Ministry of Defence

Synopsis of Causation

Osteochondritis Dissecans (incorporating Osteochondrosis)

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Disclaimer

This synopsis has been completed by medical practitioners. It is based on a literature search at the standard of a textbook of medicine and generalist review articles. It is not intended to be a meta-analysis of the literature on the condition specified.

Every effort has been taken to ensure that the information contained in the synopsis is accurate and consistent with current knowledge and practice and to do this the synopsis has been subject to an external validation process by consultants in a relevant specialty nominated by the Royal Society of Medicine.

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1. Definition

1.1. **Osteochondritis dissecans (OCD)** is a condition in which a segment of articular cartilage and subchondral bone separates from the remaining articular surface. The title was first coined in 1888. Although the etymology of the word "osteochondritis" suggests an underlying inflammatory process, subsequent research has failed to elicit evidence of inflammation. The word "dissecans" derives from a Latin root meaning "to separate".

1.2. In OCD, an area of subchondral bone becomes necrotic, resulting in a loss of structural support for the overlying articular cartilage. This process leads to degeneration and fragmentation of the articular cartilage and underlying bone. In the absence of treatment or spontaneous regression, the osteochondral fragment gradually separates from its bony bed, eventually forming a loose body. Four stages of OCD are recognised:

   - Stage I: small area of compression of subchondral bone
   - Stage II: partially detached osteochondral fragment
   - Stage III: completely detached fragment that remains within the underlying crater bed
   - Stage IV: a displaced osteochondral fragment (loose body)

1.3. OCD affects two distinct populations with lesions occurring in skeletally immature children with open growth plates (known as **juvenile osteochondritis dissecans**) and in skeletally mature adults. OCD arising in adulthood has a more unpredictable course and is more likely to require surgery.

1.4. The precise incidence of OCD is uncertain. One study reported an incidence during teenage years at around 29 per 100,000 in males and 19 per 100,000 in females. The estimated incidence in adults at 30-60 cases per 100,000. A study that included all ages but focused solely on OCD of the knees reported 15-21 cases per 100,000.[1,2]

1.5. The term **"osteochondrosis"** is used to describe a diverse group of disorders, which are separate from osteochondritis dissecans. A common feature of the osteochondroses is a predilection for the immature skeleton, although complications may come to light in adulthood. Osteochondrosis can occur at any epiphysis, although the long bones are particularly vulnerable. The condition can involve the articular surface, the epiphyseal plate, or the apophysis (secondary ossification centre or site of ligament or musculotendinous attachment). These areas are significantly weaker than surrounding soft tissue structures and are particularly vulnerable during growth spurts when musculotendinous tightness contributes to apophyseal disorders.

1.6. In the osteochondroses, ossification is disrupted at a previously normal site of growth. This process can lead to fragmentation, collapse, and sclerosis at the affected site, whilst subsequent re-ossification may result in an alteration in bone contour. Three major categories of osteochondrosis have been identified:

   i. disorders characterised by osteonecrosis, occurring either as a primary event or secondary to trauma;
   ii. conditions related to trauma or abnormal stress, without evidence of osteonecrosis; and

iii.   alterations that represent variations in normal patterns of ossification.

1.7.   The ICD-10 classification identifies more than 20 specific anatomical sites of osteochondrosis that are known by eponyms.[3] A list of eponyms appears at appendix A, whilst several of these specific entities are discussed in more detail in section 3.3.

2. Clinical features

2.1. Osteochondritis dissecans can occur in any joint and on any part of the articular surfaces. The condition occurs most commonly in the distal femur (knee), which is affected in around 75% of cases. Other commonly affected sites are the talus (ankle), capitellum of the humerus (elbow), and femoral head (hip). The condition has also been described in the shoulder, trochlea, wrist, patella, and distal tibia. OCD may occur bilaterally or affect different joints in an individual.

2.2. Over 80% of OCD lesions of the knee involve the medial femoral condyle, most of these being found at the "classical" site at the lateral aspect of the condyle. Approximately 15% of lesions affect the lateral femoral condyle, whereas patellar lesions account for fewer than 5%.

2.3. OCD usually presents with progressive symptoms of activity-related pain. Osteochondral fragments may produce symptoms of locking, clicking, catching, and giving way. Physical examination may reveal local tenderness, swelling, effusion, crepitus, and limitation of movement. In some cases, OCD is asymptomatic and lesions are detected as an incidental finding when an x-ray has been obtained for an unrelated reason.

2.4. Investigations for OCD include:

- X-rays: plain radiographs or specialised views (e.g. tunnel or notch view of the knees)
- Serial bone scans (scintigraphy) can provide an indication of the healing potential of the osteochondral fragment
- Magnetic resonance imaging (MRI) is particularly helpful in evaluating lesions and determining treatment
- Computed tomography (CT) may also prove helpful in pre-operative planning

2.5. Features of several of the eponymous conditions that are classified as osteochondroses are described separately in section 3.3.

3.   Aetiology

3.1.   Osteochondritis dissecans can occur at any age and is around twice as common in males as in females. The precise aetiology is unknown. The most widely held theories implicate trauma, ischaemia, and genetic predisposition. These factors may act singly or in combination.

3.1.1.   **Trauma.** Separation of an osteochondral fragment may be associated with **direct trauma** but, in the majority of cases of OCD, a clear history of acute antecedent trauma cannot be identified. **Indirect trauma** may play a role at certain anatomical sites. **Repetitive microtrauma** may be involved, either in isolation or subsequent to an initial acute macrotrauma. The suggestion that repetitive microtrauma is implicated could explain the increased incidence of OCD that is observed in children who are active athletically and who train for, and participate in, organised sports.

3.1.2.   **Ischaemia.** The vascular supply to subchondral bone is considered tenuous because the terminal branches of the arterioles supplying this tissue form poor anastomoses with their neighbours. It has been suggested that OCD may develop when end artery obstruction occurs, leading to necrosis of a wedge-shaped piece of bone immediately beneath the articular cartilage. A zone of granulation tissue develops between the viable bone and the necrotic wedge, the latter being held in place by intact overlying articular cartilage. Additional trauma causes the articular cartilage to fracture, leading to loosening and detachment of the wedge.[4] However, doubt has been expressed by some authorities as to whether ischaemia acts as an initial event leading to OCD, especially as histological studies have revealed OCD fragments that are not necessarily necrotic.[1]

3.1.3.   **Genetics.** Convincing evidence for OCD as a heritable condition has yet to be established.[5] Some case reports have described a familial pattern, often in association with short stature. However, other studies have failed to establish a familial background.[6] For most patients, a genetic link, if present, is likely to play a relatively minor causative role.

3.2.   The predominant causative factor of OCD may vary depending on the site affected:

3.2.1.   **Knee.** OCD of the knee can present at any age, but is rare in patients younger than 10 years of age or older than 50. About 40% of patients give a history of previous knee trauma, in some cases of a minor nature. In theory, direct trauma to the patella could be transmitted to the classic site of OCD on the lateral aspect of the medial femoral condyle. However, mathematical models have suggested that the transmitted force necessary to produce a fracture at this site is greater than that which could occur clinically. Therefore, acute trauma, when it occurs, appears to be incidental rather than causative.[7] Indirect trauma is considered a more likely cause of OCD of the knee. Indirect trauma could arise from repetitive impingement of the tibial spine on the lateral aspect of the medial femoral condyle during internal rotation of the tibia. The increased incidence of juvenile OCD of the knee in children and adolescents who are heavily involved in sports adds support to the repetitive microtrauma aetiology.

Observations of familial incidence could be related to anatomical peculiarities found in those families. Indirect trauma resulting from internal derangement, such as patellar dislocations, meniscal tears, and instability syndromes, has also been linked to OCD. However, the association remains controversial, and in one series no relationship was found between OCD and trauma, patellar dislocations, or abnormally large tibial spines.[4]

3.2.2. **Ankle.** Direct trauma is more widely accepted as a prime cause of OCD of the ankle. The average age at presentation of OCD of the ankle is 15-35 years and approximately 90% of patients reveal a clear history of previous trauma to the affected ankle. Just under half of the lesions are found in the middle third of the lateral portion of the talus. It is considered that all these lesions occur secondary to trauma, and experimental work has implicated inversion and strong dorsiflexion as the mechanism of injury. These lateral lesions tend to be symptomatic, rarely heal spontaneously, and develop arthritis early. Just over half of OCD ankle lesions affect the medial portion of the talus, usually in the posterior one third. The mechanism of injury in medial lesions appears to be inversion, plantar flexion, and lateral rotation of the tibia on the talus. However, not all medial lesions are associated with trauma, and the aetiology at this site may be multifactorial. Medial lesions produce fewer symptoms than lateral lesions, frequently heal spontaneously, and develop little or no arthritis.[8]

3.2.3. **Elbow.** OCD of the elbow occurs typically between the ages of 11 and 16 years, predominantly affecting the dominant arm. Occasionally, a single traumatic insult to the elbow may be recalled. More commonly the condition arises in individuals who are engaged in gymnastics, racquet and throwing sports (including baseball pitchers in the US), activities that produce valgus stress on the elbow and lateral compression of the radio-capitellar joint.[5,9] This distribution suggests a link to repetitive microtrauma, occurring in the setting of a tenuous blood supply to the capitellum of the developing elbow. The typical age distribution of OCD of the elbow may be explained by the fact that the capitellar epiphysis in the developing elbow receives its blood supply from one or two isolated vessels until the age of approximately 19 years. Subsequently, the metaphyseal vascular anastamoses make a more significant contribution to the circulation.

3.2.4. **Hip.** OCD of the hip rarely arises as an isolated entity. More often it develops following Legg-Calvé-Perthes disease, being reported in 2-4% of patients with this condition (see section 3.3.1). Although a loose body is only rarely found, OCD of the hip almost always involves a weight-bearing surface and, consequently, late osteoarthritis is a frequent complication.

3.3. The **osteochondroses** are found most commonly in children and adolescents, with males affected three times more frequently than females. Aetiological factors are thought to be mechanical (macro- or microtrauma), vascular, and genetic. The principal causal mechanism varies according to the site involved.

I. Disorders characterised by osteonecrosis, occurring either as a primary event or secondary to trauma

3.3.1. **Legg-Calvé-Perthes disease** presents most commonly in boys aged between 4-10 years, causing pain, limp, and limitation of hip movement. The condition is

generally attributed to an interruption of the blood supply to the capital femoral epiphysis leading to avascular necrosis. It has been suggested that thrombophilia may promote venous thrombosis in the femoral head and a number of thrombophilic risk factors have been linked to Legg-Calvé-Perthes disease, although the evidence is conflicting. The conditions that have been implicated include factor-V Leiden mutation (associated with activated protein C resistance), anticardiolipin antibodies, and inherited deficiencies of protein C and protein S.[10,11,12] Inherited fibrinolytic disorders, which reduce the body's ability to break down clots, may also facilitate venous thrombosis in the head of the femur. Reduced fibrinolytic activity has also been linked to passive exposure to cigarette smoke, an association that may explain an observed increased incidence of Legg-Calvé-Perthes disease in children who have been exposed to second-hand cigarette smoke.[13,14] Apart from these vascular causes, a history of trauma is elicited in approximately 25% of cases, leading to suggestions that trauma, with compression of the femoral head by the adjacent acetabular roof, may play a part in the aetiology.

Disease progression is variable and complete recovery may occur. Osteochondritis dissecans develops in 2-4% of individuals with Legg-Calvé-Perthes disease (see section 3.2.4).[15] Legg-Calvé-Perthes disease may lead to the development of osteoarthritis of the hip in early adulthood. However, in many cases, progression may be prevented if the predisposing deformity can be corrected before the onset of degenerative changes. Surgery is a major undertaking and caution is always exercised before embarking on this course. In one study of unilateral disease treated non-surgically with a weight-relieving sling or harness and followed-up for an average of 22.4 years, 80% had good results, 11% demonstrated unsatisfactory radiological appearance but were free of symptoms, and 9% had both an unsatisfactory radiological appearance and significant symptoms.[16]

3.3.2. **Coxa plana**: As a late complication of Legg-Calvé-Perthes disease, a child will be left occasionally with a malformed femoral head, usually either a large mushroom shape (coxa plana) or a lateral protuberance of the head outside the acetabulum.

3.3.3. **Freiberg's infraction** usually presents between the ages of 13-18 years. The condition affects the head of the second metatarsal bone, although the heads of the third and fourth metatarsals may occasionally be involved. It is more common in females and has been linked to the wearing of high-heeled shoes at an early age. Freiberg's infraction may give rise to loose bodies, deformity of the metatarsal head, and secondary degenerative joint disease. Surgery may be required.[16]

3.3.4. **Kienböck's disease** of the carpal lunate is uncharacteristic of the osteochondroses in that it has been reported in children, adolescents, adults, and the elderly. It occurs most frequently in the dominant hand of people in the 20- to 40-year age range. In this age group, the condition affects more men than women, and is diagnosed most commonly in patients who perform heavy manual work. The lunate may be predisposed to injury and subsequent osteonecrosis because of a vulnerable blood supply and its fixed position in the wrist. It is postulated that a short ulna (known as minus or negative ulnar variance) may lead to increased application of force to the radial side of the

wrist and lunate bone, although this association with Kienböck's disease remains controversial.[17] Other contributory factors may include a significant traumatic event, minor repeated trauma, and genetic predisposition. It is plausible that strenuous use and repetitive minor trauma are not primary causes of Kienböck's disease, but factors that cause symptom aggravation of already present disease. Treatment options include immobilisation, revascularisation, and joint-levelling procedures. Kienböck's disease may eventually proceed to osteoarthritis.

3.3.5. **Köhler's disease** of the tarsal navicular occurs in childhood, especially in boys between the ages of 3 and 7. A possible mechanism is compression of a dominant artery supplying the tarsal navicular bone. Köhler's disease is usually self-limiting and operative treatment is rarely indicated.[16]

3.3.6. **Panner's disease** of the capitellum is a rare disorder that usually affects the dominant elbow in boys younger than age 10 years. A suggested cause involves alteration in the vascularity of the developing capitellum. The natural history is benign and self-limiting with eventual restoration of normal appearance in the vast majority of patients.[5] Panner's disease and osteochondritis dissecans of the capitellum may represent a continuum of disordered endochondral ossification with presentation and prognosis dependent primarily on age at onset.[18]

II. Conditions related to trauma or abnormal stress, without evidence of osteonecrosis

3.3.7. **Scheuermann's disease** is the most common cause of structural kyphosis in adolescence, predominantly affecting individuals in the 13-17 year old range. The condition is estimated to affect 0.4-8.3% of the general population. It primarily affects the thoracic region and diagnosis requires the radiographic presence of 5° or more of anterior wedging in at least 3 adjacent vertebral bodies. When adolescents present for treatment, it is often due to their concern at the development of a progressive cosmetic deformity. Although pain may occur during adolescence, it is a more common feature in adults with a long-standing deformity, who often present with lumbar spondylosis and mechanical low-back pain situated below the deformity. Non-surgical treatment with bracing may be considered, primarily in adolescents. Surgery, where indicated, aims to improve spinal alignment and alleviate pain by achieving an arthrodesis throughout the length of the kyphosis.

Various underlying causes have been proposed including avascular necrosis, hormonal abnormalities, osteoporosis, and growth aberrations, but it seems unlikely that any one of these factors is the sole cause of the condition.[19] The condition may be attributable to mechanical or genetic factors, or a combination of the two. Evidence regarding mechanical factors appears conflicting. An increased incidence has been reported in adolescents who engage in sports that involve jumping, including alpine skiing, ski jumping, and water skiing.[20,21] However, another survey found no convincing link between Scheuermann's disease and sports that involve a lot of potential compression stress, weight lifting, or heavy lifting work outside school.[22] An atypical variant of Scheuermann's disease that affects the lumbar spine appears to be more common in athletic adolescents.[23] With regard to genetic factors, an autosomal dominant pattern of inheritance has been proposed and the genetic aetiology is

lent support by reports that describe the condition in monozygotic twins.[24] The presence of the COL9A3 tryptophan allele (Trp3 allele) has been reported in association with Scheuermann's disease.[25] A link with tall stature has also been postulated.[22,26]

3.3.8. **Osgood-Schlatter disease** of the tibial tuberosity mainly affects adolescents between the ages of 11 and 15 years, with the onset of symptoms often coinciding with a rapid growth spurt. The condition tends to be benign and self-limiting, and is often linked to participation in sports that involve kicking, jumping, and squatting. The aetiology is thought to be traumatic, associated with tensile forces that cause disruption along the site of attachment of the patellar tendon to the tibial tuberosity. The condition usually resolves with restriction of activity or cast immobilisation.

3.3.9. **Sinding-Larsen-Johansson disease** of the patella tends to be benign and self-limiting. The condition is most common in the 10-14 year age range, and has been linked to participation in sports. The cause appears to be a traction tendinitis involving the proximal attachment of the patella tendon with subsequent calcification and ossification.

3.3.10. **Blount's disease** affects the proximal tibia, causing the leg to bow outwards. Infantile, juvenile, and adolescent variants are recognised. The infantile form is the most common, affecting children between the ages of 1-3 years, and developing when the normal physiological bowing of the leg in infants persists and worsens. The cause of the adolescent variety is unknown although an arrest of epiphyseal growth, trauma, or infection may be responsible.

3.3.11. **Iselin disease** affects young adolescents and is caused by a traction epiphysitis of the base of the fifth metatarsal. The condition is associated with participation in running and jumping sports that produce inversion stresses on the forefoot. Treatment is usually non-surgical.[16]

III. Alterations representing variations in normal patterns of ossification

3.3.12. **Sever's disease** of the calcaneus, which occurs most frequently in adolescence before or during the peak growth spurt, and **van Neck's disease** of the ischiopubic synchondrosis are generally considered to represent normal variations of ossification.

4. Prognosis

4.1. Treatment of osteochondritis dissecans depends on the severity and duration of symptoms coupled with the size, location, and stability of the fragment, and on skeletal maturity. It is generally accepted that juvenile OCD has a much better potential for healing with non-surgical treatment than does adult OCD. Non-surgical treatment usually involves the cessation of athletic activity and rest from aggravating activities in the affected extremity. These measures are supplemented as indicated with joint immobilisation, anti-inflammatory medication and rehabilitative exercises. Non-surgical treatment for stable juvenile OCD lesions has been reported as achieving 50%-94% healing rates. Most authorities recommend a trial of at least 3-6 months of non-surgical treatment in cases with a favourable outlook before resorting to surgery.

4.2. Surgical treatment is primarily indicated for skeletally mature patients and those approaching skeletal maturity, those with detached fragments, and those who fail to improve with non-operative intervention. Surgical options include internal fixation of the fragment, drilling of the base of the lesion, debridement of the crater, and excision of loose bodies. Newer procedures that are still being evaluated include autologous osteochondral transfer and autologous chondrocyte transplantation.

4.3. The prognosis of OCD depends largely on the patient's age, the stage of the lesion, and site affected. Both juvenile and adult OCD can lead to premature osteoarthritis.

    4.3.1. **Knee.** Although OCD of the knee occurring before skeletal maturity is generally considered to be associated with a good prognosis, studies have yielded varying outcomes, with one multicentre study reporting that 22% of adolescents had an abnormal knee at follow-up.[27] When OCD of the knee occurs later in life, the prognosis is less favourable as there is a high chance of the person developing degenerative features in the joint.[28] Non-surgical treatment in the adult patient has been shown to accelerate degenerative arthritis.[2] In contrast, patients with juvenile OCD of the knee who have a favourable situation at diagnosis have significantly better results after conservative treatment than with surgery.[27] Degenerative changes are more likely to occur with larger lesions or lesions of the lateral femoral condyle.[4]

    4.3.2. **Ankle.** Osteoarthritis is a less common sequel to osteochondritis of the ankle than that of the knee.[16] The response to non-surgical treatment is better for juvenile OCD than adult OCD, but when surgery is required, adults appear to respond better than juveniles.

    4.3.3. **Elbow.** Approximately half of all patients with OCD of the elbow will continue to experience chronic pain or limitation of movement. The results of surgery are often suboptimal. The outlook for continued sports participation is guarded and degenerative arthritis may develop in the long-term.[9,29]

    4.3.4. **Hip.** OCD of the hip is associated with a substantial risk of developing early arthritis in the joint.[30] Surgery is reserved for patients with severe lesions and disabling symptoms.

4.4. Management of **osteochondrosis** is usually non-surgical, incorporating restriction of activity and immobilisation as appropriate. Overall, most cases are self-limiting, although a poorer prognosis has been linked to larger lesions and an older age at presentation. Features of several of the specifically named conditions have been described in more detail in section 3.3.

5. Summary

5.1. Osteochondritis dissecans is a condition in which a segment of articular cartilage and subchondral bone separates from the remaining articular surface. OCD can occur in any joint and on any part of the articular surfaces. The condition occurs most commonly in the distal femur (knee). Other commonly affected sites are the talus (ankle), capitellum of the humerus (elbow), and femoral head (hip).

5.2. The precise aetiology is of OCD unknown. The most popular theories implicate trauma (direct, indirect, and repetitive microtrauma), ischaemia, and genetic predisposition.

5.3. OCD affects two distinct populations with lesions occurring in skeletally immature children with open growth plates and in skeletally mature adults. OCD arising in adulthood follows a more unpredictable course than juvenile OCD and is more likely to require surgery. Both juvenile and adult OCD can lead to premature osteoarthritis.

5.4. The osteochondroses are a diverse group of disorders, with a predilection for the immature skeleton. Aetiological factors are thought to be mechanical (macro- or microtrauma), vascular, and genetic. Most cases are self-limiting, although complications may arise in adulthood, particularly with Legg-Calvé-Perthes disease and Scheuermann's disease. Kienböck's disease is uncharacteristic in that it occurs most frequently in people in the 20- to 40-year age range with a possible association with heavy manual work.

6. Related Synopses

Osteoarthritis of the Hip

Osteoarthritis of the Knee

Internal Derangement of the Knee

Low Back Pain

Spondylosis

Appendix A – Specific sites for juvenile osteochondrosis and their related eponyms as listed in ICD-10[3]

Anatomical area	Specific site	Eponym
Humerus	Capitellum of the humerus	Panner's disease
	Head of humerus	Haas' disease
Radius and ulna	Lower ulna	Burns' disease
	Radial head	Brailsford's disease
Hand	Carpal lunate	Kienböck's disease
	Metacarpal heads	Mauclaire's disease
Spine	Vertebral ring epiphysis	Scheuermann's disease or Scheuermann kyphosis
Pelvis	Iliac crest	Buchanan's disease
	Ischiopubic synchondrosis	van Neck's disease
	Symphysis pubis	Pierson's disease
Hip	Head of femur (capital femoral epiphysis)	Legg-Calvé-Perthes disease
Patella	Primary patellar centre	Köhler's disease of the patella
	Secondary patellar centre	Sinding-Larsen-Johansson disease
Tibia and fibula	Proximal tibia	Blount's disease
	Tibial tuberosity	Osgood-Schlatter disease
Tarsus	Calcaneous	Sever's disease
	Os tibiale externum	Haglund's disease
	Talus	Diaz's disease
	Tarsal navicular	Köhler's disease
Metatarsus	Fifth metatarsus	Iselin disease
	Second metatarsus	Freiberg's infraction

7. Glossary

acetabulum	The cup-like hollow in the pelvis into which fits the head of the thigh bone (femur). Hence: *acetabular*.
allele	Any one of a series of two or more different genes that occupy the same position on a chromosome.
anastomosis (-es)	A joining of branches of tubular structures so as to make them continuous.
apophysis (-es)	A marked prominence or process on any part of a bone. Hence: *apophyseal*.
arthrodesis	The surgical immobilisation (fusion) of a joint.
articular	Of, or pertaining to, a joint.
autologous	Derived from the patient's own body – of identical genetic content.
autosomal dominant	Requires that only one parent need have the trait (characteristic) in order to pass it to the offspring.
avascular necrosis	Death of tissue due to a depletion of blood supply.
calcaneus	The heel bone.
capitellum	The small rounded eminence on the lower end of the humerus (upper arm bone) that articulates in the elbow with the radius.
cartilage	Connective tissue that is more flexible and compressible than bone.
computed tomography (CT)	An investigation technique that uses a computer to assimilate multiple X-ray images into a two-dimensional cross-sectional image.
crepitus	A grating sensation on movement.
epiphysis (-es)	The part of a long bone from which bone growth occurs. Hence: *epiphyseal*.
femoral condyles	Two bony protuberances, termed medial and lateral respectively, at the lower end of the femur (thigh bone).
fibrinolytic	Relating to the dissolution of fibrin (an insoluble protein formed during the blood clotting process) by enzymatic action.
granulation tissue	New connective tissue and tiny blood vessels that form on the surface of a wound during the healing process.

ischaemia	A low oxygen state usually due to obstruction of the arterial blood supply or inadequate blood flow.
ischiopubic synchondrosis	The cartilaginous joint in a child between the ischium and pubis (bones of the pelvis).
kyphosis	Increased posterior convexity of the spine when viewed from the side.
lunate	One of the carpal bones of the wrist.
magnetic resonance imaging (MRI)	An investigation technique used to image internal structures of the body, particularly soft tissues.
metaphysis	The conical section of a long bone between the diaphysis (main shaft) and epiphysis (q.v.). Hence: *metaphyseal*.
navicular	One of the tarsal bones of the ankle.
necrosis	Changes indicative of cell death. Hence: *necrotic*.
osteochondral	Composed of bone and cartilage.
osteonecrosis	A condition in which derangement of the blood supply to an area of bone leads to cell death and subsequent degenerative changes.
scintigraphy	A diagnostic procedure that entails the administration of a radioactive material with an affinity for the tissue of interest. The distribution of radioactivity is subsequently recorded.
sclerosis	Pathological hardening or thickening of tissue.
subchondral	Beneath the cartilage.
talus	The bone in the ankle that articulates with the leg bones to form the ankle joint.
tendinitis	Inflammation of tendons and tendon muscle attachments.
thrombophilia	An increased tendency to thrombosis. Hence: *thrombophilic*.
trochlea	The end of the humerus that articulates in the elbow with the ulna.
valgus	A force acting in an inward direction: i.e. towards the midline of the body.

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