REPORT AND COMMENTARY UPON A SERIES OF CASES OF UNUNITED FRACTURE.

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INTRODUCTION.

In the course of his work in the Royal Infirmary of Edinburgh, the writer observed that ununited fractures of the tibia and fibula were relatively common. The patients were all healthy men in early adult life, and from the economic aspect alone it appeared to be a subject well worth closer investigation.

A series of six cases of ununited fracture of the tibia and fibula is reported: each case history is followed by a short discussion of its particular features: the Commentary is a general review of the subject of ununited fracture.

All the cases have been reported with the kind permission of Professor John Fraser.
CASE I.

Stanley Burfoot, 24 years of age, clerk, was admitted on the 15th of August 1935. At the end of a fortnight's climbing holiday in Skye, the patient met with an accident at 4.30 p.m. on the day before admission. He was on a steep hill and his left leg was pinned to the ground by a boulder which came rolling down the hill. He was carried for some miles to the local cottage hospital; here the wounds were cleaned and packed with flavine gauze; the leg was put in a box splint. He was kept overnight, and as the local practitioners did not think they were fitted to treat the fracture, the patient was sent to Edinburgh by car; he arrived at 10:15 p.m. after a journey lasting more than 12 hours.

The patient's general condition was remarkably good. He showed no sign of shock, was wide awake and cheerful. His leg was lying comfortably in the box splint. Temperature 99.0°; pulse 96 per min. There was a large open longitudinal wound 8 inches long by 3 inches broad on the anterior aspect of the left leg; through this wound projected ¾ inch of a bare spike of the proximal fragment of the tibia. The wound had been packed with flavine gauze, which was left in position. Fragments of torn muscle could be seen round the edges of the wound. There was a second wound over the/
the posterior aspect of the knee: it ran from \( \frac{1}{2} \) inch behind the head of the fibula towards the medial aspect of the head of the tibia; the skin edges had retracted, leaving a wound about 4 inches by 4 inches. The common peroneal nerve could not be seen, but there was no evidence of its having been severed.

Operation was carried out by Mr Mercer on 15-16th August. The skin round the wound on the anterior aspect of the leg was cleaned and shaved; the ragged edges of the wound were excised. The packing was removed, exposing a deep cavity. The cavity was swabbed out with spirit, then with Stiles' paste. Some unhealthy-looking muscle was removed. An attempt was made to plate the fragments as they were very unstable, but this was given up after a fragment of screw had been broken and left in the bone. When traction was exerted, the fragments could be made to dovetail into each other satisfactorily. The wound was closed as far as possible: there was some skin missing, and an area the size of half-a-crown was left uncovered and packed with flavine and paraffin gauze.

The wound on the posterior aspect of the knee was then dealt with. The skin edges were excised and the wound swabbed out with Stiles' paste; the knee had to be flexed to bring the skin edges close to each other: the wound was closed with silk-worm gut sutures, and a dry dressing applied.

A/
A plentiful dressing of sterile cotton wool was applied over the wounds, and the leg was put in plaster from the toes to 8 inches above the knee. The foot was dorsiflexed to almost a right angle; the medial side of the head of the first metatarsal bone was in line with the medial border of the patella; extension was applied to the leg; the knee was kept flexed at an angle of about 135 degrees; the limb was held in this position until the plaster hardened. It was found that the leg lay comfortably on a Bohler's extension splint.

While he was in hospital after operation there were no signs of local infection. The patient was happy and free from pain; his appetite was excellent. Radiological examination showed that the fragments were in good position. He was discharged on 3rd September in plaster, to the Astley Ainslie Institution. The smell became so offensive that on the 17th of October - two months after injury - the plaster was bivalved, and fresh dressings were applied to the wound on the anterior surface of the leg. In a fortnight the wound had healed, and a new plaster was applied; he was able to get about easily in this plaster, but he grew too rapidly, and it had to be removed. The wound broke down and he was readmitted on 8th January 1936 with the wound still open and discharging a little sero-pus. There was still fairly free/
free movement between the fragments. X-ray examination demonstrated a small sequestrum at the site of fracture, considerable decalcification of the fragments, and little union.

Mr Mercer operated again on the 11th of January. An incision was made enlarging the existing wound. The sequestrum was removed and some necrotic-looking parts of the bone-ends were chiselled away. The fragment of metal screw, now quite loose, was removed too. The wound was closed with silk-worm gut sutures. The limb was put in plaster. He was discharged on 4th February, to the Astley Ainslie Institution. Here he made but slow progress, and as there was still little evidence of callus formation, he was readmitted on 8th May.

The general health was good; he was not toxic.

The wound was still open and discharging a little sero-pus. The fragments had not united; there was free movement at the site of fracture. X-ray examination showed no evidence of union of the tibial fragments, although there was a fair amount of callus around the fibular fracture. There was no sclerosis of the bone ends. It was decided that a sequestrectomy should be performed, so on the 11th May the wound edges were excised and a probe passed into the depths; a small sequestrum was identified; this was removed.
DISCUSSION.

This is a case of delayed union in a compound fracture. To this delay there have been many contributory factors.

A bony sequestrum between the fragments obviously forms a mechanical barrier to the reparative granulation tissue which is trying to bridge that part of the gap occupied by the sequestrum. The sequestra therefore played a part in the production of delay in this case.

The fracture was compound, so there was probably a mild infection of the wound, as shown by the inability of the skin to heal; the infection produces a mild inflammatory reaction with hyperaemia. There was a fragment of foreign metal in the bone: this exerts an irritant action, also producing hyperaemia. This hyperaemia leads to decalcification of the bones, so the local excess of calcium, essential for the formation of bone, is reduced, and there is delay in bony union.

The hyperaemia may be relative, producing the decalcification of disuse. Watson Jones explains this phenomenon by saying that the arterial blood-supply to the leg in plaster is as generous as it was to the leg at rest before fracture. The muscles are now absolutely immobilised and atonic, so do not use the blood/
blood, which is therefore diverted into the bone, producing there a hyperaemia and decalcification. This suggests a remarkably poor provision on the part of Nature for the regulation of the blood-supply to the various tissues, and is surely not a complete explanation.

The fracture was in a plaster shell for 2 months. The dressing of cotton wool over the lower part of the leg was voluminous, and it is more than likely that there was some degree of movement inside the plaster. This constant movement produces recurring trauma to the granulation tissue with recurring reactionary hyperaemia, again leading to decalcification of the bones.

This case has not gone so far as the stage of non-union. In non-union the essential feature is a dense plaque of sclerotic bone at the end of the fragments, obstructing any effort at union: there is no such plaque here. It is a case of delayed union, the delay having been produced by decalcification of the bones which has arisen from hyperaemia, which is the result of the unhappy coincidence of at least four contributing factors.

The prognosis is good: a prolonged period of immobilisation - 6 months or more - will probably lead to union. This case now corresponds closely to Case II/
II which, after 8 months of delayed union and new plasters, was cured by more or less accidental immobilisation for a year.
CASE II.

Thomas Dunn aged 20, a farm worker, was admitted on the 7th of May 1933, with a history of having been involved in a motor-cycle accident on the day before admission. He was taken to the local Cottage Hospital where he was given an anaesthetic and it was found that he had a compound fracture of the right tibia and fibula with considerable displacement. The fracture was reduced and put in a box-splint. Some twelve hours after injury there was considerable haemorrhage from the wound, and it was considered advisable to send him in to Edinburgh, where he arrived 30 hours after the accident.

The patient was a strong healthy young man. His temperature and pulse-rate were normal. There was a clean superficial abrasion just under the chin. The right leg was in a posterior splint with side-pieces. The dressings were soaked with blood: on removing these, a wound 1\(\frac{1}{2}\) inches long and \(\frac{3}{2}\) inch broad through the skin over the medial aspect of the middle of the tibia was exposed: from this there was a steady ooze of blood. There was no gross displacement of the fragments. There was no other injury.

On admission he was given 2000 units of tetanus antitoxin intramuscularly.

The wound in the leg was cleaned with saline and hydrogen/
Case I

9 May 1933

16 May 1933

5 June 1933
hydrogen peroxide: the bleeding points were sought for and the vessels ligated: the wound was swabbed out with flavine solution and closed: a dry dressing was applied. A plaster was then applied from mid-thigh to the toes.

The wound healed well. X-ray examination showed that the fragments were displaced and overriding, so on the 15th of May a Kirschner wire was put through the lower end of the tibia, and the leg was fixed in a Bohler splint with 20 lb. extension upon it: this produced considerable hyperextension, and the weight was gradually reduced until the fragments impinged upon each other: forward bowing of the fragments was reduced by moving the wire to the calcaneus on 24th of May. With the extension still in action a plaster case was applied on the 18th of June. X-ray examination showed that the bones were in good position. He was sent to the Convalescent House for a short while and discharged on 23rd of June.

The plaster was bivalved on 2nd August: there was no clinical or radiological evidence of union. On 14th September, the position of the fragments was good, and there was slight evidence of union: the limb was put in plaster again.

On 14th January 1934 this plaster was bivalved. Union was still poor, so his name was put on the waiting list for admission and operative treatment. He did not/
not report again until the 9th of January 1935, having apparently waited for a twelvemonth for the promised postcard. He was still in the bivalved plaster case.

He was readmitted to the Ward on the 12th of January, and it was found that there was much wasting of the muscles of the right leg and thigh: the joint movements were not quite normal, but were wonderfully good after a year's complete immobilisation. There was only slight springy movement at the site of fracture. X-ray examination showed that during the 12 months' immobilisation a good amount of callus had been formed, and that the fragments had united almost completely.

The condition of delayed union having been cured, the patient was fitted with a short walking caliper and discharged on the 17th of January.

He reported on the 19th of June. Union was progressing and movements of the joints were improving. On the 5th of July the fracture was completely healed: he was given an 'Elastoplast' bandage and the caliper was removed.
DISCUSSION.

This is another case of delayed union in a compound fracture. The case history resembles quite closely that of Case I. Both were severe compound fractures packed in outlying hospitals, then sent to the Royal Infirmary some 24 hours after injury and treated there.

As in Case I, there was probably a low-grade infection of the wound, producing hyperaemia and a tendency to delay in union. The fracture was not immobilised until some 5 weeks after injury; it was in extension in a Bohler's splint for that time. The extension was varied in intensity and in direction from time to time. The ends of the fragments therefore moved upon each other, producing hyperaemia, decalcification and delay in union.

The decalcification of disuse, whether Watson Jones' explanation is accurate or not, is well illustrated by comparison of the X-ray plate dated 17th January 1934 and that dated 9th January 1935. The whole length of the bone, and particularly the ends of the fragments concerned in healing of the fracture, are decalcified.

This case in January 1934 is comparable to Case I in May 1936. Both were compound fractures of the tibia in healthy young men: both were of some 8 months' duration.
duration and both were ununited, one of the main causes of the delay being hyperaemic decalcification of the bones. This case united satisfactorily in the course of a year's immobilisation after January 1934, so there does not seem to be any reason why Case I should not unite if it is immobilised for long enough.

For some years the patient had been a known diabetic he used insulin, but kept to a diet which was supervised and adjusted from time to time by his doctors. It contained a generous quantity of fats, which the patient liked.

He was a keen ruddy man, obviously in considerable pain, but not grossly shocked. There was no glycosuria. In the lower third of the left leg there was marked swelling and bruising; there was a slight angular deformity of the leg at this point, the lower fragment being inclined forwards at an angle of about 7 degrees to the upper fragment. The lower fragment was everted and did not meet accurately with it. There was marked swelling round the ankle region. Crepitation could be easily elicited on the tibia. The skin was not broken.

There/
CASE III.

William Graham, aged 52, a fish-hawker, was admitted on the 20th of March 1934. He had been driving a horse and cart when the reins became entangled with the horse, which kicked, pinning the patient's left leg against the front of the cart. It was extremely painful and he realised from its uselessness that it was broken; he drove on until he came to a policeman, who summoned an ambulance and sent the patient to hospital.

For some years the patient had been a known diabetic; he needed no insulin, but kept to a diet which was supervised and adjusted from time to time by his doctor; it contained a generous quantity of fats, which the patient liked.

He was a thin ruddy man, obviously in considerable pain, but not grossly shocked. There was no glycosuria. In the lower third of the left leg there was marked swelling and bruising; there was a slight angular deformity of the leg at this point, the lower fragment being inclined forwards at an angle of about 7 degrees on the upper fragment. The lower fragment was everted and did not move accurately with it. There was marked swelling round the ankle region. Crepitus could be easily elicited on the tibia. The skin was not broken.

There/
Case III

15 March 1934

24 March 1934
There was a large bursa on the medial side of the left big toe. Radiographical examination showed a fracture of the tibia and fibula in the lower third with displacement.

Treatment was carried out by Mr Bruce on the 20th of March: traction was exerted upon the leg; plaster was applied in two sections, one below the fracture and one above it: the fragments were then manipulated into position and the plaster completed. X-ray showed the fragments to be in unsatisfactory position so on 27th March, the plaster having been removed, Mr Mercer corrected the position of the fragments under the X-ray screen. When the position was satisfactory, manual extension was maintained, and a new plaster shell was applied. He was discharged on 29th March with the fragments in good position and the plaster fitting comfortably.

He reported on the 18th of May: the plaster was bivalved. The fracture was fairly firmly united, so he was started off on a course of active and passive movements of the legs, particular attention being paid to the restoration of mobility to the left knee joint and ankle joint. With the object of promoting union, a course of "hamming and damming" was instituted. "Damming" is the production of venous stasis: it is done by the application of a Martin's bandage to the thigh just tightly enough to produce venous engorgement: the/
the bandage is kept on for about twenty minutes at a time. "Hamming" is stimulation of the bone ends by striking the heel - covered with a felt pad - sharply with a hammer or mallet for perhaps five periods of a minute each during the twenty minutes' duration of the treatment.

This treatment was persevered with until the 6th of January 1936, when he reported at the Ward. He was complaining of pain in the left ankle and mobility at the site of fracture. There was a certain amount of painless movement at the fracture site. X-ray examination showed dense sclerosis of the bone ends; there had been a considerable amount of callus formed, but there was no evidence of bony union. There was evidently a fibrous union present. There were osteoarthritic changes in the ankle joint. His name was put on the waiting-list for a bone-graft, but he was impatient to have it attended to, so he was admitted to the Western General Hospital, where a cortical sliding graft operation was performed in March 1936.
DISCUSSION.

This case, initially one of delayed union, progressed to the more serious state of non-union, in spite of having received the accepted active treatment to promote healing.

The cause of the delay and of the non-union was ischaemia, this being the result of arteriosclerosis. The blood-supply of the area round the junction of the middle and lower thirds of the shaft of the tibia is almost wholly dependent upon the nutrient artery as will be explained in some detail later: so in a fracture in this area, the lower fragment especially has a poor blood supply. If there is any obstruction in the lumen of the artery, the ischaemia will be still more severe. Calcified vessels can be seen in the X-ray prints of the leg. This man was a diabetic, and the arteries of diabetic subjects are particularly liable to undergo degenerative changes: one of the most widely held explanations of this is that it is the high-fat diet so often employed which has the deleterious effect on the vessel walls, and not the disease itself: this man had a mildly restricted diet containing as much fat as he wanted, which was a considerable quantity. The effect of ischaemia is to slow down the production of granulation tissue and to impair its nutrition: osteoblasts are poorly nourished, so enough calcium is not deposited/
deposited to bridge the gap before the stage of con-
traction of fibrous tissue sets in. The bone ends
later become sclerotic, sclerosis being the result of
ischaemia, and are united by a mass of dense avascular
fibrous tissue. This is the only case of non-union
following a simple fracture of the tibia and fibula
which the writer has seen in Wards 7 and 8 during the
two years from March 1934 until March 1936, during
which time he knew something of most of the patients
in these Wards.

The therapeutic production of venous stasis was
introduced empirically many years ago by Hugh Owen
Thomas.

It has recently been shown both experimentally
and clinically by Pearse and Morton that fractures
in limbs with venous congestion heal more rapidly than
do controls. The hammering produces a frictional
stimulus to bone ends, an increase in vascularity and
in cellular activities, so tending to promote union.
These efforts to awake activity having failed, the
bone ends became sclerosed., and an end-result was
thus arrived at, the position being one of non-union.
The essential parts of the treatment of this con-
dition are the removal of the whole or part of the
sclerotic plaques at the bone ends and the fixation
of the fragments.
CASE IV.

Andrew Hamilton, aged 30, a farm labourer was admitted on the 16th of May 1935, having collided with a motor-car while riding a pedal cycle; he was thrown off the cycle onto his right side. The left leg apparently did not touch the ground; he did not know whether it was crushed by the car or not.

The patient was a stocky healthy equable man. He was fully conscious; he was not shocked. There was no evidence of intracranial damage. Over the right eyebrow there was a deep wound 2\frac{1}{2} inches long with clean-cut edges: there had been some venous oozing from this. There was no other head injury.

The left foot was lying externally rotated upon the upper part of the leg. There was a painful discoloured swelling in the lower third of the leg. Over the medial border of the lower third of the tibia there was a round wound with a diameter about that of a match, from which blood was oozing. Just above this there was another even smaller perforation of the skin.

The patient could not lift the foot off the bed. There was abnormal mobility in all directions at the junction of the middle and lower thirds of the tibia. Crepitus could be elicited.

Treatment/
Case IV

12 May 1935

30 May 1935

13 June 1935
Treatment was carried out by Mr Millar on the day of admission: the site of fracture was completely exposed by an anterior incision 6 inches long; the muscles overlying the tibia were divided. The great saphenous vein was found to have been transfixed by a spicule of bone: the vein was ligated. It was found that there was marked comminution of the fragments, there being two completely free pieces of bone and two which retained only their periosteal attachment. Good alignment was assured by traction on the foot: the loose fragments were replaced between the bone ends. The muscles were united with catgut. The skin wounds were closed, a dry dressing put on, and plaster applied from the toes to above the knee. An anterior gutter was made in the plaster.

No signs of local or general infection developed. X-ray examination showed that the fragments were in good position. He was discharged on the 23rd of May, with the plaster fitting comfortably.

He reported on the 30th of May, in plaster: the lower fragment had been displaced a little medially: there was no evidence of callus formation.

On the 13th of June the plaster was bivalved. There was no clinical evidence of union. X-ray examination showed that there was still medial displacement of the lower fragment, and that there was no callus. A new plaster was applied, and the displacement of the lower fragment corrected.
Case IV

24 July 1935

21 August 1935

16 October 1935
He was seen on the 24th of July: he was comfortable; there was no evidence of union, but the fragments were in good position.  

On the 13th of August, the plaster was taken off. There was still considerable movement between the fragments: a new plaster from the toes to mid-thigh was applied and he was instructed to get about as much as possible on his crutches. He reported again eight days later having fallen the day after the new plaster was fitted: X-ray examination showed that there was angulation medially and forwards of the tibia, decalcification of the bone ends, and the doubtful early signs of sequestrum formation, there being a dense piece of bone intervening between the fragments of the tibia, clearly defined from the rest of the bone: there was no union.  

The plaster was bivalved on the 16th of October. There was no clinical evidence of bony union, the fragments still being freely mobile. X-ray examination with the plaster off showed inward angulation of the tibia, no further separation of the sequestrum, and no callus formation. The bivalved plaster shell was put on again. He was sent for a course of "hamming and damming" which was carried out as in Case III. In spite of this he made little progress, so he was readmitted on the 13th of December.  

He/
He complained that the fracture had not properly united, there being movement of such a degree that he could not bear weight upon the left leg. He was free from pain.

At the junction of the middle and lower thirds of the tibia there was a swelling indicating the site of the original fracture. At this point the leg was bent, the lower fragment being directed medially from the upper. The swelling was not painful; crepitus could be elicited. There was a pseudarthrosis present: the lower fragment could be moved on the upper to an angle of approximately 20 degrees medially, anteriorly and posteriorly, and to about 10 degrees laterally. There was no shortening. X-ray examination showed decalcification of the bone ends: there was no bony union: there was no increased density or further separation of the suspected sequestrum.

On the 16th of December Mr Mercer inserted a bone-graft. A longitudinal incision was made over the medial surface of the tibia, extending for 3 inches above and 3 inches below the site of fracture. The periosteum was reflected with difficulty from the surface of the bone, being adherent to the bone and surrounding dense fibrous tissue. The fibrous tissue between the bone-ends was cut away, the sequestrum being within this mass, and two strips of the full thickness/
Case X

17 December 1935

11 March 1936
thickness of the cortex were cut with an electric circular saw of the Albee single-bladed type.

A strip 2 inches long and $\frac{1}{4}$ inch broad was cut from the distal fragment and preserved in a swab moistened with saline. A strip 4 inches long and $\frac{3}{4}$ inch broad was cut from the proximal fragment and slid down into the trench in the distal fragment, thus filling it completely, bridging the gap, and filling part of its original position in the proximal fragment. It was lashed into position with Kangaroo-tendon. The blade of the saw was kept cool and moist by a continuous irrigation of the operation field with cold normal saline. The strip from the distal fragment was then laid into the cavity left in the proximal fragment. The wound was closed with silk-worm gut sutures and the limb immobilised in a plaster case from mid-thigh to the toes. He was discharged on the 20th of December, the leg feeling very comfortable. X-ray examination showed that the fragments were in good alignment.

On the 22nd of January 1936 he reported with the leg in plaster: X-ray examination showed no change since December. On the 11th of March, the plaster was removed. Healing was found to be incomplete. X-ray examination showed that union was slowly progressing. The fragments were in satisfactory position, although there was slight outward angling of the tibia. A new plaster was applied. This plaster was removed on/
on the 22nd of April. The fragments were found to be firmly united. A short walking caliper was ordered, and the patient was pleased to be able to walk again with a boot on his left foot. He was seen again on the 26th of May, when the bones were firmly united. He was able to walk 1½ miles with the help of two sticks. The caliper was retained.
DISCUSSION.

This is a case of delayed union following a compound comminuted fracture.

As in the previous compound cases, low-grade infection probably played a part in producing hyperaemia and delay in union. The main factor causing delay, however, was the presence of a sequestrum.

At the original exploration of the fracture, two fragments of bone completely free from even periosteal blood-supply were exposed, inspected, and returned to their original position between the ends of the fractured tibia. The series of X-ray plates shows a progressive increase in density and gradual separation of a piece of bone, probably one of these replaced fragments. Sclerosis of bone indicates ischaemia; this fragment therefore had an insufficient blood-supply for its needs, and it became a sequestrum. The area of bone around the fracture was decalcified: this indicates hyperaemia, probably produced by infection.

The fragments were not completely immobilised during the first fortnight, as was shown by the medial displacement of the lower fragment which developed between the 17th and the 30th of May, while the limb was in plaster: this was another factor in the production of hyperaemia and delay.

It is unfortunate that in this case and in Case III "hamming and damming" should have failed in its object/
object of promoting union. This form of treatment is widely recommended by recognised authorities, and it has become part of the routine treatment in the Royal Infirmary for delayed union.

This fracture did not go to the stage of non-union, so it would probably have healed with the simple removal of the sequestrum and immobilisation in plaster; there were no sclerotic plaques of bone to be removed. The use of a sliding graft, however, made quite sure of the immobilisation of the fragments. If the loose fragments of comminuted bone had been removed at the time of original treatment of the fracture, the delay might have been avoided altogether.

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The left tibia had been fractured 3 years before and had united well.

The patient was a strong healthy man.

There was an irregular swelling visible about the middle of the left tibia: the skin over it was of a deep brown colour, and of the consistence of parchment. On palpation the swelling was hard but not tender, and arising from the bone.
CASE V.

Alexander Macleod aged 32, a police constable was admitted on the 15th of February 1934 with an un-united fracture of left tibia. He gave the history that on the 12th of December 1932 he was riding a motor cycle in the wilds of Sutherland when he was thrown off it and struck his left leg against a projecting iron standard by the roadside. His leg, he said, was doubled up under him. He crawled through the snow for some two hours towards the nearest house; he was picked up and taken to the Lawson Memorial Hospital where he was treated for about a year for a compound fracture of the tibia. The leg was put in a wooden splint until the wound had healed, which was in ten days, then it was immobilised in plaster: the plaster was changed more than once, and the fracture failed to unite, so he was sent to Edinburgh.

The left fibula had been fractured 5 years before and had united well.

The patient was a strong healthy man.

There was an irregular swelling visible about the middle of the left tibia; the skin over it was of a deep brown colour, and of the consistency of parchment. On palpation the swelling was hard but not tender, and arising from the bone. There was painful/
painful mobility of the fragments at this point, but no crepitus was elicited. Both malleoli were large and surrounded by a mass of callus. Movement of the ankle was full. Movement of the knee-joint was full. There was considerable muscular wasting, but he could bend his tibia by contracting his calf muscles, an achievement which afforded him considerable joy.

There was 1½ inch of shortening of the leg. X-ray examination showed free formation of callus, sclerosis of the bone ends and no bony union.

Operation was carried out on the 17th of February by Mr Mercer. A six-inch incision was made over the medial surface of the tibia. The fragments were exposed: they were surrounded by exuberant callus, the exposed part of which was chiselled away: a small loose fragment of tibia was removed. Strips were cut from the cortex of the tibia and a sliding graft inserted as in the last case. The wound was closed and plaster applied from the mid-tarsal region to mid-thigh.

He was sent home on the 1st of March with the plaster fitting well. He was instructed to use his crutches freely and to return in 2 months.

He reported on the 9th of May. X-ray examination was carried out in plaster: the plates showed that union was progressing satisfactorily: there was considerable disuse decalcification of the tibia.
Case V

30 October 1934
On the 17th of July 1934, the plaster was bi-valved: there was a slight degree of springy movement between the fragments: X-ray examination showed that there was a complete bridge of dense callus. He was sent home in a short walking caliper, to get massage and active movements of the leg. By the 3rd of October the fragments had united firmly, and he was given a certificate to return to full duty.

A letter was written early in 1936 to find out what progress he had made, and whether the leg ever caused him any disability or discomfort, but no reply was received.
DISCUSSION.

This is a definite case of non-union following on a compound fracture.

There is a great likelihood that the wound was infected, as the patient crawled about with an open wound for two hours before his leg was even put to rest; the infection would delay union as in the other cases. There was a bony sequestrum removed at operation; this doubtless delayed union too. The fractured bone was not effectively immobilised until ten days after injury. After that the plaster was changed on at least two occasions. There was thus absence of that complete prolonged immobilisation which is essential for the rapid healing of fractures, especially compound ones. The presence of a large amount of callus around the bone ends but not between them suggests that the tissues were well able to synthesise the materials to build a single-span bridge between the fragments, but that the land from which the two ends of the arch were projecting was of such an unstable nature that the bridge was never completed.

This patient now has complete restoration of the function of the leg: he limps a little, but is quite able to carry out his duties as a police officer in the district of Tongue, which is as good a test of cure as can readily be imagined.
CASE VI.

Victor de Spiganovicz, aged 55, a journalist, was admitted on the 21st of August 1934, the story being that whilst riding a solo motor-cycle on the day of admission a lorry had run into him from the right side: he fell over towards the left side, with the cycle on top of him, and his left leg was pinned between the cycle and the road.

The patient was a healthy man who looked less than his 55 years: he was considerably shocked, but quite conscious: there was no history of unconsciousness.

The left foot was inverted, and completely out of the control of the rest of the limb. There was a cut 3½ inches long right through the skin on the anterior aspect of the lower third of the tibia and another cut 1½ inches long down to the bone just above the medial malleolus. There was a skin wound 2 inches long under the chin about the level of the left mental foramen. The wounds were not grossly contaminated with road dirt.

Shortly after admission he was given intravenously 20 c.c. of concentrated anti-gas-gangrene serum, and 2000 units of tetanus antitoxin intramuscularly.

Soon after admission, Mr Bruce treated the fracture/
Case VI

22 August 1934
fracture. Under general anaesthesia the skin was prepared and the wound edges cut clean; the bed of the wound was swabbed out with hydrogen peroxide solution, then with spirit. It was found that the fracture was a comminuted one, there being a fragment of tibia about 4 inches in length between the large proximal and the small distal fragments. The skin edges were approximated with silk-worm gut sutures and the limb encased in plaster from mid-thigh to the bases of the toes.

The patient had retention of urine for 48 hours after operation and had to be catheterised on three occasions - twice by the writer. There was no history suggestive of prostatic enlargement, nor could the prostate be palpated on rectal examination. The patient was of a rather excitable disposition, and the retention was probably largely nervous in origin. Towards the end of these 48 hours, the temperature rose a little, but there was no local pain or other sign of wound infection: the temperature fell to normal in 2 days and was there for the rest of his stay in hospital. He was discharged on the 1st of September with the plaster fitting comfortably and the fragments in fairly good position considering the difficulty of immobilising the middle one.

He reported on the 3rd of October in plaster with no/
Case VI

21 November 1934

21 December 1934
Diagram of combined cortical inlay graft and intramedullary peg operation
28th December 1934

Surface view before operation.

Surface view after operation.

Longitudinal Section before operation.

Longitudinal Section after operation.

a — Strip cut from cortex of proximal fragment.
b — Strip cut from cortex of middle fragment.
c — Marrow cavity.
no evidence of union. The position was similar on the 24th of October.

On the 21st of November the plaster was bivalved: the fragments were still definitely mobile: X-ray examination showed increased anterior displacement of the middle fragment, and no union. A new plaster was applied.

He was readmitted on the 21st of December. The fragments were still un-united, there was mobility at the site of both fractures. It was thought that it was well-nigh impossible to immobilise the three fragments of tibia in a plaster case, so a bone-grafting operation was decided upon. This was performed on the 28th of December. An incision 7 inches long was made over the medial surface of the tibia. The periosteum was freed from the middle fragment, and a strip of cortex 2\(\frac{1}{2}\) inches long and \(\frac{1}{2}\) inch broad was cut from it: a strip 3\(\frac{1}{4}\) inches long and \(\frac{1}{2}\) inch broad was cut from the proximal fragment and slid down into the trench in the middle fragment, filling it, bridging the gap, and filling part of the trench in the proximal fragment and lashed into position with catgut. The distal fragment was too short to hold a sliding graft, so the strip which had been taken from the cortex of the middle fragment was manipulated into the medulla of the distal fragment and into the medulla/
Case VI

1 January 1935

29 March 1935
medulla of the middle fragment, in fact was used as an intramedullary peg. It was then found that the catgut was inadequate to fix the upper graft, so a piece of silver wire was bound round the tibia and the graft. The wound was closed and the plaster applied. There was a considerable degree of local pain after operation, but this passed off in a day or two, and he was discharged on the 9th of January 1935, comfortable.

He was readmitted on the 27th of March. He had been having calcium and thyroid and arsenic from his own doctor in an effort to hasten union. He had had occasional pain at the site of operation, usually described as 'pleasantly active', but sometimes of a dull gnawing character. The plaster was bivalved: the limb was in good alignment; there was but slight mobility and no crepitus. X-ray examination showed that union was progressing well. A light plaster was applied and he was discharged on the 29th of March 1935 in plaster, to the Astley Ainslie Institution, where he rested in bed in the open air. The plaster was bivalved, and he was given massage and graduated exercise. Calcium chloride gr. XXX and 'Adexolin' 3 tablets were given daily to promote more rapid union.

The plaster was removed on the 27th of August: there was a fair degree of union and a moderate degree of/
of ankle movement. X-ray examination showed that both fractures of the tibia were uniting, but union was not complete; the amount of callus was poor; the alignment of the upper fragment was good, but there was backward bowing of the lower; there was a good deal of decalcification in the tarsus. A short caliper was fitted.

He reported again on the 3rd of September; his condition was satisfactory and union was progressing. On the 11th of March 1936, there was firm union of the upper fragment, but still a little movement at the lower fracture. The general alignment was satisfactory. The fractures of the fibula had not completely united, a small separate fragment being displaced outwards. The short caliper was retained.
DISCUSSION.

This is a case of non-union following an uncommonly severe compound fracture.

As in the other cases of compound fracture reported, the element of low-grade infection probably played its part in hindering the repair. Probably the most important factor in the production of non-union was the mobility of the large middle fragment: it can be seen in various positions in the earlier X-ray prints. The problem of its blood-supply is a difficult one to solve. The fragment showed no sign of sequestration; the blood supply must therefore have been considerable: it could not have come from the nutrient artery, because it would surely be torn in the process of such a violent injury. It could not for the same reason have come from the metaphyseal vessels: so it must have been from the periosteum and neighbouring tissues. This is in agreement with the views of Gallie and Robertson, who attach great importance to the periosteal blood supply of bone.

The problem here presented to the reparative tissues was to build a double-arched bridge, the outer end of each arch being on firm ground, and the inner end being on a pontoon moving with the currents: this it was quite unable to do for obvious mechanical reasons. The only useful treatment was to fix/
fix the middle fragment in relation to the two other fragments, so that the repair of the two separate fractures could proceed. This was most effectively done by the insertion of two bone grafts.

The patient was given thyroid, calcium, arsenic and 'adexolin' during his convalescence. Thyroid speeds up many of the bodily processes, but it does not seem to speed up repair of a fracture. It has been shown that variations in the blood calcium compatible with life have no effect on the rate of healing; it is the local supply of calcium that is of importance. These measures, then, are of little value at the best of times, and are of no value whatever unless the fragments are in close apposition and immobilised.

The end-result in this case is highly satisfactory. The patient is rather over the age when the rapid healing of youthful tissues is to be expected: the injury was a severe one, and compound. He should, and does, consider himself very fortunate to be able to walk on his two legs eighteen months after his accident.
THE NORMAL REPAIR OF A FRACTURE.

Two factors must be present for new bone to be formed. They are:

1. Granulation tissue present, which needs an adequate blood supply.
2. Local excess of calcium.

In the repair of a fracture, there are four stages:

1. Outpouring of calcium; the fragments soon after the latter.

II. Preliminary fibrous tissue free of granulation tissue - the soft zone - granulation tissue contains osteoblasts which are able lining the bone in the developing stage of the endo- and immediately underneath the osteoblasts - the "calclium layer" of the periosteum by metaphysical osteoblasts.

The early work of Galeta and Behar has shown that the periosteum itself has no osteogenetic function, and that it is the layer of osteoblasts lying immediately beneath the periosteum which produces bone.

At this stage the ends of the fragments are being decahillal. The replacement at this early stage. It is probably the results of the initial trauma, hyperemia, decahillation being immediately the source of increased blood supply, whether it be of traumatic infective/
Two factors must be present for new bone to be formed. They are:

1. Granulation tissue growth, which needs an adequate blood supply.
2. Local excess of calcium.

In the repair of a fracture, there are four stages:

I. Outpouring and clotting of blood between the fragments soon after the injury.

II. Preliminary fibrous union from the production of granulation tissue - the soft callus. This granulation tissue contains osteoblasts derived from the cells lining the bone in the Haversian canals, in the endosteum and immediately underneath the periosteum - the "cambium layer" of the periosteum or subperiosteal osteoblasts.

The early work of Macewen and the later work of Gallie and Robertson have shown that the periosteum itself has no osteogenetic function, and that it is the layer of osteoblasts lying immediately beneath the periosteum which produces bone.

At this stage the ends of the fragments are being decalcified. The explanations of this are many. It is probably the result of the initial traumatic hyperaemia, decalcification being invariably the result of increased blood supply, whether it be of traumatic, infective/
infective or neoplastic origin. The fluid in the haematoma becomes acid; this increases the solubility of calcium, so according to Stirling it is withdrawn from the bone into the haematoma. Greig says the sole function of the osteoblast is "salt loss": with hyperaemia these cells are more active, so calcium salts are removed from the bone.

At this point, the formation of a "preosseous substance, probably a secretion of the osteoblasts" is described by Albee.

III. The calcium salts which have been accumulating are now precipitated in the soft callus (or in the preosseous substance) to form hard callus. The precipitation is brought about partly by the increasing density of the fibrous tissue which, being relatively avascular, produces a local ischaemia and leads to deposition of calcium salts, ischaemia always producing calcification. Stirling says that the fluid becomes alkaline again, so calcium is precipitated.

Much attention has been paid of late to the physico-chemical aspect of the problem of calcification. Blood, in addition to inorganic calcium salts, contains a phosphoric ester, the calcium salt of which is soluble. Tissues which become the site of calcification contain an enzyme - phosphatase - which hydrolyses this ester, and sets free insoluble inorganic calcium phosphate. It has been shown that if the pH falls below 7.3 the ester/
ester is hydrolysed and inorganic phosphates increase: so if there is a local excess of calcium this will lead to calcification.

IV. The hard callus is now ossified. The calcified tissue is invaded by bloodvessels carrying with them osteoblasts which clear the way in front of the vascular buds. It is reconstructed and adapted, in this way, bone being laid down around the vessels in layers, these trabeculae being arranged longitudinally or transversely according to the particular stresses at the point of fracture.

Around the ends of the fragments the lacunae contain no cells and no blood vessels. The nearest adult bone cells are separated from the site of fracture by this band of devitalised bone: so an essential part of repair is the revitalisation or removal of this tissue. Cowan says that injured periosteum produces granulation tissue which is quickly transformed into dense avascular fibrous tissue. The granulation tissue from the bone itself - chiefly from its marrow tissue - has delicate scanty fibrils and many bloodvessels: this may be the pre-osseous substance of Albee: calcium salts are deposited early in this. So Cowan thinks that the occurrence of union or non-union depends upon the result of the race between the granulation tissue from the periosteum and that from the bone.
This is the usual method of repair of bone, by the activity of specific osteogenetic cells derived from living bone, or by the metaplasia of fibroblasts, two "buds" of bone appearing, one from each fragment, and growing out to meet each other. Bone and cartilage formation result from the differentiation of osteogenetic cells in different environments, a local deposit of calcium and a good blood-supply being essential for differentiation into bone.

It used to be thought that all bone was formed by osteoblasts coming as such from bone; heterotopic bone was formed by osteoblasts from the blood-stream settling down in an area of injury where there was excess of calcium; this was the view of Macewen, who did so much of the early work on this subject.

A different theory is now more widely held - that there is no need for specific cells, and that ossification will proceed in any undifferentiated mesodermal tissue, provided that there is an adequate blood supply and a local excess of calcium.
I. GENERAL CAUSES:

The reparative process is strikingly independent of the general condition of the patient. Henderson's investigation has shown that most cases occur in patients between 20 and 50 years of age; thus advancing age, he says, has little effect on the rate of union. On the other hand, by far the greatest number of severe fractures is in patients between 20 and 50 who are exposed to the risks of the road, dangerous occupations, and the like, and the most common fracture in the aged - the neck of the femur - is notoriously liable to become the site of non-union.

Macewen found that fractures in young bones united much more rapidly than those in old bones; Albee found that the most stubborn non-unions were in the infant or young child.

The general health, nutrition and presence of intercurrent disease have no demonstrable effect on the rate of union, although Harris described lines of sclerosis indicating arrest of growth of bone in children during the course of infectious diseases.

The rate of repair is largely independent of the content of the blood. Variations of the blood calcium compatible with survival of the patient have no effect on the process. Kellogg Speed varied the calcium and phosphorus balance in healthy dogs and found that the only/
only variation producing any demonstrable effect on the healing of experimental fractures was absolute lowering of the blood calcium and relative raising of the phosphorus - produced by total thyroparathyroidectomy: union was markedly delayed: this is obviously of little clinical significance.

The administration of large doses of irradiated ergosterol to animals produces calcification in blood-vessels and soft tissues, but not at the site of fracture. Administration of calcium, parathyroid extract, thyroid extract and a host of other substances have all been found to have no influence on the rate of repair.

II. LOCAL CAUSES:

A. The granulation tissue may fail to bridge the gap.

This may be caused by the interposition of tissue which the granulations cannot penetrate, such as muscle, periosteum or detached avascular fragments of bone.

Cowan’s theory suggests the growth of dense avascular fibrous tissue from the periosteum blocking the growth of fine granulation tissue from the bone.

The granulation tissue may fail to bridge the gap because the fragments are so far separated that the two areas of granulation cannot meet; this is produced by extensive loss or removal of living bone, or by inadequate/
inadequate reduction or immobilisation of the fracture. In case VI of the series reported, there was a fragment of tibia which could not be immobilised except by operative means.

Bankart suggests that if there is absence of haemorrhage between and around the bone ends the usual irritant action of the outpoured blood will be absent, so there will be no stimulus for the formation of granulation tissue; he declares in no uncertain manner that absence of haemorrhage around the bone ends is the only common cause of non-union. He recalls the haemostatic effect of fresh muscle tissue, and goes on to say that in a violent fracture, if the ends of the fragments are thrust into muscle - as they may well be - there will be haemostasis, little formation of haematoma and non-union. Clan Murray, however, found that bones buried deep in muscles healed rapidly, there being a generous blood-supply from the lacerated muscles around the site of the fracture.

B. If there is arterial ischaemia, the granulation tissue may degenerate into scar tissue before ossification has been established. This is illustrated in Case III, where there was arteriosclerosis.

It is generally accepted that the nutrient vessels of a long bone maintain viability throughout the medulla, and supply the deeper parts of the cortex; the metaphyseal vessels supply the same components, and the superficial/
Blood supply of diaphysis
a - Periosteal supply
b - Nutrient
 c - Metaphyseal
superficial cortex as well, but they only supply the ends of the bone. The periosteal vessels supply the superficial layers of the cortex of the shaft. If a bone, then, is deprived of the supply from its nutrient artery, the middle of the shaft will be solely dependent upon its scanty periosteal vessels, there will be relative ischaemia there, and repair will be slow. The lower third of the tibia is a typical area of this kind, as the nutrient artery enters the tibia in its upper third.

Great importance is attached to the periosteal blood supply by Gallie and Robertson who say that stripping of the periosteum for any distance from the site of fracture will almost certainly result in non-union. Other writers stress the importance of the muscles and other soft tissues rather than of the periosteum itself. Ischaemia may also be produced by arterio-sclerosis. There being an ischaemia, the formation of granulation tissue is slow and its nutrition is not well maintained. The osteoblasts are poorly nourished and multiply slowly: if they do secrete phosphatase, they secrete that slowly too, and so sufficient calcium is not deposited to bridge the gap before the stage of contraction of fibrous tissue sets in. Bankart says that if there is a poor blood-supply, there will be little haematoma and so insufficient stimulus to repair.
It has been suggested that the devitalised area of bone next to the line of fracture is in a state of aseptic necrosis. If this avascular area is revitalised, it will join in the process of repair. If it is not, it will stay avascular, and form a dense plaque of sclerotic bone, obstructing further healing. This is particularly liable to happen if there is an ischaemia.

In this group, the end-result is usually a non-union of the fibrous type, the bones being fairly firmly united by dense avascular fibrous tissue.

C. Inadequate immobilisation has a definite bearing on the production of non-union, as is shown in Cases I, II, V and VI. It is obviously very difficult for two buds of bone to meet and unite if they are moving about in relation to each other; in addition to this, there are two conflicting views upon the effect of mobility of the fragments. Watson Jones maintains that "the only important cause of non-union is inadequate immobilisation; the many theories of inadequate blood-supply, impaired nutrition, inaccurate apposition of fragments, failure of impaction, inhibitory action of synovial fluid and absence of blood-clot are of no real significance." He points out that angulatory movement is usually controlled, but that rotatory torsion strains inhibit union just as certainly. He says that the repeated trauma to young connective tissue produced by/
by mobility leads to recurring hyperaemia and decalcification of the bone ends, so that a crack fracture becomes a cavity; if the fracture is now completely immobilised, it will unite. This is the stage of delayed union, which is cured by immobilisation and radiographically characterised by decalcification of the bone ends and little or no visible callus. If the fracture is not immobilised at this stage, there is no continuous bridge of callus to recalcify, the bone ends become sclerosed, calcium being deposited in them, and the fracture goes on to the stage of non-union, which is a permanent end-result, in which preliminary revascularisation is necessary to bring about union, and which radiographically shows a dense calcified barrier plate at the end of the fragments. On the other hand, Albee says that movement between the fragments produces disruption of young vessels, and the blood-supply becomes inadequate for connective tissue union and ossification.

If absolute fixation is maintained until soft callus is formed, and then interrupted, the vascular continuity between the intact bone and the mass of callus will be ruptured, and the callus will become an obstacle to union until vascularity is re-established. Movement of the fragments may mechanically upset the finely adjusted level of the pH described by Stirling and/
and Jones and Roberts by allowing leakage of other body fluids into the haematoma.

Bankart says in support of his "absence of haemorrhage" theory that it is quite untrue that imperfect immobilisation is a common cause of non-union, and points out that the ribs unite readily in spite of constant movement: but the fragments of ribs are more or less fixed by their strong soft tissue coverings, and there is little or no relative movement of the fragments.

Many fractures in the upper limb which united satisfactorily without being immobilised, even in the humerus, where non-union is so common, were described by Dowden. Movement of the limb as a whole, or of the muscles covering a fracture is beneficial because it promotes vascularity.

D. Hyperaemia has been blamed for many causes of delayed union. Hyperaemia, which may be produced by trauma or by infection in a compound fracture, or by the physical, chemical, or bacterial irritation of plating operations, decalcifies the bone ends and hinders the process of repair, the essential excess of calcium being reduced. This was a contributing factor in Cases I, II, IV, V and VI.
E. Delayed and non-union are phenomena most often seen in certain sites in the body, and not in individual patients with reasonably proper treatment. The most common sites are the shaft of the humerus, the middle of the shaft of the femur, and the lower third of the shaft of the tibia. Clan Murray thinks the explanation lies in there being a poor covering of muscle bellies in these regions: tendinous structures, he says, do not form a suitable nidus for granulation tissue growth. If the nutrient artery is damaged, there is in these three areas a poor blood supply, derived almost wholly from the periosteum: there is thus produced an ischaemia, the effect of which has been discussed. Gallie and Robertson point out that, especially in the lower third of the tibia, there is a large proportion of compact bone, the cancellous tissue with its active marrow being relatively little in amount. A further point which they do not discuss is that such marrow as there is in this area is composed mostly of fat, and has a low content of cells which could take any part in the process of repair.

F. The severity of the original trauma plays some part. Severe trauma frequently produces a compound fracture which may or may not become infected, and always unites less rapidly than a simple fracture. Severe trauma may also devitalise the tissues; it may deprive/
deprive them of that part of their blood-supply which they derive from the periosteum and neighbouring soft tissues. Severe trauma may produce gross displacement, as in Case VI.

G. Compound fractures nearly always unite more slowly than corresponding simple ones. This may be because many of them are infected. If they are irritated, packed or swabbed out, the haematoma will be disturbed, and Stirling's acid medium or Bankart's irritant action will be upset, thus encouraging the onset of non-union. Any interference tends to separate the soft tissues from the bone, diminishing its blood supply.

Of the 19 cases of fracture of the tibia and fibula admitted to Wards 7 and 8 between December 1934 and December 1935, 9 were compound and 10 simple. Two of the compound fractures went on to non-union and had to undergo operative treatment; none of the simple fractures went on to non-union. In the case of the remaining 7 compound fractures, the time from injury until the whole plaster was removed and a bivalved plaster or a walking caliper substituted ranged from 6 to 16 weeks, the average time being 11 weeks. The corresponding times for simple fractures were 6 to 12 weeks, and 8½ weeks respectively.

Upon these figures, small as they are, certain observations/
observations may safely be made:

(1) Compound fracture of the tibia and fibula occurs roughly as often as does simple fracture of these bones. This is probably because the whole of the anterior border and medial surface of the tibia is immediately subcutaneous.

(2) Compound fractures of these bones heal more slowly, on the average, than do simple fractures.

(3) Considering that five of the six cases reported in the first part of this paper were compound fractures, it may be said that compound fractures of the tibia and fibula are more susceptible to non-union than are simple fractures.

It is also found that the application of B.I.P.P., or the inefficient application of plates or screws is frequently associated with upset of union; this may be from their irritant action producing hyperaemia, and decalcification of the bone ends.

H. Pathological fractures, at the site of a primary or secondary osteolytic tumour, rarely unite without operative treatment, the cause of the fracture still being present.

I. The effect of upset of the nerve supply upon healing does not appear to have been worked out, or even seriously considered. McMaster and Roome performed/
performed experimental lumbar sympathectomy on dogs: they found that this did not hasten bony repair in the fibula; in the majority of cases healing was retarded. Section of the sciatic nerve, they found, had no effect on healing of the fibula. Pearse and Morton found that removal of the lumbar sympathetic trunk and ganglia made very little difference to the process of bone repair in the lower limb.

There are no easily found reports in the literature on the rate of healing of fractures in such conditions as tabes dorsalis, which has a marked effect upon the joints, or in anterior poliomyelitis. The writer has seen one case of this kind: a man of 46 sustained a fracture of the femur; while in the ward he developed G.P.I. and died 22 days after the injury; there was no sign - on X-ray examination - of production of callus even two days before he died. Permission for autopsy was refused.
CLINICAL FEATURES.

It is most important to distinguish between delayed union and non-union, because their treatment is quite different.

Delayed union is a condition where the reparative process takes longer than the accepted normal time: a fracture of the tibia is said to show delay if it takes more than eight weeks to unite.

There is a considerable degree of movement at the site of fracture, accompanied by pain and complete loss of the supporting function of the leg: there may be crepitus. Radiographic examination shows lack of distinction between the cortex and medulla and decalcification of the adjacent ends of the fragments.

Non-union is a permanent end-result: if a tibia has not united in six months, there must be a strong suspicion of non-union, although a delayed union may still heal after that time. There are three varieties of non-union:

1. Fibrous union, which is particularly liable to follow arterial ischaemia. The erstwhile complete bridge of granulation tissue has degenerated into avascular scar tissue, which holds the bones together. There is present a degree of movement dependent upon the density of the fibrous tissue and on the distance that the fragments are apart: it is usually only slight.

There/
There may be little disability: Hey Groves reports that Livingstone explored for many years with an un-united fracture of the humerus. There is little or no pain on movement: there is no bony crepitus. The bone ends are thickened and eburnated, and they show a dense shadow on the X-ray plate.

2. **Pseudarthrosis** is found where bony union has been prevented by the movement of the fragments: there is a space between the bone ends filled with pseudo-synovial fluid enclosed in a capsule representing the peripheral portion of the original granulation tissue. There is limited painless movement, free within the range allowed by the tension of the joint capsule, and limited by the capsule and pain felt on too zealous movement. The radiographic appearances are as in fibrous union - a dense calcified area at the end of the fragment.

3. **Gap fracture** is present where there is no evidence of granulation tissue ever having bridged the gap, the fragments being entirely disconnected. This is most frequently seen when there has been no attempt at treatment, or where a bone such as the fibula is being held apart by its companion bone. There is free mobility of the fragments in relation to each other. Radiological examination shows dense sclerosed plates at the ends of the fragments.
TREATMENT OF DELAYED UNION AND NON-UNION.

PREVENTION.

Prevention is important because it is frequently possible and because of the great saving of time, energy, and money made by securing early union.

The essential features are the complete angulatory and rotatory immobilisation of the fracture with the fragments in reasonably close apposition: movements of the limb as a whole and of all joints which do not need to be immobilised promote vascularity, and are therefore beneficial. There is evidence to show that venous stasis promotes union: this can easily be produced in the leg by an efficient walking splint or by simply hanging the leg down as in sitting. A minimum of interference with the site of fracture is desirable. Stirling suggests that at sites where delayed union is common the routine injection around the fracture of a slightly alkaline mixture containing calcium chloride, magnesium sulphate, sodium glycerophosphate, and calcium phosphate should be carried out, and says that the process of repair seems to have been expedited in some cases. Other writers recommend a mixture of powdered calcium triple phosphate, calcium carbonate and blood, with fairly good results. The most common view is that neither the salts in the proportion in which they occur in bone, nor bone powder, stimulate osteogenesis/
osteogenesis when implanted in a bone defect. Rather than this, Clan Murray advocates routine early open reduction of all fractures in regions prone to non-union: nearly all writers disagree with this view. Bankart recommends the local injection of 15 c.c. of blood, and quotes no less than one case in support of his treatment.

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Convincing clinical and experimental evidence has been produced by Pearce and Darby to show that venous stasis hyperemia promotes the growth and repair of bone; they recommend its use in cases where either the fracture is delayed and where, from the position of the fragments and the condition of the patient, union might be expected. They experimentally produced venous stasis in one limb by ligation of venous trunks, and found that the experimental fracture of the tibia healed much more rapidly than that in the normal side. Various pathological conditions causing venous stasis were also described.
TREATMENT OF DELAYED UNION.

Delayed union being little more than a state of torpor or exhaustion of the reparative tissues, the treatment is essentially to refresh them and give them time to do their work.

Prolonged complete immobilisation in good position will nearly always result in union, as is shown in Case II, and will probably be shown in Case I. Careful active movements with a splint to prevent displacement frequently promotes union by forcing the bone ends together, thus producing a frictional stimulus and hyperaemia. A Thomas' short walking caliper or Bohler's walking iron are effective in this way for a case involving the tibia.

Convincing clinical and experimental evidence has been produced by Pearse and Morton to show that venous stasis hyperaemia promotes the growth and repair of bone; they recommend its use in cases where union is delayed and where, from the position of the fragments and the condition of the patient, union might be expected. They experimentally produced venous stasis in one limb by ligation of venous trunks, and found that the experimental fracture of the fibula healed much more rapidly than that on the normal side. Harris described pathological conditions causing congestion of various/
various degrees, producing increased growth of the affected bones in children: these diseases included such things as haemangioma of the leg, femoral and iliac thromboses, and recurring haemarthrosis of the knee. Venous stasis may be produced therapeutically by hanging the leg down as in walking or sitting. It is often produced by the application of a Martin's bandage to the limb, just tightly enough to produce venous engorgement, for periods ranging from twenty minutes to several hours: this may be reinforced by jarring of the bone ends by striking the suitably protected heel several times with a hammer: this has the same effect as walking. This method was employed in Case III: the fracture went on to non-union.

Albee says that the stasis treatment is of no benefit, but he approves of the hammering.

The treatment of delayed union, then, is not operative, and consists of immobilisation and measures to promote vascularity of the healing area. It should be controlled by repeated clinical and radiographic examination and continued for at least three months or until the fracture unites. If no progress is being made, the case should be considered as non-union.
TREATMENT OF DELAYED UNION IN COMPOUND FRACTURES.

In an ununited fracture which was originally compound, if a sequestrum forms the infection is still active and the bones are pathologically in the de-calcified state of delayed union: the only treatment called for is removal of the sequestrum and prolonged immobilisation without further disturbance: union will nearly always follow. This is the result expected in Case I.

If the infection is quiescent, hyperaemia has died down, sclerosis is present and the condition is treated like any other case of non-union.
Back's Operation.

a - Proximal Fragment.
b - Distal Fragment.
c - Skin incision, showing direction of drilling.
d - Sclerotic bone-ends.
e - Marrow cavity.
TREATMENT OF NON-UNION.

In non-union the sclerosed bone ends form an impenetrable barrier to the osteogenic cells; being a permanent condition, it is not cured by conservative means: the essential part of successful treatment is the wide contact of healthy vascular bone surfaces: this can only be produced by active surgical measures. So operative treatment is indicated in all cases of non-union. It is also indicated in certain cases of delayed union - where mobility is increased and X-ray examination shows rounded bone ends separated by a definite gap, or where union is excessively delayed - more than nine months in the tibia. The presence of sepsis is a definite contra-indication to major operative procedures: it is not of such serious import in the simpler revitalising operations.

Stimulation of the bone ends may well be effective in early cases if the fragments are close together, or they can be easily brought into contact. The simplest form of this treatment is Beck's operation, in which the bone ends are drilled in different directions with a stout Kirschner wire through two small skin punctures, one over each fragment. This produces hyperaemia, the sclerosed bone ends are mechanically broken up, and the imprisoned cells are liberated: the fracture is then completely immobilised in plaster, and callus usually/
Step-Cut

Conical Shaping
usually begins to form in four to six weeks. This method is recommended before more drastic treatment is attempted. It is simple; it frequently cures the condition; if it fails, no harm has been done, and more radical measures have not been prevented.

A rather more severe procedure is to cut down upon the fracture, remove all the dense scar tissue between the bones and raw the bone surfaces by scraping or chiselling them; multiple drill holes or saw cuts are then made in the bone ends, the periosteum being preserved intact if possible, and the fracture put in plaster as before. Albee disapproves strongly of the drilling method.

**Plastic Operations** of various kinds have been devised with the object of securing wide contact, close apposition and firm fixation of fresh bone surfaces: they are recommended by Hey Groves. Wide contact may be secured by shaping the ends as a double cone; a step-cut operation is particularly recommended for oblique fractures, and sectorial interlocking for atrophic bone. Close apposition is produced by the use of metal bands, plates, or catgut. Fixation is in plaster or an external splint for at least three months. All these methods necessarily lead to shortening, and so are not often used in the tibia where it is important to avoid shortening if possible.
Diagrammatic representation of Cowan's operation.

A: Incision of periosteum.
B: Lifting up of periosteum, and making of gutter in fragment.
C: Removal of fibrous tissue.
D: Approximation of fragments.
E: Fragments held by band of rib cortex.
F: Suture of periosteum.

Redrawn from "Annals of Surgery"
Cowan, in the light of his theory that dense fibrous tissue from the periosteum chokes the fine granulation tissue from the bone, has a special treatment for non-union. The periosteum is elevated and a gutter cut in the cortex right down to the marrow to provide a wide channel for callus; the sclerosed bone ends are now removed; the fragments are brought into apposition and the line of fracture is surrounded by a thin ring of the cortex of a rib: the object in applying this ring is not to provide a new source of osteogenesis, but to shut off the thick granulation tissue from the periosteum; the limb is then immobilised. The treatment, involving the removal of the bone ends, the production of a new source of osteogenesis, an increase in the vascularity, and immobilisation, is usually effective, although whether the shutting out of the periosteum has any effect or not is a matter of some doubt.

Mechanical fixation has been recommended: freshening of the bone ends, followed by suturing, wiring or metallic plating may promote union. Metals, with the possible exception of stainless steel, appear to inhibit osteogenesis, and all foreign bodies tend to favour low-grade infection, which may appear early or late. Wiring may be followed by a spontaneous fracture years after treatment from a line of osteoporosis at the site of the wire.

Juvara/
Juvara in 1928 described his "fixateurs externes" - they consist of four long metallic screws, two of which are introduced into each fragment through small skin punctures: the other end of each screw projects through the skin and is attached to a longitudinal external bar, upon which it can be adjusted; the fragments are thus immobilised, the bone ends having been freshened. The advantage of this apparatus is that when union occurs, the screws can be taken out without disturbing the fracture.

Fixation by plating and screwing with boiled bone is recommended by some writers; bone is not irritant as metals are, and is absorbed and replaced by new living bone. This appears to be the only safe way of plating fractures without having to open up the wound again some time after operation to remove the metal plates.
BONE GRAFTING.

This is the method of choice if the attempt to promote union by Beck's drilling operation has failed.

Gallie and Robertson found that when a piece of bone has been cut free from its circulation and transplanted to some other position in the same animal and its surfaces are freely bathed in lymph, those cells which are present on the surface and in the open mouths of the Haversian canals live and undergo rapid proliferation; the cells in the lacunae and deep in the Haversian canals die. If this piece of bone has been transplanted into another bone, the proliferating cells on the surface, whether from the graft itself or from the neighbouring living bone, immediately set about the absorption of the graft, and do not stop until every vestige of it has disappeared. Meantime new bone is being laid down on the surfaces and a circulation is being established through the whole graft by the ingrowth of new blood-vessels: osteoblasts follow the vessels and continue the process of absorption in the interior of the bone. During the first two months, absorption is more rapid than replacement, and if the graft has no strain put upon it, as is the case when it is experimentally transplanted into muscle, the absorption will go on until the place of the graft is taken by ordinary scar tissue. If the graft has a strain/
strain put upon it and has a function to perform, as when it is transplanted into bone, it steadily thickens and hardens until it assumes a form and density suitable for its work. MacEwen said in 1912 that diaphyseal grafts lived and actively proliferated in their new surroundings; it is now thought that the graft has no power of independent growth.

If heterogenous or boiled bone is placed in extensive contact with living bone, the result is the same as that which follows the implantation of autogenous bone. The cells in the heterogenous graft die, it is absorbed by cells from the living bone, and new bone is laid down in the excavated areas. In bridging a gap in an ununited fracture, only an autogenous graft can be used, as cells survive on its surface to replace it, whereas a boiled or heterogenous graft is only invaded at its end by osteoblasts from the fragments.

A graft provides an internal fixation splint, it may act as a strut, restoring continuity; it acts as a scaffold in and around which new bone is built. Its power of producing new bone is disputed and limited. To ensure union the graft must be firmly fixed in position; it must be efficiently immobilised, and there must be wide contact between living bone surfaces. The blood supply to the bed of the graft must be adequate or the process of non-union will be repeated.

The graft may be external, cortical or intramedullary.
The external graft may be of autogenous bone or of boiled bone. The application of slivers or wafers of healthy bone covered with periosteum to the rawed external surface of the fractured bone has now been almost universally discarded. The slivers have to be fixed in position: if the fracture is to unite the sclerosed ends of the fragments must be taken away, and the addition of a wafer of bone on the outside is a waste of time and of no value in promoting union. The same may be said for the use of living bone chips: it is not the presence of the chips, but it is the rest of the operative and post-operative treatment that produces union. External grafts of boiled bone fixed with bone screws, as recommended in some cases by Gallie and Robertson, may bring about union, being themselves absorbed and replaced.

The sliding cortical graft is enthusiastically advocated by Albee. It has four distinct points in its favour: it is probably the easiest of the grafting operations to carry out: the graft is brought into intimate contact with the bone for a considerable distance: the fragments are freely cut into, the osteoblasts being liberated and the sclerotic plaques broken down: it helps in the maintenance of immobilisation. This method is the most widely practised form of operative treatment for non-union in the tibia; it is not, however/
Diagram of sliding cortical bone grafting operation for non-union.

I (before operation)

II (after operation)

III (after operation)
(Cross section of II on line r)

a. Strip of cortex cut from proximal fragment.
b. Strip of cortex cut from distal fragment.
c. Dense fibrous tissue between fragments.
d. Plaques of sclerotic bone.
e. Proximal fragment.
f. Distal fragment.
g. Marrow cavity.
h. Catgut suture, showing method of looping.
however, suitable for some fractures, such as those of the mandible, ulna and radius in which an intramedullary peg is better. This type of graft was used in Cases III, IV, V and VI.

The tibia is approached through a generous incision along the medial surface of the bone: the region of the fracture is freely exposed, the soft tissues are separated from the bone and the periosteum incised longitudinally and carefully reflected: it may be necessary to raise the superficial bony lamellae with the periosteum. The bone ends are freshened with a chisel or osteotome. The graft is then cut from the whole thickness of the cortex of the upper fragment: it is about four inches long and about half-an-inch wide. A single-bladed or double-bladed Albee saw is used; if the operation field is kept at a normal temperature by the liberal use of saline, this has no bad effect upon the vitality of the bone. A trench of the same width and about two inches long is then cut in the lower fragment, and the graft is slid down into its new position: there it fills the trench in the lower fragment, bridges over the gap and occupies the lower part of its original bed: the piece of bone removed to make the trench fills the upper part of the cavity in the upper fragment. The use of a fixed double-bladed saw ensures accurate fitting of the graft into its bed, but if a single blade is used, the edges of the/
the bed and of the graft can be bevelled: the graft will then lie more snugly in position and a firm turn of kangaroo tendon round the bone will press it home. The graft may be fixed by kangaroo tendon looped as in the diagram instead of tying the tendon right round the bone. The periosteum is stitched back into position, the soft tissues are allowed to fall back and the skin wound is closed. The tibia is immobilised in a plaster from the toes to mid-thigh for eight weeks: the plaster is then bivalved and physiotherapy, in the form of diathermy and massage, instituted: active movements are started early. If the fracture is not consolidated in twelve or fourteen weeks, the patient should be given a walking caliper and encouraged to carry out movements of the limb as a whole.

In the massive cortical inlay graft the trenches are cut in the proximal and distal fragments as before, but they are filled by a strip of cortex some 6 inches long from the medial surface of the other tibia. This possibly gives a more stable graft, but if the affected tibia is otherwise healthy, there is no great advantage in this type of graft.

Intramedullary grafts are particularly suitable for ununited fractures of the mandible, radius or ulna, where there is relatively thin cortex upon which to work for a cortical graft. The bone ends are exposed and the sclerotic parts removed. The graft is trimmed to/
to a suitable size and shape and driven hard into the marrow cavity of one fragment until it is firmly wedged there. It may be possible to manoeuvre the rest of the graft into the medulla of the other fragment, but it is usually necessary to lay open the cavity, put in the graft and close it up again. If the fracture is near the end of a bone as in supracondylar fracture of the humerus, the graft may simply be driven upwards into the medulla through the end of the bone. In a gap fracture, separate pegs may be wedged into each marrow and then the pegs fixed to each other. The peg itself may be of ivory, boiled beef bone, or living cortical bone: there seems to be no particular advantage of one over the other.

The intramedullary peg method has not found great favour because it involves the digging out of valuable marrow, which is one of the best sources of blood, so the ischaemia is apt to be aggravated.
CONCLUSION.

1. The most common cause of delayed union or non-union is absence of complete and sufficiently prolonged immobilisation. Infection of the area of healing is also a common cause of delay.

2. The best treatment for delayed union is prolonged complete immobilisation, preceded by sequestrectomy if necessary.

3. The best treatment for non-union is drilling of the bone ends by Beck’s method. If this fails, a sliding cortical graft is indicated.
REFERENCES TO THE MORE AUTHORITATIVE LITERATURE CONSULTED:


Harris: "Bone Growth in Health and Disease" 1933.


