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Etiology and treatment of the united fracture

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Introduction

Fractures of bones have always presented to the medical profession one of its most important problems. It is a problem that has necessarily grown up with civilization. This is necessarily true because the causes are mechanical in nature and hence variable with man's environment. The relative frequency of various types of fractures has been materially changed since the advent of modern machinery and modern methods of warfare. A study of remains from ancient Egypt by Elliot Smith (Garrison) indicates a much lower relative frequency of fractures of the upper extremity than we see today. Not only the locus, but also the type of fractures, have changed. We now see many more of the severe and formerly rare varieties. In the wake of these violet injuries there are a series of complications which much inevitably follow. Among these can be listed compound and comminuted fractures, infections, various severe soft tissue injuries and finally delayed union and non-union. These last complications have become a very real problem to the profession. Their present status and incidence seems to have been reached during the last century. Up to 1800 they are mentioned now and then, but no great attention is given to them. Hippocrates devoted three books of his work to the subject of fractures and

associated injuries. I cannot find any mention of delayed union or non-union in his work (as translated by Adams). Celsus mentioned delayed union and recommended that it be treated by rubbing the ends of the fragments together (Norris 1842). Ambriose Parè, writing in the sixteenth century, refers to the subject several times. Before that period it had been recognized that fractures of the olecranon and patella did not heal by bony union as other fractures did, but became joined by bands of fibrous connective tissue. Parè found that some cases that had been considered as dislocations of the hip were actually fractures inside of the joint capsule and that these fractures seldom unite by bone.

From 1800 on there are available statistics showing the frequency of delayed union and non-union. A comparison of these figures with those of contemporary writers is decidedly interesting. Walker (1815) reported that he had seen six cases of non-union while attending over a thousand fractures. Liston(1836) only saw one case fail to unite. Hammick discharged only three cases from the Plymouth Hospital with ununited fractures (Norris 1842). Hamilton (1863) stated that non-union occurred in one case out of five hundred. According to Agnew (1889) the Pennsylvania Hospital cared for over seven thousand fracture cases between the years 1830 and 1874. They did not have a single case of non-union during that time. The only writer of this period who did not agree that non-union was an exceedingly rare condition was

Amesberry. In 1829 he reported fifty-six cases of non-union which he had seen. This figure drew considerable comment at the time and lead other writers to wonder what kind of surgery was being practiced at Edinburough (Norris 1842).

If the foregoing figures are compared to those of twentieth century writers, the contrast is rather startling. Hey Groves (1930) states that from three to five percent of fracture cases suffer from delayed union and non-union. Arvid (1933) places the incidence of non-union in uncomplicated simple fractures at .23%. Foster (1933) had seven cases in a series of one hundred and seventy-five. Scudder (1926) reports an incidence of two to three percent for delayed union and non-union. H. R. Owen (1932) presents statistics on a series of 11,683 fracture cases. He has one hundred and one non-unions in the series giving an incidence of a little less than one percent. (Still five times greater than Hamilton's). Cubbins and Scuderi (1933) report an incidence of three percent in fractures of the humerus.

Before drawing any conclusions from these figures, it is important to remember several modifying factors. Delayed union is included in many of the modern statistics. This is a relatively common condition, much more so than non-union (Cotton 1928, Eisendrath 1907, Stimson 1905) and by itself probably affects the statistics a great deal. Statistics like those of Cubbins and Scuderi taken from one specific region particularly subject to non-union are also apt to

mislead. On the other hand, if the recent figures be discounted two or three hundred percent they still show a higher incidence than those compiled in the last century. Figures like those of Owens which deal specifically with non-union only are also much higher.

Obviously such an increase in incidence must be due to one of three factors, namely, a change in the human organism, less effective methods of treatment, or an increasing incidence of injuries of a type likely to result in a failure of repair. The first of these three factors is necessarily a purely hypothetical one. It deals with phenomena on which we have no way of checking. The responsibility has been divided almost equally between the other two. Estes (1920) Cotton (1928), Robinson (1928) and Darrach (1933) are inclined to blame the nature of injuries being sustained today from massive machinery and high speed travel. Campbell (1932) and Owens (1932), on the other hand, consider the cause to be too enthusiastic attempts to obtain perfect reductions as shown by the X-Ray. An attempt to get such results leads them to repeated manipulations of fractures which should be severely left alone once a position compatible with good function has been obtained. Whatever the cause of our increasing incidence of poor results may be, the problem is a very important one. At best fractures are productive of long disability and hence considerable economic loss. Add to this loss an additional period of several months or per-

haps years of incapacity and for many people the results are serious. It therefore is up to the medical profession to take stock of themselves and see what can be done to cut down this rising incidence.

The following review of the literature may uncover a few trends in the conception of the etiology and treatment of ununited fractures and help us see where we are going with this problem.

Physiology and Pathology

The method of normal growth of bone and its method of repair are important in any study of what takes place or does not take place when a fracture fails to unite. Writers on these subjects now are divided into two groups, those who hold the cellular theory of bone formation and those who hold the newer biochemical theory. The present controversy is similar to the one started by Duhamel (1741) when he announced that the periosteum is the mother tissue of bone. This theory was attacked by Haller (1763) who claimed that the function of periosteum was nutritive and that it had nothing to do with the actual process of ossification.

Duhamel was sustained by Breschet (1801), Meischer (1836), Vellermé (Cheluis 1843), Ollier(Lerrich and Policard 1928) and, in part, by Dupuytren (1839). Haller's work was defended by Scarpa (1828) and John Hunter (1837). The osteoblastic theory seems to have grown out of this latter conception. It was first proposed in 1845 by Goodsir (Holdeman 1932) and has since become the generally accepted theory. The periosteal theory of Duhamel has continued to receive support and is still held in a modified form by Blaisdell and Cowan (1926), Cowan (1928) and Holdeman (1932). These men, however, have also accepted the osteoblast as the means by which the periosteum works. They describe a thick layer of these cells on the deep surface of the periosteum of young animals.

The osteoblastic theory as it is usually thought of today is briefly as follows: Bone is considered to be a highly specialized form of connective tissue (first taught by Reichart 1854 (from Lerrich and Policard 1928)). The cellular elements have become specialized and endowed with the specific power of laying down calcium salts in the matryx with which they are surrounded. It is to these cells that the term osteoblast has been applied. They are responsible for all deposition of bone and as bone cells they have the function of nourishing this tissue once it has been formed. Osteoblasts are to be found in three localities, the osteal surface of the periosteum and endosteum and as isolated bone cells in the lacunar spaces of the bone itself.

The other modern theory of a biochemical process of bone formation has been brought out by two Frenchmen, Lemrdch and Policard. These men started an extensive study of bone from all possible angles. At first loyal supporters of Ollier, they have since tried to upset all of the established conceptions of the physiology of bone. Their present conception is that bone represents a specific phase of connective tissue metabolism rather than a result of cellular differentiation. According to them, a mass of connective tissue young and vascular, actively growing and more or less edematus, constitutes an ossifyable medium. Add to this a local concentration of calcium salts high enough (their so called calcific surcharge) and bone will always be formed. This

accounts for the possible formation of bone in the kidney, the muscles, and other out of the way places where it is sometimes seen.

The biochemical theory has become increasingly more popular since it first appeared in 1926. Bancroft (1926) was one of the first to accept this view. The most active exponent of the biochemical theory in this country has been C. R. Murray (1930-31).

With the understanding of the possible sources of bone growth, the question of healing of fractures began to be at least partially understood. Before the time of Duhamel, fracture healing was thought to be a process quite similar to the glueing together of sticks of wood. The broken ends of the fractured bones were supposed to exude a viscid juice which stuck the fragments together. The so called osteol juice then acquired substance and the union gradually became solid. Haller (1764) thought that callus was a jelly like substance produced in the marrow cavity and the fractured end of the bone. This jelly went through a process of organization and chondrification and finally became bone.

John Hunter thought the blood clot thrown in between the fragments became organized and then transformed into bone. Chelius (1843) accepted this view. Dupuytrens (1839) first introduced the differentiation of the callus into two early temporary parts and one definitive or permanent portion. The provisional callus he located under the periosteum and

and in the medulary cavity respectively. This conception is still held by many of the modern authors. Breschet, Villermé and Meischer studied the fermentation of callus quite completely. According to Norris (1842) it was the best work on fractures up to that time.

All of the then existing theories were taken up by the osteoblastic theory soon after it was offered and the conception of callus formation became quite stable until the advent of the biochemical theory.

All authors agree that the first thing that happens when a bone is fractured is the formation of a blood clot between the fragments. Blood vessels in the medulary cavity, the cortex of the bone and at times in the surrounding soft tissues are disrupted. The clot comes as a result of this vascular injury. This vascular injury sometimes sets up more or less disturbance in the circulation of the bone and may have profound effect on the later steps of callus formation.

The blood clot soon begins to be invaded by granulation tissue which carries with it new blood vessels. These vessels run at right angles to the Haversion systems of the bone. The source of the granulation tissue is a matter of some dispute. According to Lerrich and Policard (1926 and 1928) and Murray (1930) it comes from all available tissue sources, namely, the medulary cavity, the cortical bone, the periosteum and the surrounding muscle and fascia. To

others (Holderman 1932) the chief source of this tissue is the periosteum. At all events, the original blood clot becomes organized and replaced by a vascular young connective tissue which now fills the space between the fragments and may invade the surrounding tissue to a greater or less extent. From this point on, the various schools of thought begin to diverge on what happens.

According to the holders of the osteoblastic theory specialized cells now begin to migrate into the granulation tissue and line up along the course of the newly formed blood vessels. Holderman (1932) and Cowan (1928) maintain that the periosteum is the one important source of these cells. Those cells in the bone laminae have died due to the loss of their blood supply. To the endosteum they attribute very little osteogenetic power. Kolodomy (1923 A) believes that the endosteum is endowed with osteogenetic powers. He states, however, that it cannot function in this way until its blood supply has been restored through the new vessels in the developing callus. Campbell (1932) considers the endosteum and periosteum of equal importance in the formation of new bone. Extensive injury to either of these areas, he believes is deleterious to the progress of the callus.

The osteoblasts around the new blood vessels begin to deposit layers of bone and there are soon formed Haversian systems running, like the new vessels, at right angles to the old system in the cortical bone. When union is solid

and function is restored new stresses and strains begin to fall on the new bone and an adaptive response begins to take place. The new Haversian systems change their alignment and assume one better adapted to meeting the new stresses and strains. The property of bone to respond in this way was described by Wolff (1868) and definitely proved in the case of the femeur by Koch (1917).

Let us go back now and follow the organized clot in the fracture in the way Lerrich and Policard (1928), Bancroft (1926) and Murray (1930) lead us. They see in the whole process a simple fate of connective tissue which can occur in any part of the body given the proper conditions. Bearing in mind the necessary conditions set by Lerrich and Policard; eg. an ossifiable medium and a local calcific surcharge one can see that conditions are ideal in the fracture area. The succulent connective tissue medium just formed and still edematus is the ossifyable medium. The divitalized fragments of the broken bone, undergoing autolysis supply the calcific surcharge. According to Murray (1930) there are two additional factors necessary. These are surrounding devitalized tissue and a Ph, proper for the precipitation of calcium salts. These two conditions also exist at the fracture site. The first is due to the initial injury and the second to the vascular reaction which follows. The architecture in the callus is restored to normal through the molecular reaction of the bone to stress and strain.

When a fracture fails to heal in the usual length of time, or perhaps does not heal at all, it is obvious that the foregoing process of repair has been interrupted or altered at some stage. This interruption may occur at any point (Campbell 1932).

, In general the nature of the alteration of callus formation will determine whether a case is to be classed as non-union or a delayed union. As long as the fracture shows a normal picture of some stage in the process of healing it cannot, according to Cotton (1928) be considered non-union no matter what the date may be. John Hunter (1837) and Amesbury (1829) report that cases of very long standing may unite. Henderson (1926 A) states that union has occured in cases of more than a year's standing. For this reason Eisendrath (1907), Scudder (1926) and Cotton (1928) call a case non-union only when repair has become altered in such a way as to make consolidation definitely impossible. Stimson (1905), Forrester-Brown (1927) Shearer (1931) and Henderson (1926 A) do not go quite so far. They consider a case as definitely one of non-union when all clinical and roentgenological evidence of repair has ceased and the condition becomes a stable one.

It might be well to mention at this point that many clinicians establish a diagnosis of non-union on a purely chronological basis. Ely (1922) calls fractures ununited after thirty days. Foster (1933) states that a fracture not

completely healed in six months is to be considered as non-union. From a practical point of view as we shall see later such a distinction may be justified. On the other hand, the term non-union had better be used rather cautiously for cases six months old since some of them can and do unite. The term ununited fracture is a more accurate one to use as long as consolidation is possible.

Just what happens in the area surrounding a healing fracture to cause repairs to be prolonged is rather hard to say. A pathological description is of little value since there really is no pathology. What one finds in a delayed union is a normal process slowed or stopped.

In non-union (using this term in its limited sence) on the other hand there is a pathological picture. Here the process of repair has gone on but has been altered by some complicating factor (probably local). Thus one may find soft tissues caught between the fragments preventing any callus from uniting them; one fragment may become completely devitalized and absorbed (as many writers show in the case of the femoral neck), poor blood supply or slight injury may leave a fracture with no blood clot and no stimulus for repair (Potts 1933) or slight motions of the reduced fragments may break the forming callus and allow dense fibrosis to crowd out bone formation (Lerrich and Policard 1928) (Jones and Roberts 1934).

According to Murray (1930 and 1931) the process of bone autolysis may become extensive and leave a wide separation of the fragments. Mudd (1896) mentions a case where the whole humerus was transformed into a fibrous cord.

The true pseudarthiosis represents the most extreme step that altered repair can take. This condition was known by 1800 for Sir Astley Cooper (1832), Crovelhire (1842) and others of this period speak of it. Boyer (1822) and Chelius (1843) did not think that there was such a thing. The term pseudarthiosis is used according to Eisendrath(1907) and Cotton (1928) both to those cases in which this is deposition across the fracture line, and perhaps a certain amount of cartilage formed between the fragments and those cases in which a complete new joint with a synovial cavity is developed. Cowan (1928) believes that pseudarthiosis is due to pressure and trauma to a pre-existing fibrous union.

It is interesting and important to note that the pathology of non-union does not include a failure of osteogenesis. It is simply a prevention of normal healing by some local complication which makes it impossible.

Henderson (1926 A) has stated that fractures of over a year's standing may unite. He also states that non-union can definitely be diagnosed in other cases at the end of three months. I really believe that a study of the pathology of delayed union and non-union indicates that they are not varying degrees of the same thing. They are rather separate and distinct entities having different causes, differentiat

reactions and running a different course.

Etiology

Ununited fractures may be the result of either general or constitutional distmrbances of the patient or of complications located at the site of the injury.

The consitutional causes which have been suggested are age, general condition including nourishment, various endocrine disorders, food deficiencies, faulty metabolism, pregnancy and disease. Among the diseases, syphilis, tuberculosis, diabetes, gout, chronic arthritis, chronic nephritis, all diseases of the bone, and the blood disturbances have bean blamed.

Age is, and always has been, considered to be an important factor in the prognosis of fractures. It is known, however, that ununited fractures are found in all ages. Mudd (1896) denies any influence of age on the occurance of non-union. Owens (1932) and Arvid (1933) state that the condition is rare in children. Most writers believe that ununited fractures are most frequently seen in young adults. This does not indicate any material effect of age on healing as this is the period of life when most fractures occur. (Cotton 1932, Eisendrath 1907, Stimson 1905).

Owen's cases of non-union were distributed mostly through the third, fourth and fifth decades of life. He had three cases in the first decade and two in the ninth. Whitman (1905) states that fractures in old people will

unite if they are given proper treatment (his results with fractures of the pride of the femeur will prove his point).

Most writers of our time do not mention general nutrition as a cause. Mudd (1896) and Hewson (1828) speak of it. Malnutrition might well be a factor in delayed union that we would hear more about if actual starvation were more common among us.

Pregnancy and lactation have been spoken of as possible causes of delayed union. It is interesting to note that their association with ununited fractures entirely antidates the work on their metabolic effects. Fabricius Hildanus (1687) thought that fractures in pregnant women were prone to slow healing. Hammick (Norris 1842) reported three cases of fractures in women who were in early gestation. All three of them remained ununited until late gestation and then suddenly consolidated. Norris himself doubted the effect of pregnancy on the healing of bones. His opinion is in accord with the more recent work of Stimson (1905), Eisendrath (1907) and Cotton (1928).

Of the diseases, acute infections (except in the locus of the fracture) are not generally thought to be of any importance so far as fractures are concerned. Forrester-Brown (1927) has suggested they may have some effect. Chronic diseases, on the other hand, have aroused much more suspicion. This has been particularly trueof syphilis. The older writers very definitely have considered syphilis to be deleterious

to the healing of fractures. Norris (1842) and Cheluis (1843) report several cases in which they thought it to be a definite cause. Erichsen (1867), Agnew (1889) and Mudd (1896) all give it a place in their textbooks. Estes (1920) and Forrester-Brown (1927) are inclined to consider lues a cause of slow union. According to Cowan (1928) there is a specific toxin definitely altering the course of fractures in luetic patients.

Most modern writers do not believe that syphilis has anything to do with the progress of fractures. Owen (1932) shows in his review of cases that those with a positive Wassermann get along as well as those without. Cotton (1928) states that syphilis is a much better excuse for poor results than a cause for them.

I can find no authority for the belief that the other chronic diseases (diabetes, gout, etc) have any affect on fractures.

There are some other general diseases which are not so easily disposed of as causes of poor healing of fractures. These include the endocrine disturbances and dietary deficiencies affecting bone and the calcium metabolism, and primary diseases of bone such as Pagets and Osteitis fib rosa cystica.

Sir Astley Cooper (1822) noted that ununited fractures were relatively frequent in sea faring men (among whom scurvy was common.) Scurvy, rickets and osteomalacia are considered to cause delayed union by Stimson (1905), Eisendrath (1907)

and Cotton (1928). According to Kolodony (1923 B) the endocrine deficiencies definitely hinder the repair of bones. Peterson (1924) maintains that if the product of the blood calcium and phosphorus falls below thirty, union cannot be expected. Därrach (1933) regards a low blood calcium as a very important cause of poor results.

On the other hand there are plenty of cases in the literature to prove that union can occur in spite of any of the foregoing conditions. Bohler (1929) states that constitutional diseases affecting the bones may delay union but do not prevent it. Murray (1931) reports that fractures in people with rickets, osteomalacia, scurvy and Paget's disease can and usually do unite promptly. In this he is supported by Henderson (1926 B) and Owen (1932) Henderson, Noble and Sandeford (1926), Ravdin and Jonas (1926), Lacy (1929) and Cuthbertson (1930) have failed to confirm Peterson's findings on blood calcium.

The present trend of opinion is rather definitely away from general factors as a cause for non-union and to a less extent for delayed union. Henderson (1926), Owen (1932) Eliason (1932) and Jones and Roberts (1934) maintain that they have no effect at all on the repair of fractures.

Granting perhaps, the possibility of delayed union upon a constitutional basis, it is among the local factors that one must look for the important cause of non-union.

Probably the most important single factor in the etiology of ununited fractures is the location of the break.

"Non-union and delayed union occur in places not in people" (Murray 1931). Omitting for the moment the olecranon and the patella where fibrous union is so common that it is usually considered the rule (Stimson 1905, Eisendrath 1907 and Treves 1917) and the neck of the femeur which is influenced by several unique anatomical factors, the commonest site of both delayed union and non-union is the shaft of the humerus (Norris 1842, Chelius 1843, Agnew 1889, Mudd 1896, Eisendrath 1907, Treves 1917, Estes 1920 and Cotton 1928). This point is a focus of several anatomical conditions all of which have been pointed out as the chief cause for non-union. There is not a great mass of soft tissue here below the insertion of the deltoid muscle and abbve the origins of the medial head of the triceps and the brachialis. The bone is very hard to fix completely because of the mobility of the pectoral girdle and the nutrient artery enters the bone at a favorite point of fracture. There is less complete agreement as to the next most frequent site of ununited fractures. According to Mudd (1896), Abbott (1922) Henderson (1926 B) and Murray (1931) it is the tibia. Treves (1917) places the radius next while Norris (1842), Agnew (1889), Eisendrath (1907) and Estes (1920) think it is the shaft of the femeur. There is no doubt but that these sites together with the navicular of the wrist, the ulna, the clavicle, and the mandible include most all of the cases. (Lerrich and Policard state that fibrous union is very common in skull fractures. Considering the

great disability produced by such a non-union we should expect to hear more about these cases in the future).

The intracapsular fracture of the neck of the femeur is a problem by itself. Like the shaft of the humerus it is a focus of predisposing factors for non-union. Case for case, these fractures are five times as apt to end in non-union as fracture of the humerus if the figures of Wallinsky (1922) for the femeur and Cubbins and Scuderi (1933) for the humerus are to be credited. Sir Astley Cooper (1822) stated that fractures of the femeur within the joint capsule never unite by bone. Certainly they did so rarely that the cases of Stanley (1833) and Amesbury(1829) were regarded as clinical curiosities. Estes (1920) made a study of the possible danger areas within certain bones. He places most of the cases of the humerus in the middle third of the shaft, those of the femur in the neck, delayed union in the upper end of the tibia and non-union in its lower end.

By contrast to the foregoing sites, ununited fractures are practically unknown in the ribs and the sternum.

The degree of violence of the injury producing a given fracture is very important in its prognosis. In the wake of the more violent injuries are such complications as multiple fractures, comminuted fracture, impacted fractures, compound fractures and a variety of soft tissue injuries. Stimson (1905) Cotton (1928), Owen (1932), Campbell (1932) and Darrach(1933) point out that such complications are

particularly apt to be followed by non-union.

Comminuted fractures are apt to be accompanied by wide spread devitalization of bone, periosteal stripping and disturbances of the blood supply to the ends of the fragments. Any of these complications may interfere with the future progress of healing.

Compound fractures are especially prone to non-union. This is due in part to the high incidence of infection in such cases. Foster (1933) reports at best two cases of non-union out of a series of one hundred and twenty-seven compound fractures. Another series of his had seven non-unions in one hundred and seventy-five cases. According to Arvid (1933) seven percent of compound fractures fail to unite. Estes (1920) and Darrach (1933) consider compound fractures as an important source of ununited fractures.

Various soft tissue injuries are said to interfere more or less with the healing of fractures. According to Mudd (1896) and Stimson (1905) injuries to the local nerve supply are apt to affect progress. Stimson maintains that this is true only where the fracture is severed from its trophic center. This is in accord with the more recent theory mentioned by Campbell (1932) and discussed by Colp, Kassabach and Mage (1933) that local vaso-motor upsets through the medium of the sympathetic nervous system may have a profound effect on healing. Owen (1932) denies any importance of associated nervous injury and other authors make only

casual mention of it.

Stripping of the periosteum from the fragmented bones is generally given as one of the most important of the local causes. In the light of the foregoing opinions on the nature of the process of healing the importance of this complication is obvious. To those holding the biochemical theory it means the loss of one of the important sources of blood supply, a source of granulation tissue and a later overgrowth of fibrous connective tissue. To the adherents of the osteoblastic theory it means all of and the this loss of one of the most important sources of bone forming cells.

According to Lerrich and Policard (1926 & 1928) the most important effect of periostal injury on a healing fracture is the loss of a membrane which should prevent the infiltration of blood and serum into the surrounding tissues. The false cyst thus formed block the growth of granulation tissue into the interval between the fragments. With organization blocked the formation of callus is impossible and a non-union is the result. The functioning of the periosteum as a limiting membrane is held by Cowan (1928) and Blaisdell and Cowan (1926) They consider the interruption of a continuous periosteol bridge between the fragments as very important.

According to some (Kolodony 1923 A and 1925, Blaisdell and Cowen 1926, Cowan 1928, Ely 1922 and Robinson 1928) periosteol injury cuts off an important source of blood supply to the fracture area and to the adjacent bony fragments.

Kolodony (1923A) considers this isolation of the cortical bone and the medulary cavity from their blood supply (until an anastomotic supply can be set up through the fracture) as one of the most important causes of non-union. According to him the interval during which the endosteum is thus made inactive is ample to allow fibrosis to get ahead of callus formation.

A poor blood supply to the fracture, due either to periosteol and vascular injuries, compression, from tight dressings and swelling and vaso-motor spasm, or to fracture in a relatively avascular area, is probably a very productive cause of non-union. Norris (1842), Erichsen (1869), Mudd (1896), Stimson (1905), Cotton & Loder (1913), Henderson (1918, 1926 A & B), Eliason (1921), Blaisdell & Cowan (1926) Cowan (1928), Lacy (1929), Albee (1930), Fite (1931) and many others consider the blood supply to be a very important factor in healing. According to Estes (1920), Campbell (1924 & 1932) Kolodony (1925), Murray (1930) and Darrach(1933) vascular inadequacy is the principal cause of non-union. Kolodony (1923 & 1925) and Bozan (1932) believe that the sole cause of non-unions of the femoral neck is the poor blood supply. Kolodony has shown that the neck of the femeur inside of the joint capsule receives its blood from three sources, the periosteum, the diaphyseal vessels and epiphyseal vessels. The latter channels reach the bone through the round ligament. In older people they become progressively smaller and often

disappear. When a fracture occurs the other two sources of blood are cut off from the proximal fragment. The joint capsule cuts off vessels from growing in from the surrounding muscle leaving on isolated proximal fragment with no blood supply.

That the destruction of the nutrient artery is of importance is not universally agreed. Estes (1920), Cotton (1932) and Eisendrath (1907) regard it as very important. Lacy (1929) claims he has produced the clinical picture of non-union in experimental animals by fracturing bones through the nutrient artery. Kolodony (1923A & 1925), Cowan (1928), Murray (1930 & 1931) and Campbell (1932) on the other hand do not believe that loss of the nutrient artery is serious. To them the important blood supply of a bone comes through the periosteol vessels and vessels in the surrounding soft tissues. They point out that the cortical vessels and usually the medulary vessels are ruptured and thrombosed at the time of injury. This produces exactly the same circulatory effect as destruction of the main vessel.

Drinker, Drinker and Lund (1922), Johnson (1927) and Robinson (1928) have carefully studied the circulation of bone. Johnson describes the blood supply of the tibia as coming from three sources. These are the periosteol vessels, the metaphyseal vessels and the nutrient vessel. Robinson describes the same series of vessels and points out that there is free anastamosis between all of them. That such

an anastamosis is complete is doubted by Drinker, Drinker & Lund who were unable to completely perfuse the tibia by injections into the nutrient artery. Johnson states that the nutrient vessel alone is capable of completely supplying the bone. He believes that its injury is a serious complication to a fracture. The other authors think that the bone can carry on and heal without this source. Robinson believes that the venous drainage of the fractured bone is just as important as the arterial supply. This, however, is less frequently disturbed because the vessels do not become completely obstructed and there is a freer collateral net work.

Cotton (1928) and a few others think that the direction taken by the nutrient artery as it enters the bone is an indication of the part of the bone which may unite poorly. Norris (1842) was unable to prove this from all of the cases available at that time. If the above mentioned studies on the circulation of bone are to be credited this is not a factor at all.

Nutter (1922) and Bloch(1919) believe that tight dressings and bandages often cause non-union by embarrasing the blood supply in cases which have escaped severe vascular injury. Robinson considers marked swelling as an important factor in cutting off the circulation.to a fracture surrounded by muscles. Eliason (1921) believes the same thing held in fractures of the tibia where the inelastic fibrous tissues force vascular compressions. He, however, was unable to verify this theory on experimental animals.

Given a fracture with a damaged blood supply there are several ways in which this may interfere with progress. In the first place the clot ordinarily found between the fragments may be scanty or absent. Bankhart (1930) and Potts (1933) believe that absence of this clot is the sole cause of non-union of intracapsular fractures of the femeur. They consider the blood clot as the important stimulus for repair and claim that dry fractures never unite. To others (Lerrich and Policard 1928 and Murray 1930) the blood clot represents the matryx into which early granulation tissue grows. In its absence this phase of repair is hindered or rendered impossible. Loss of blood supply always predisposes to fibrosis and this process may override all others in an avascular fracture.

Those workers who believe that the source of calcium salts used in repair is the blood stream (Tisdall and Harris 1922 and Peterson 1924) must pre-suppose delayed healing because of inadequate material to ossify the callus. To those who see a local calcium supply (Murray 1930) the cause is an improper matryx for its deposition and an improper Ph. for its precipitation.

Local infection of a fractured bone or of the surrounding soft tissue, either primary in nature or secondary to a compound fracture has usually been considered as a cause for non-union. Norris (1842), Chelius (1843), Erichsen (1869), Agnew (1889), Mudd (1896), Stimson (1905), Eisendrath (1907)

and Cotton (1928) all give it an important place. Some authors however (Bohler 1929 and Jones & Roberts 1934) consider infections only as a factor in delaying unions.

Tumors and cysts are almost unanimously thought to cause non-union. Eliason (1933) doubts this. He maintains that most local bone diseases causing pathological fractures do not affect repairs. He has seen sarcomata unite and quotes Bloodgood as having observed the same thing.

The final group of causes of united fractures (excepting those having to do with treatment of the new cases) is purely mechanical. The most important of these are wide separation of the fragments leaving a gap which the callus cannot fill and the interpositum of soft tissues and foreign bodies between the fragments blocking the growth of callus. All authors writing on the etiology of non-union consider these factors important. Speed (1928) and Stimson (1905) believe this to be the principal cause of non-union. Forrester-Brown (1927) and Holdeman (1932) show that periosteum caught between the fragments is as effective in blocking callus formation as muscle or tendon.

All of the foregoing possible causes for ununited fractures with the possible exception of the last group are unfortunately things with which the patient is found when first seen by the surgeon. One has little or no control over them and can only do his best to cope with them when

they appear. The remaining causes are in the hands of the surgeon. Regardless of what point of view he may choose to take on the mooted questions (and there are several) they are things he can do something about.

The first of these problems has to do with the effect of fixation on the process of union.

Thomas (1889) thought that improper fixation was the most important cause of non-union. More recently Jones & Roberts (1934) stated that it was the sole and only cause. Eisendrath (1907), Estes (1920) and Owen (1932) believed that fixation is essential to union. Henderson (1926B) stated that sixty-three percent of non-unions are traceable to inadequate immobilization. Jones & Roberts pointed out that non-unions occur in precisely the regions that are most difficult to secure firmly. Treves (1917) and Magnuson (1933) ascribe to this cause the poor results on the shaft of the humerus.

Others, however, do not consider fixation as such an important factor. Bankhart (1930) maintains that it is not a factor at all. He points out that the ribs, which are impossible to imobilize, always unite.

Animals with fractures completely untreated often get a bony union.

Mammel (1928) and Ashhurst (1922) go a step farther and claim that too complete and too long fixation is a cause of non-union. Cotton (1928) and Darrach (1933) mention this

possibility. According to Ashhurst, the formation of callus and its subsequent transformation into bone are arrested unless stimulated by slight movements of the fractured ends.

The treatment of the fresh fracture is very important. According to Campbell (1932) we have ourselves to thank for many non-unions because of enthusiasm over getting perfect reduction as shown by the x-ray. This is apt to lead to repeated manipulation which he considers destructive to the process of repair. Ashhurst (1929), Albee (1930) and Owen (1933) consider repeated manipulations the most important cause of non-unions. They show that the effect suddenly produced is the same as that which affects a poorly fixed fracture. The fresh granulation tissue is destroyed, repeated hemorrhages are produced and the endosteum is again cut off from its blood supply. By the time this damage is repaired, fibrosis has advanced far enough to choke out the callus.

It must be remembered on the other hand that a poor reduction can also interfere with healing. Speed (1928) and Swart (1930) consider improper reduction as one of the major causes of poor results. Delayed reduction according to Ashhurst (1929) and Bancroft (1929) also causes many non-unions.

Taken as a whole the foregoing review indicates several interesting trends in our conception of the etiology of ununited fractures. For a long time general conditions were

thought to be very important causes. This conception has been carried in the textbooks of surgery up to the present time. More and more material is accumulating in the literature to show that general conditions have nothing to do with the etiology of non-union. With delayed union, on the other hand, we cannot be so sure. Some constitutional factors do seem to affect the speed with which a fracture unites. It is noticable that most writers who confine their discussion to non-union alone have much less to say about the general condition of their patients than those who also include delayed union. In 1869 Erichsen stated that the causes of delayed union were general and those of non-union local. This remains a conception which seems to have some value.

It is obvious that the local factors must be the important ones in ununited fractures. Of these local factors many do not seem to be of more than slight significance. There are, however, a few almost universally emphasized. These are the compound and comminuted fractures, extensive soft tissue injury, poor or damaged blood supply, inadequate fixation of fragments and the interposition of soft tissue and foreign bodies. That a few cases may be caused by other factors is doubtless true but among those conditions always mentioned and discussed must be the real offenders.

Treatment

The most important phase in treatment of ununited fractures is preventitive. It is in proper handling of the

fresh injuries. All authorities agree that if the causitive factors are kept in mind and watched for many cases of prolonged disability can be prevented.

In the first place the fact that we no longer consider the general conditions of the patient so important as we used to as far as the healing of fractures is concerned, in no way excuses us from doing all that is possible to correct any such conditions. The changing conceptions of modern medicine should tell us what to watch for and not what to neglect. It is also important to remember that although these constitutional factors are doubted, they are by no means disproved. As stated before, Peterson(1924) considers the calcium level of the blood stream important in the prognosis of a fracture.

Kolodony (1923B) and Campbell (1924) have attached considerable importance to endocrine disturbances. For this reason some surgeons (Cotton 1928 and Darrach 1933) recommend the use of parathyroid extract, irradiated ergosterol, bone meal, milk and cod liver oil in the treatment of fractures. They think that such treatment will overcome delaying union and promote the healing of fractures. There have been some doubts cast on the value of this sort of therapy. Swart (1930) showed that the effect of irradiated ergosterol on the healing time of fractures in experimental animals was negligable. Lewis (1930) failed to find any benefit from its use in clinical cases. He not only caused no decrease

in the healing time of normal cases, but also had two cases of delayed union develop among the patients he was treating. Forty cases of delayed union were treated by this method and not one showed any response. The failure of this treatment to produce a response rather proves the theory of a local source of calcium in repair.

The actual local treatment of fresh fractures is the most important thing in prevention of both delayed union and non-union. There are several points which are of importance in the treatment of all fractures and particularly of those occuring in regions where ununited fractures are to be expected. In the first place it is now considered highly important to splint the fracture before the patient is moved at all (Owen 1932) and to reduce it as soon as possible. Formerly surgeons thought it best to suspend or to simply splint a fractured limb until the swelling had gone down and then attempt the reduction. (Chelius (1843) devotes a full paragraph to the impropriety of setting fractures early.

H. O. Thomas (1886) (the inventor of the Thomas splint) was one of the first to see the necessity of immediate splinting and early reduction. During the World War, which did serve one useful purpose, the importance of this early treatment was clearly demonstrated. Willard (1920) gives the immediate use of the Thomas splint credit for the prevention of vast numbers of non-unions following

war injuries. Figures presented from the records before and and after it went into use in army field work are quite convincing. Owens (1932) recommends that all fractures be considered as emergencies and that they always be reduced at once. He believes that if this were made routine in hospitals and clinics and if proper fixation apparatus was always at hand, the incidence of ununited fractures could be materially diminished. Bancroft (1929) pleads for reduction before any marked swelling has had time to occur. He blames many cases of non-union on the older method of suspending a fracture until swelling has subsided. Ashhurst (1929), Henderson (1918) and Shearer (1931) consider prompt reduction the most important preventative of non-unions. It is not only necessary to reduce fractures early but also to see to it that the reductions are properly done and that repeated manipulations are avoided. The damage that they may do has already been indicated. Campbell (1922) emphasized the importance of getting an accurate reduction in one attempt.

Once reduction is obtained the next question is one of fixation. Campbell (1924 - 1932), Ashhurst (1922), Shearer (1931), and Owen (1933) consider an adequate period of fixation essential. Thomas (1886) treated his patients with prolonged absolute fixation. Darrach (1933), Ashhurst (1929) and Memmel (1928) caution against fixation being too complete or too long. I doubt, from the little I have

seen of fractures if over-fixation is often a serious problem. On the other hand, too little fixation is very uncomfortable for the patient and this deserves some thought whether it has any effect on healing or not.

The early treatment of complicated, compound and comminuted fractures presents some special problems which are vital to future results.

In the first place there are those fractures which are hard to reduce and maintain in proper position. According to Owen (1932) such cases should be reduced by open operation at an early date. Murray (1931) states that all fractures with a marked displacement of fragments occurring in a region where non-union is common should have an open reduction. Forrester-Brown (1927) recommends open reductions in any case where mechanical difficulties are to be overcome. Willinsky (1922) considers immediate open reduction the method of choice in fractures of the neck of the femeur. It is the universally recommended for the patella and oleeranon if the fragments have become separated.

Shearer (1931) and Wardle (1933) are against open reduction. They believe that more ununited fractures are caused than prevented by the surgery of fresh fractures. Arvid (1933) shows that 2.4% of cases reduced by operation fail to unite, while only a tenth of that number handled conservatively have any trouble. He also states that results are worse if the operation is done early than if it

is delayed. One must remember, however, before taking such figures too seriously that most indications for open reduction are causes of non-union and the incidence must inevitably be high in such cases.

Another question, which has caused a great deal of comment in literature, is the propriety of using screws, nails, pegs, wire and plates, either in compound fractures or in open reductions. The problem of course centers around the damage which may follow the introduction of any foreign body into the fracture area. Jones (1916), Thomas (1922), Willard (1920), Albee (1930) and Magnuson (1933) are all frankly against the use of any foreign material whatever. According to them, the reaction against a foreign body is sufficient to prevent union in many cases. Hey Groves (1930) is more conservative. He cautions against the use of plates in any infected case, but uses them in his open reductions. Foster (1933) goes to the other extreme. He regularly treats his compound fractures with plates unless they are so badly comminuted that this method is mechanically impossible. He reports better results in the series of cases on which the plates have been used than those treated by other methods.

Perhaps the most difficult problem of all is the compound fracture. The treatment of this type of injury before the introduction of antiseptic and aseptic methods was a rather hopeless task. Cheluis (1843) presents a serious

discussion on the question of immediate amputation for all of these cases. It was a question in those days whether the risk of life to save a limb was justified. Up to the time Lister's paper on the results of antisepsis in compound fractures, appeared not enough of these cases escaped death or amputation to affect the incidence of non-union materially. Since then, however, ununited fractures from this source have been very frequent (as has been shown under etiology). We can again thank the war for most of what we know about these cases. Willard (1920) presents the method of treating these cases which was found to give best results in war injuries. According to him, early splinting and accurate reductions are just as important in compound as in simple fractures. For compound fractures he recommends early debridement of the wound and internal fixation for the fragments if they are difficult to reduce. This fixation is to be accomplished by the use of grafts and not by plate or wires. Hey Groves (1930) agrees that open cases should be reduced at once. He points out that most of them unite even if infection is present and unless they are properly reduced a mal-union may be established. Murray (1931) recommends rigid fixation by means of plates in open cases. This, according to him will be less conducive to non-union than ordinary means of fixation during the period when the wound must be cared for. This is in accord with Foster (1933) whose opinion on plating compound fractures has been given above. In addition to his plating, Foster uses routine debridement and Carrel-Dakin

treatment on all of his cases. He reports excellent results with this method for a large number of very severe fractures.

In regard to comminuted fractures, Campbell (1932) points out that the greatest mistake that can be made is the removal of bony fragments other than small isolated chipps. By so doing an important local calcium source is removed and at the same time a gap between the fragments is created.

If these facts are kept in mind and one is careful to be as sure as possible that no soft tissue is caught between the fragments and nothing is shutting off the blood supply, a lot should be accomplished in the prevention of ununited fractures. Shearer (1931) believes that proper treatment of fresh fractures should prevent almost all cases of delayed and non-union.

In the treatment of ununited fractures, once they have become established, the first important thing for one to decide is whether he is dealing with a delayed union or a non-union. This problem has already been discussed. The differentiation is important because the methods applicable to the one condition are not to be used for the other.

There have been a great many methods of treatment suggested for fractures in which union has not occured in the usualy time. The actual value of any of them must necessarily be rather hard to determine because most such cases will eventually united (according to Campbell, over ninety percent of them).

Obviously the treatment of delayed union is aimed at two ends. One of these is to shorten the unusually long course of repair as much as possible by trying to stimulate callus formation. The other is to prevent the delayed union from becoming a non-union (if such an event is possible).

The articles and books of fifty years or longer ago present an amazing array of procedures recommended for the treatment of the ununited fracture. Many of these procedures are now of historical interest only, but with them are included most of the methods still in use.

Norris (1842) lists twenty-three procedures which had been used to incite repair in ununited fractures. These are as follows: 1. Friction, or rubbing the fractured ends together; 2. Application of blisters over the fractured area; 3. Applications of iodine; 4. Compression; 5. Shocking with strong electric currents; 6. Salivation; 7. Local applications of caustic alkalies; 8. Use of the seton; 9. Setons near the extremity of the bones; 10. Passing of ligatures around the ligamentous masses and drawing them tight every day; 11. Introducing a wire between the fragments; 12. Acupunctations; 13. Scraping or rasping the fragments; 14. Scraping the fractured ends and retaining lint between them; 15. Hot irons; 16. Injections of such stimulating substances as port wine, salt and water or copper sulphate; 17. Resection of the fractured ends; 18. Resection of one fragment only; 19. Engaging the point of one fragment in the medullary canal of

the others; 20. Rubbing the ends of the bones with caustics; 21. Actual cautery; 22. Wiring; 23. Amputation. Since Agnew (1886) mentions exactly the same procedure humanity has evidently been spared any new developments of the kind listed above. Norris only considered five of his methods of any great importance. These are friction, compression, the seton, caustics, and resection of the ends of the bone. Three of these procedures find a place in the literature of today.

The methods now in use are massage, diathermy, application of heat, local injection of calcium salts, local injections of blood, the induction of passive hyperemia, periarterial sympathectomy, percussion over the fragments, weight bearing and exercise, prolonged fixation, friction, drilling of the fragments and operation.

Massage is mentioned by Cotton (1928) who rather questions its possible value. This procedure, according to him, is useful only in the restoration of muscle tone and the preservation of joint function. Willard (1920) has recommended the use of massage in old infected cases as a means of determing whether or not there is a chance for recurrance. Most authorities are against massage. They think its use is a cause rather than a therapeutic agent in delayed union.

Cotton (1928), Bankhart (1930) and Shearer (1931) mention the use of diathermy. Their remarks are confined to casual observations to the effect that it may be of value. They evidently do not use it much themselves and

are not enthusiastic in their recommendations of it.

Hot applications are also mentioned by Cotton (1928) and Speed (1928). Speed thinks they have some value.

Darrach (1933), Cotton (1928), and Albee (1920) speak of the local administration of calcium salts as an important therapeutic procedure. It will be remembered that Lerrich and Policard (1928) and Murray (1930 & 1931) consider the source of calcium used in bone repair to be a local one. If this be true such administration of calcium salts has obvious value. Murray (1930) has shown that rapid repair of experimental fractures can be induced by a rich local supply of calcium. (Albee (1920) had already shown the same thing ten years before.) Key (1934) has just published experiments which show no effect at all from local calcium. This type of treatment appears to need a little more trial before it can be definitely accepted.

Eisendrath (1907) speaks of the use of local injections of blood. He credits this method of treatment to Bier, who used it with the object of replacing or building up the clot between the fragments. The fate of this treatment I do not know. No one speaks of it in the more recent literature. To Bier is also credited the use of passive hyperemia of the fractured area, (Pearse and Morton 1930). Thomas (1886) had used venous stasis some time before. Bier developed this type of therapy on the assumption that it was reproducing a normal reaction usually found in reparative processes. Pearse and Morton (1930) have used venous stasis on a series

of cases showing delayed union and they report excellent results. They attribute its value to the induction of a local Ph. favorable to the chemical process of bone formation. They also believe that venous stasis raises the local concentration of phosphates, carbonates, iron salts and calcium salts. Murray (1930) believes that damming is of value only in setting up the proper Ph for ossification (one of his pre-requisite factors). He states that occasionally this method works where others have failed. Eisendrath (1907) and Jones (1916) have also recommended the use of damming. There can be no doubt but that their method is worth some consideration. More uniformly good results are reported from its use than from most of the others.

In a recent paper Colp, Kassabach and Mage (1933) report very encouraging results from the treatment of experimental fractions by periarterial sympathectomy. The rationalle of this procedure lies in the increase in the local blood supply from vaso-motor paralysis. Whether this method will ever be of value in clinical work or not is hardly safe to say. It may prove to be of service in those cases in which vaso-motor disturbances are a troublesome factor.

Percussion over the site of the fracture is of value according to Jones (1916). It sets up just enough local reaction to stimulate repair and at the same time does not disturb the fragments. Percussion, however, does not enjoy widespread use. Many writers mention it but few consider it worth any discussion.

The remaining conservative or semi-conservative methods of treating ununited fractures have to do with direct initation of the fracture site in order to stimulate repair, and the freshening of surfaces which may have become more or less fibratic. The oldest method known for treating ununited fractures is rubbing together the adjacent ends of the fragments. This method was spoken of by Celsus about 28 A.D. and seems to have been in general use by the surgeons of his time. John Hunter recommended it and evidently used it repeatedly. His method was to repeat the process daily, each time rubbing the bones until it became painful. Cheluis (1843), Norris (1842), Erichsen (1869), Agnew (1889), Mudd (1896) and Eisendrath (1907) consider friction a valuable method of treatment. During the last twenty-five years friction by this method has lost its standing. We do not hear any more. This probably is because other methods of producing the same local results have been found more satisfactory. These methods are drilling of the fragments, and a certain amount of use of the fractured limb (particularly weight bearing in the lower extremity).

Drilling in ununited fractures is recommended by Campbell (1932), Speed (1928), Bozan (1932) and Eisendrath (1907). This process not only gives an added wound stimulus to the fractured bones, but also reproduces the hemorrhage and dead tissue zones found in a fresh fracture. Bozan considers it valuable in overcoming the anemia of the proximal fragment in fractures of the neck of the femeur.

Abbott (1922), Speed (1928), Ashhurst (1929), Owen (1932) and Darrach (1933) all advise early use of the fractured limb as a preventitive of delayed union and as treatment for ununited fractures.

Weight bearing is the method recommended for the lower extremity. This must, of course, be done under fixation sufficient to protect the fracture from any excessive strain or motion. Others, however, fear any method other than prolonged rest and do not consider weight bearing safe until complete consolidation has taken place.

In taking up the various operative procedures used in ununited fractures, one must first consider their indications. Campbell (1932 and 1923), Speed (1928), Cotton (1928), Darrach (1933) and others consider operation to be the treatment when and not before non-union is established. Delayed unions are to be treated conservatively and non-unions by surgery. This is the general rule. This brings us back to the question of when a case shall be called non-union. Many of them are obvious, of course. The true pseudarthrosis and the case in which all reparative processes have become stationary admit of no question. The doubtful case is the one with a picture of delayed union still persistant after a period of from six to nine months. It has been shown that some of these cases will unite after a longer period than this, but the question is how long a patient shall be allowed to stay disabled under conservative treatment waiting for a union that may occur. No doubt the economic status of the patient, his occupation,

and the available facilities for safe bone surgery are to be the deciding factors.

The question of surgery in any case is the question of treatment of non-union. In general the diagnosis of the one and the indications for the other are the same. The surgical procedures used in the past for non-union are the same as those given a few pages back for delayed union. There is no need to review these methods again. Surgical procedure for non-union is now definitely narrowed down to one general method, namely, resection of fibrotic tissue and eburnated bone with a re-opening of the medullary cavity and the introduction of some type of bone graft. These grafts according to almost all writers should be autogenous. This does away to a large extent with any foreign body reaction. Living grafts are for the same reason to be preferred.

What a bone graft does when introduced into a fracture area is a matter of debate. According to Lerrich and Policard (1928) and Murray (1930) it simply supplies a local source of calcium which is utilized in ossification of the locally formed pre-callus. According to Murray, bone meal or triple calcium phosphate serve just as well as a living graft.

According to MacEwen (1912), Taylor (1915), and Thomas (1923) on the other hand, bone grafts supply new actively functioning osteogenetic elements. MacEwen maintains that these elements spread throughout the old system and are responsible for any subsequent repair.

There have been several types of bone grafts recommended in the treatment of ununited fractures. Among them may be mentioned the osteoperiosteol transplant, the onlay graft, the inlay graft, the medullary graft, the chip graft, and the local sliding graft.

Osteoperiosteol grafts are recommended by Taylor (1915) Albee,(1915 and 1920), Thomas (1923) and by Phemister (1931) for some cases. These grafts according to Taylor possess the advantage of being rich in bone forming elements and at the same time occupying comparatively little space. They are best used, according to Phemister, bridging across the fracture line and held in place by suture of the soft parts. MacEwan (1912) sees no reason for including periosteum in bone grafts. He maintains that it has no effect on the efficiency or viability of the transplant.

Small chip grafts are recommended by Cotton (1918). These grafts can be used in considerable numbers filling in any bony defect and have the advantage of a large surface area (MacEwan claims this is important). Their disadvantage lies in the fact that they are of little aid in fixing the fragments. Willard (1920) used small wafer grafts for fractures of the forearm. He did not have very good results with large transplants in this region.

Campbell (1932) and Albee (1915) consider the inlay graft to be the most efficient form. This graft has considerable contact with cut surfaces of the old bone and can

give some support to the fracture. Phemister recommends a wide inlay graft turned on edge and made to partially fill the medulary cavity. Owens (1933) maintains that onlay grafts are even more efficient than the inlay grafts because they add strength by increasing the circumference of the bone.

Phemister and Compere (1933) have had good results with massive full thickness grafts. These are not quite so efficient in supplying osteogenic elements, but give greater supporting strength to the system than any of the other forms. In factures without any great displacement Phemister also has had good results with local sliding grafts.

The medulary graft has no place in the treatment of fractures. Its only value is fixation of the fragments which can be better done by some other means. Campbell (1932), Phemister (1931) and Albee (1915) mention this method only to condemn it.

I have not been able to find any satisfactory report on the results to be expected in the operative treatment of non-union. Most authorities are of the opinion that the prognosis is not very good. At best these conditions are long in their convalescence and uncertain in their outcome and a much happier state of affairs will be reached when more are prevented and fewer treated.

Conclusions

1. Ununited fractures are more common today than they were fifty years ago. A higher incidence of violent injuries is probably responsible.
2. Non-union and delayed union are probably not varying degrees of the same process. Non-union is non-union from the onset. Delayed union of very long standing may simulate non-union in that all healing processes have stopped. Such cases may; however, spontaneously unite.
3. The causes of delayed union are both general and local. The causes of non-union are purely local.
4. True non-union is a rare condition. Delayed union is quite common.
5. Most ununited fractures can be prevented by proper treatment of fresh fractures.
6. The treatment of delayed union is conservative, that of non-union is operative.
7. The proper operation for non-union is resection of all pathological bone and tissue from the ends of the fragments and the insertion of some form of autogenous, living bone graft.

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