarso-phalangeal joint may arise early in life, usually without preceding injury, suggests that abnormal mechanical conditions may be an important etiological factor. We have investigated the nature of such mechanical abnormalities, and have formulated a hypothesis to explain the origin of hallux rigidus. We consider that hallux rigidus is due to an abnormal gait which so alters the dynamics of the foot that osteoarthritis develops in the joints of the great toe. The causes of the abnormal gait are pain or discomfort in the great toe and hypermobility of the first metatarsal.

Pain or discomfort in the great toe may arise from: 1) *Valgus deformity of the feet*: this is common in patients with hallux rigidus; most of the weight of the body is borne on the inner side of the foot, and excessive pressure acts on the proximal great-toe joint. 2) *Defective footwear*: shoes that are too short or too narrow predispose to repeated minor injuries or compression forces acting on the great toe; excessive length of the great toe may have similar effects. 3) *Injury to the joint of the hallux* by severe stubbing, by forcible hyperflexion or by dropping a weight on the toe. 4) *Damage to the joint* by rheumatoid arthritis or gout.

Hypermobility of the first metatarsal may be a developmental anomaly. But more often it seems to be caused by squeezing the foot into a shoe that is too narrow. In order to make such a thing possible, the manufacturer often designs shoes with the sole concave upwards. As a result, the first and fifth metatarsals are displaced dorsally and become hypermobile. The middle three metatarsals sink into the hollow of the sole (Fig. 18). The anterior metatarsal arch, normally concave downwards, becomes reversed. The first and fifth metatarsals cause the upper of the shoe to bulge over the sole medially and laterally. A hypermobile first metatarsal is mechanically incompetent. Instead of providing a firm and stable point for weight-bearing and locomotion, its head becomes displaced dorsally. Some patients overcome this handicap by relying for stability on the second metatarsal;
others develop the hallux rigidus gait. From a study of the callosities on the patient’s feet, from the peculiarities of wear seen in old shoes and from statements made by observant patients, this abnormal gait can be analysed. The weight of the body rolls along the outer side of the foot until the heel is well off the ground and the heads of the fourth and fifth metatarsals are bearing weight; it then passes medially across the callosity covering the middle three metatarsal heads to the second phalanx of the great toe, by-passing the metatarso-phalangeal joint (Figs. 19 and 20). The push-off is effected mainly by the four outer toes and to some extent by the pad of the great toe. The proximal joint of the hallux plays no part: its function is taken over by the hypermobile interphalangeal joint. In this gait the metatarso-phalangeal joint is protected from direct weight-bearing pressure. The joint is held in flexion by the flexor brevis hallucis and is not in contact with the sole. At the same time the first metatarsal is stabilised: for contraction of the flexor brevis prevents the dorsal displacement of the bone and converts it and the first phalanx into a single, stable, downpointing unit. As a result, the forces acting in the long axis of the first metatarsal are transmitted direct to the shallow articular cup on the base of the first phalanx (Figs. 21 and 22). The head of the first metatarsal causes attrition of the centre of the basal articular cartilage in exactly the same way as a pestle removes the glaze from the deepest portion of a well-used mortar. The dorsal erosion appears later. It is due to dorsiflexion of the joint against the resistance of the shortened flexor brevis tendon, which holds the two articular surfaces closely applied together. In early cases full dorsiflexion of the joint can be obtained under anaesthesia. It is probable, therefore, that the shortening of the flexor brevis at this stage is due to spasm. In the chronic case contracture of the muscle prevents full dorsiflexion even under anaesthesia.

The outlines above were traced from radiographs of the two feet of a patient with left hallux rigidus. The feet were examined in the push-off phase. Figure 21—In the normal right foot the two phalanges of the great toe are horizontal; the body-weight is transmitted into the base of the phalanx, the flexor brevis tendon and the sesamoids. Figure 22—The first phalanx of the left foot is dorsiflexed and the sesamoids are well above the horizontal line; the arrows indicate the compressive effects of passive dorsiflexion.
Once the cycle of osteoarthritis has been begun by a cartilaginous erosion, fibrillation and disappearance of the cartilage and formation of osteophytes follow. But even in advanced cases the situation of the early lesions is recognisable. Cartilage loss and eburnation of underlying bone are most marked in the centre of the base of the phalanx; osteophytic lipping is most pronounced at the dorsal margin of the joint. Later, abnormal strains and stresses cause osteoarthritis in the interphalangeal joint as well.

Two observations suggest that the abnormal gait precedes the appearance of symptoms. 1) Patients who had had pain only for a few weeks showed such well-marked changes in their feet and shoes that the duration of the abnormal gait was clearly a matter of months rather than weeks. 2) It was observed in cases of unilateral hallux rigidus that the painless great toe of the other side often lacked dorsiflexion, that the foot showed the characteristic callosities, that there was smooth skin under the proximal joint of the great toe, and that subsequently this toe became painful.

It is reasonable to expect that the strains and minor injuries of normal day-to-day activity would affect mainly the right foot in right-footed persons and the left foot in left-footed persons. In our adolescent group eleven of the fifteen unilateral cases of hallux rigidus were ipsilaterally footed. In bilateral cases, the more painful side is almost invariably determined by the patient's footedness.

**SUMMARY**

1. Clinically, hallux rigidus is a painful condition of the joints of the great toe associated with loss of dorsiflexion of the first phalanx.
2. Pathologically, the morbid changes are those of a traumatic synovitis followed by an early development of osteoarthritis, the initial lesions of which are erosions of the cartilage at the centre and near the dorsal margin of the base of the proximal phalanx. There is no fundamental pathological difference between the adult and adolescent varieties of hallux rigidus. Both represent stages in the developmental cycle of osteoarthritis in the proximal joint of the great toe.
3. The radiographic density and apparent fragmentation of the phalangeal epiphysis do not represent an abnormality of the bone and have no significance in the etiology of hallux rigidus.
4. The cause of hallux rigidus is an abnormal gait developed either to protect an injured or inflamed metatarso-phalangeal joint from the pressure of weight-bearing, or to stabilise a hypermobile first metatarsal. The effects of this gait are to transfer most of the pressure from the flexor brevis tendon and the two sesamoids to the base of the first phalanx. Excessive pressure on this joint predisposes to osteoarthritis.
5. Evidence of this abnormal gait is found in the peculiarities of wear seen in old shoes.
6. There is a high correlation between unilateral hallux rigidus and the patient's footedness.

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**REFERENCES**


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