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Definition and classification of fracture non-unions

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Summary Classifications in general provide relevant information for clinical purposes to compose a suitable treatment strategy and for research purposes to be able to define comparable study groups. Two distinct types of non-unions are described in the established literature. In the first type the ends of the fragments are hypervascular or hypertrophic and are capable of biologic reaction. In the second type the ends of the fragments are avascular or atrophic and are inert and incapable of biologic reaction. Hypervascular as well as avascular non-unions may be complicated by the presence of infection, poor soft-tissue quality, short peri-articular fragments or significant deformity, demanding multi-stage treatment strategies with concomitant worsened prognosis and subsequent increased frequency of amputation.

Introduction - definitions

Although there is a large variation in the healing time of fractures in different sites of the skeleton, almost all fractures heal within 3 to 4 months. There is still no generally accepted definition of union, but most clinicians rely on clinical and radiographic examinations.

Unions and non-unions

The clinical criteria used for the assessment of union include absence of motion and pain in response to physiological stress of the fracture and/or the ability of full weight bearing of the involved limb without pain or support. A radiographic criterion that has recently come into use is the presence of a bridging callus in at least three of the four cortices that can be evaluated if radiographs are taken in two transverse levels.[1] Non-unions are defined according to the American Food and Drug Administration (FDA 1988) as "established when a minimum of 9 months has elapsed since injury and the fracture shows no visible progressive signs of healing for 3 months".[2] But this criterion cannot be applied to every fracture:[3] a fracture of the shaft of a long bone should not be considered a non-union until at least 6 months after the injury to allow delayed unions to heal, especially after some local complications such as infection. In contrast, a fracture of the femoral neck can sometimes be defined as a non-union after 3 months. The final status of a non-united fracture is the formation of a synovial pseudarthrosis.

Delayed union, non-union and pseudarthrosis[4]

The distinction between delayed union and non-union is arbitrary and, in fact, there may

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Table 1
Frequent observations in patients with different stages of impaired fracture healing

	Delayed union	Non-union	Pseudarthrosis
Symptoms	Painful	Painful	No pain
Radiograph	Hypertrophic	Hypertrophic or atrophic	Hypertrophic or atrophic
Healing	Spontaneous healing	No spontaneous healing	Only surgical treatment

be no qualitative difference between the two.[5] Fractures showing a persistent fracture line on the radiograph after a period of time are designated as delayed or non-union. These terms are satisfactory only if we understand that they do not necessarily imply static processes or the end of healing potential. The distinction between non-union and pseudarthrosis also is a gliding scale. Chronic or synovial pseudarthrosis is defined as the end stage of non-union, which may take years to develop and which may occur without clinical symptoms or even be intentional such as in certain salvage procedures like Sauvé Kapandji.[6] Frequent observations of clinical, roentgenologic and scintigraphic differences between delayed union, non-unions and pseudarthrosis are outlined in Table 1.

Classifications

Several classifications systems have been introduced in the past. The one described in the famous work by Weber and Ĉech in 1976 has already survived for more than 30 years;[7] it is based upon the differing vitality and healing potential of the various types of non-union. The classification used by McKee in the AO manual[8] is derived from the same principle, with special attention to diaphyseal and metaphyseal non-unions. The classification described by Paley et al.[9] in 1989 is focussed on the tibia but may be applied to non-unions of other long bones as well. It divides non-unions, clinically and radiologically, into two major types depending on the amount of bone loss and the degree of mobile deformity produced. This classification can also be regarded as an interpretation of Weber's principle of hypervascular and avascular non-union.

Hypervascular non-union (Figs. 1, 2)[4,7]

Two main types of non-unions are differentiated in the established literature according to the viability of the ends of the fragments. In the first type the ends of the fragment are hypervascular or hypertrophic and are capable of biologic reaction. Bone scintigraphies in these non-unions indicate a rich blood supply in the ends of the fragments. The fracture line persists beyond the expected time for union and there is callus in variable amounts about the fracture site. The build-up of the external callus is the response of viable bone and periosteum to motion at the fracture site. The degree of motion is often of such low amplitude that it cannot be detected preoperatively. Nevertheless it may be sufficient to prevent bone from forming across the fracture line. Adequate decrease of the micro- or macro-movement below a critical threshold is compulsory for fracture healing. The tissues within the radiographic fracture line, the so-called "empty scaffold", have great osteogenic capacity if only the motion is eliminated.[10] Bone scans in this type of non-union show increased activity at the fracture site, implying vascularity and osteogenesis.[11] It is interesting to note that some of these concepts are very old. In 1842 Astley Cooper[12] wrote: "... is no difficulty, for example, in understanding that the materials effused for the consolidation of a fracture can never be converted into a bony callus, if subjected to frequent motion and disturbance". This is not to say that motion at the fracture site invariably leads to non-union. The excess callus stimulated by motion often serves its purpose bridging and splinting the gap. Weber and Ĉech sub-classified this form of non-union according to the amount of callus (elephant foot, horse foot, and oligotrophic) (Fig. 1). In their oligotrophic form there is no callus, but rather absorption at the fracture ends. The viability of the bone ends characterises all these sub-types of non-union.

(a) 'Elephant foot' non-unions (Fig. 1a). These are hypertrophic and rich in callus. They result from insufficient fixation, inadequate immobilization, or premature weight-bearing in a reduced fracture with viable fragments. The quietly standing empty scaffold of Pauwels is mechanically disturbed and therefore it ossifies only partly at the periphery.[13] The space between the fragments, the fibrous cartilage, however, remains as a gap. When remained untreated, a synovial pseudarthrosis

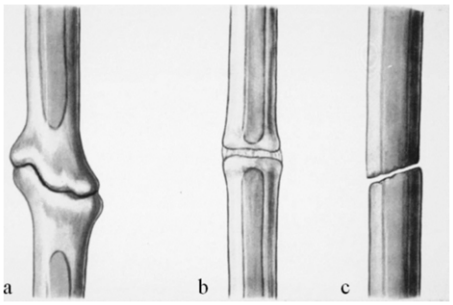

Figure 1. Schematic representation of hypervascular non-unions with vascular supply (left) and morphologic appearance (right): a 'Elephant foot' non-union. b 'Horse hoof' non-union. c 'Oligotrophic' non-union (Weber 1976).[7]

may develop depending on the degree of mobility still present.

(b) 'Horse foot' non-unions (Fig. 1b). These are slightly hypertrophic and poor in callus. They typically occur after a moderately unstable fixation with plate and screws. The ends of the fragments show some callus, insufficient for union, and possible a little sclerosis. The osteosynthesis, usually a plate, becomes loose, and fails secondarily to weight bearing and loading of the limb. Insufficient stability causes 'unquiet' callus (also known as 'irritation callus'). However, callus plus osteosynthesis are not sufficient to stabilise the fragments. The metal becomes fatigued and breaks before enough 'unquiet' callus can form and develop into fixation callus. According to the inherent strength of the callus to produce stability, the non-union turns out to be lax, or rigid, or even unites spontaneously.

(c) 'Oligotrophic' non-unions (Fig. 1c). These are not hypertrophic and callus is absent. They typically occur after major displacement of a fracture, distraction of fragments or internal fixation without accurate apposition of fragments, or when gross bony defects are present. Radiologically 'nothing' happens at first: the ends of the fragments are inert. After 8–12 weeks the edges of the fragments are somewhat rounded off and progressively present radiological evidence of absorption. At the same time, inactivation leads to decalcification. The ends of the fragments are however viable although no signs of callus can be observed and absorption is impressive. The non-union is lax due to the absence of any consolidating tissue structure.

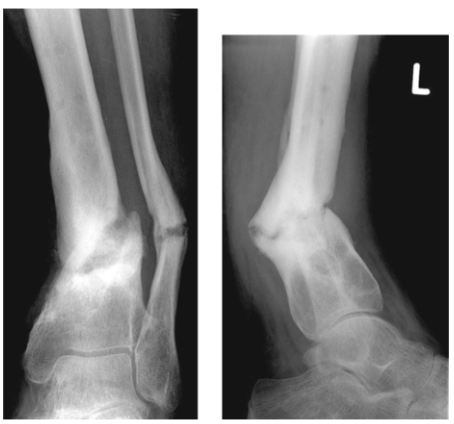

Figure 2. Radiograph in anteroposterior (left) and lateral (right) view of hypervascular non-union eleven months after initial injury.

Avascular non-union (Figs. 3,4)[4,7]

In the second type of non-union the bony fragments are avascular or atrophic, inert and incapable of biologic reaction. Bone scintigraphies in these non-unions indicate a poor blood supply in the ends of the fragments. The fracture lines persist with no demonstrable callus. There may be minimal moulding of the bony ends but the essential radiographic picture is that of no change over a very long period of time. The aetiology in this situation is extensive death of bone usually due to severe comminution and devitalisation of the fragments. In this type, immobilization alone cannot lead to bone union. Biological enhancement basically with bone grafting is necessary in addition to good immobilization. Debridement of the dead bone and the interposed tissues together with bridging between viable bone ends achieved with often large quantities of bone grafts is necessary. Avascular non-unions are subdivided as follows:

(a) Torsion wedge non-unions (Fig. 3a). These are characterized by the presence of an intermediate fragment in which the blood supply is decreased or absent. The intermediate fragment has healed to one main fragment

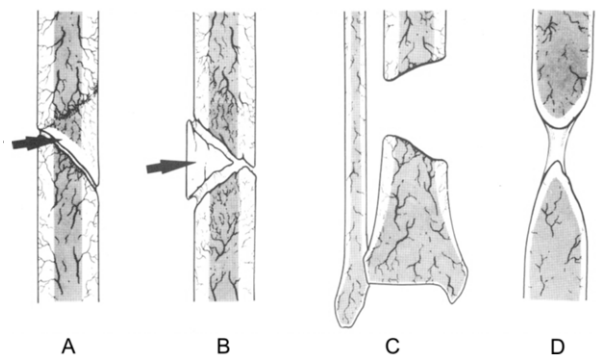

Figure 3. Avascular non-unions: (A) Torsion wedge non-union. (B) Comminuted non-union. (C) Defect non-union. (D) Atrophic non-union (Weber 1976).[7]

but not to the other. These typically are seen in tibial fractures treated by plate and screws. Using intramedullary nailing instead decreases the rate of torsion wedge non-unions considerably.

(b) Comminuted non-unions (Fig. 3b). These are characterized by the presence of one or more intermediate fragments that are necrotic. The roentgenograms show absence of any sign of callus formation. Typically these non-unions result in breakage of the hardware used for the initial fixation. Using intramedullary nailing instead decreases the rate of comminuted non-unions considerably.

(c) Defect non-unions (Fig. 3c). These are characterized by a recent fracture with the loss of a fragment of the diaphysis of a bone either by the accident (defect-open fracture) or later during the treatment period through infection (sequestrums). The ends of the fragments are viable, but union across the defect is impossible. As time passes the ends of the fragments become atrophic.

(d) Atrophic non-unions (Fig. 3d). These are usually the final result of types a, b and c when intermediate fragments are missing, either primarily at the accident (defect-open fracture) or secondary through infection (sequestrums), and scar tissue that lacks osteogenic potential is left in their place. The ends of the fragments have been partially absorbed during the long treatment period. Inactivity has led to corresponding osteoporosis and atrophy of the limb.

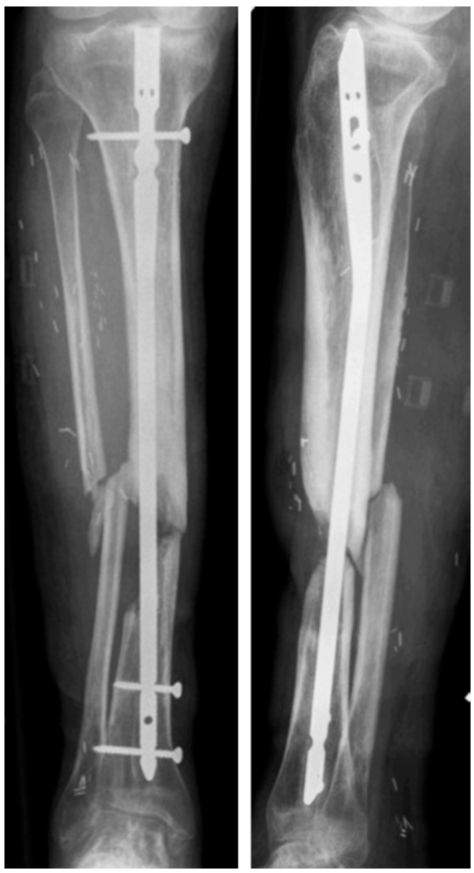

Figure 4. Radiograph in anteroposterior (left) and lateral (right) view of avascular non-union sixteen months after initial injury.

Conclusion

The definition of non-unions in fracture healing is a disturbance of normal healing with the expectation that no consolidation will be achieved without focused and accurate treatment. Classification of non-unions in fracture treatment has not changed during the last 30 years. The concept of differentiation between hypervascular and vascular non-unions designed by Weber and Ĉech in 1976 has proven to be a valuable tool for clinical and research purposes even in 2007.

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