PATHOLOGICAL OSSIFICATION IN TRAUMATIC PARAPLEGIA*

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One of the most interesting sequels of paraplegia is the formation of large masses of ectopic bone. In spite of the publication of over 100 papers, including an authoritative survey by Ceillier in 1920, knowledge of the etiology, natural history and clinical manifestations of the condition has remained rudimentary. Reports of solitary or small series of cases have been made for years and some experimental work has been done.

The type of ossification is illustrated in Figure 1. The chief feature is the great mass of new bone which is formed mainly anterior to the hip, producing an extra-articular bony ankylosis. The other principal sites of ossification are the adductor muscles in the thigh (Fig. 2) and the medial side of the knee (Fig. 3).

FIG. 1

Specimen showing typical ectopic ossification in front of the hip, resulting in extra-articular bony ankylosis.

The condition presents points of interest in orthopaedic surgery, neurology, radiology and morbid anatomy, and has been the subject of detailed study on our part for a number of years. The purpose of this paper is to report the clinical findings and indications for treatment in 100 cases and to draw attention to what we believe to be the main pathological problems that await solution.

New bone formation was found in 100 out of 603 patients with paraplegia. This incidence of 16 per cent is not necessarily a true one because only 250 patients were radiographed specifically for evidence of abnormal ossification. It is likely therefore that the true incidence is much higher. Déjerine and Ceillier (1919) found an incidence of 48.7 per cent, Liberson (1953) 53.3 per cent, Abramson (1948) 41 per cent and Soule (1945) 37 per cent.

Of the 100 cases in which ectopic bone was found, the paraplegia was due to trauma in ninety-three and to non-traumatic causes in the remaining seven. The upper level of the cord lesion was in the cervical region in five patients, in the upper thoracic region in fifteen, and in the lower thoracic and thoraco-lumbar region in seventy-eight: no definite level could be found in the remaining two. All the cord lesions had produced a severe if not total paralytic disability, which was spastic in forty-two instances and flaccid in fifty-eight.

In all the patients ectopic bone formation occurred in the soft tissues below the level of the cord lesion, and in all it occurred between the upper border of the pelvis and the knee. This conforms with the observation of Ceillier (1920), who recorded new bone in and about the front of the hips, about the ischial tuberosities and inferior pubic rami, along the shaft of the femur and on the medial aspect of the femoral condyles.

* Part of the material of this paper formed the basis of a Hunterian Lecture given by J. W. D. before the Royal College of Surgeons of England on January 22, 1959.

FIG. 2

FIG. 3

Figure 2—Characteristic "lacy" ossification in the mid thigh. Figure 3—Ossification at the medial side of the knee.

Local soft-tissue necrosis and infection were observed in fifty-two patients, but in forty-eight there was no evidence of sepsis.

On the basis of these facts and figures it was possible to divide pathological ossification into three main types: one associated with skin and joint sepsis with considerable local tissue destruction, one associated with extensive decubitus ulceration overlying the bony prominence of the greater trochanter and ischial tuberosities, and one in which new bone appeared without any local sepsis or other precipitating factor. This third type has been more commonly referred to as myositis ossificans in paraplegia. Several patients showed more than one type of new bone formation.

NEW BONE AND SEPTIC ARTHRITIS

New bone formation occurred in five patients with severe infective arthritis of the hip that had destroyed the joint. It was massive in every instance and involved all adjacent soft tissue without any preference for muscle or parts of muscle, and it followed the path of the local infection and necrosis (Fig. 4).

FIG. 4

Ectopic ossification in paraplegia, associated with infective arthritis and fracture of the femoral neck.

FIG. 4

femoral condyle. In some patients the new bone formed on both sides, in others on only one side. In several patients it occurred simultaneously in more than one site.

In thirty-two of the forty-eight patients new bone was formed about the hip joint. In eighteen patients the diagnosis was made only after radiological examination of the hips, when flakes and strips of bone were demonstrated in front of and just below the joint. The lesions appeared to be located in the flexor or adductor muscle masses and were not directly attached to the skeleton. In the remaining fourteen patients the new bone was massive enough to restrict passive joint movement. The bone bridged the gap between the ilium and the intertrochanteric line of the femur. In ten patients the lesions were bilateral and in four

FIG. 7

FIG. 8

Figure 7—Early new bone formation. The circumferential wire marks the limits of the palpable swelling; the central loop marks the highest part. Figure 8—Ossification which began to develop thirty days after traumatic paraplegia is becoming mature.

unilateral. In every case soft-tissue swelling and limitation of hip movement occurred before the bone formation became apparent radiographically. Early ossification is shown in Figure 7, the moderately developed condition in Figure 8 and the advanced stages in Figures 1 and 12.

Ossification occurred in the region of the mid thigh in eleven patients. In six of these the new bone was massive (Fig. 2) and was unrelated to a pathological fracture or haematoma. Of the rest one occurred in a haematoma, two followed pathological fracture (Fig. 9) and two occurred as a bony thickening adjacent to the periosteum. Probably only the first six of this group were cases of true ossification in muscle like the lesion described by Ceillier (1920), starting as bony needles in the interfascicular connective tissue and without any direct connection with the shaft or periosteum of the femur. As in the hip, the deposits were preceded by a palpable firm mass in the muscle before the new bone became evident radiographically.

Ossification occurred about the knee in eighteen patients. It was apparent clinically in six cases but in the rest it was discovered by radiography. It always occurred on the medial condyle of the femur, sometimes as a mere flake or button of bone, but sometimes extending down to the joint capsule and up to the shaft of the femur (Fig.) 3. New bone formation in this situation was never associated with local sepsis.

DISCUSSION

New bone formation in the soft tissues and about the joints in patients suffering from disease of the nervous system has been given many names, of which para-osteo-arthritis (Déjerine and Ceillier 1919) has become the most widely used although neurogenic ossifying fibromyopathy (Soule 1945) is pathologically more correct. Early accounts of it are mainly in German. Riedel (1883) is usually credited with the first description and later papers were by Eichhorst (1895), König (1906), Pötzl (1908), Küttner (1908), Steinert and Versé (1910) and Schlesinger (1917). In these descriptions a distinction was not always made between neuro-

FIG. 9
Hyperplastic callus after pathological fracture of the femur in a traumatic paraplegic. Marked subperiosteal new bone.

arthropathies with joint disorganisation and ectopic ossification in extra-articular tissues. No attempt was made to separate those cases in which new bone formation was clearly associated with sepsis. Since new bone formation constantly occurs in chronic pyogenic infections of bone, it is not really surprising that it occurs in paraplegia in association with suppurative arthritis, and with infected decubitus ulcers over the greater trochanters and ischial tuberosities.

The frequent formation of ectopic bone in the soft tissues in traumatic paraplegia in the absence of sepsis or of evident tissue necrosis is however surprising, and so far as we are aware there is no known explanation. The French school of neurology has made the clearest and the most detailed contribution based on cases of traumatic paraplegia from the first world war. In a series of papers Professor and Madame Déjerine and André Ceillier (1918-19) described it completely. Ceillier (1920) added to the number of cases and recapitulated his views in a thesis which remains the most important single reference. He described eighty examples in 160 patients, and postulated a modification of the intermuscular and intramuscular fibrous tissue in paralysed limbs, followed by microscopic areas of haemorrhage. Oedema was considered important. The level of the cord lesion was unimportant but its severity was. All the stages of bone formation were seen from ossification of fibrous tissue through cartilage formation to the formation of haemopoietic marrow and Haversian systems. More recent papers by Coste, Galmiche and Brion (1950) and by Benassy (1957) have amplified this work.

We are able to confirm many of the observations made by Heilbrun and Kuhn (1947), Stanger (1947) and Roche and Jostes (1948). We have not seen ossification about the shoulder or in the upper limb as described by Abramson (1948). We agree with Abramson and Kamberg (1949) that there is no relationship between ossification and the level or pattern of paralysis.

In a clinical study of heterotopic ossification in paraplegia Damanski (1961) stressed the importance of skin ulceration, hypoproteinaemia and severe urinary tract infection. We do not think that these factors play any part in the appearance of myositis ossificans although they are factors associated with bone formation arising as a secondary process in a necrotic area.

It is our experience that new bone formation in muscles is not specifically related to inadequate mobilisation of the patient or to nursing the patient in the prone position for long periods. A little over one-third of our patients with new bone formation did not at any time have skin ulceration, hypoproteinaemia or severe urinary tract infection. They all had an active programme of physical exercise beginning within days of the injury.

We are aware that ectopic ossification occurs in many neurological disorders other than those due directly to trauma to the nervous system. The neurological disorders in which ectopic ossification is known to occur include myelitis and encephalomyelitis from many

causes; poliomyelitis, hemiplegia and hemiparesis after cerebro-vascular disease; cerebral tumour, meningitis of various types, intracranial and epidural abscess, head injury, multiple sclerosis, spinal cord tumour, spinal cord thrombosis, polyneuritis of various types and even coma from carbon monoxide poisoning. Some of these features are discussed in papers by Lorber (1953), McNeur (1954), Irving and Le Brun (1954), Storey and Tegner (1955), Johnson (1957) and Spiele (1959).

The only factor common to these conditions is serious damage to the nervous system. Probably the incidence of ossification seems higher in traumatic paraplegia than in other neurological conditions only because full opportunities exist for investigating cases of paraplegia in special units, whereas other neurological disorders demand radiography of the pelvis or lower limbs relatively seldom. The more often the condition is sought the more often it may be found.

In this series of cases all the changes were found in the soft tissues below the level of the cord lesion—that is, in paralysed muscle. No new bone was found in paralysed areas above the pelvis or below the knee, even in patients with tetraplegia.

The masses of new bone were either single or multiple, and showed much variation in size and extent. When large, the mass of bone was preceded by palpable soft-tissue swelling and limitation of passive movement of the hip joint. In most patients the new bone appeared early, rapidly increased in size and then remained unchanged. The earliest appeared nineteen days after the spinal cord injury and the latest after seven months. When the changes were seen from the beginning the time taken from the first thickening to the fully formed bone was about three to four months. Once a mass of ectopic bone was fully formed further extension never occurred, even after partial surgical removal, nor did new ectopic bone appear in other sites.

There was no apparent relationship between the occurrence of ossification and the level or severity of the cord lesion, or the degree of spasticity or flaccidity, though all the patients had lesions of the spinal cord between the conus medullaris and the lower cervical segments.

In three patients swelling, limitation of movement and formation of new bone occurred during the phase of returning reflex activity after a mid-thoracic spinal cord transection. In two patients the same clinical and radiological features appeared after traumatic transection of the conus medullaris with an intramedullary haemorrhage extending upwards to the tenth thoracic segment which produced total paralysis below the ninth thoracic segment and total loss of reflex activity below that level. In one of these patients the new bone formation was unilateral and in the other it was bilateral.

If cases with coexistent or associated sepsis are excluded, all the remainder had spinal cord lesions above the conus medullaris and below the lower cervical segments. No case has so far been associated with the conversion of spastic cord lesions to flaccid lesions by intrathecal injections of alcohol or phenol.

We were unable to establish any connection between direct trauma to the muscles during passive stretching and new bone formation. In most patients the paralysis was symmetrical and physiotherapy in the form of passive movement was given to both legs; yet new bone developed in one thigh and not in the other. We were unable to establish a history of direct trauma in any patient in whom new bone formation occurred, and yet conversely quite severe injury to a limb in a severely paraplegic patient did not result in massive new bone formation. The commonly observed myositis ossificans traumatica or ossifying haematoma differs in its relatively circumscribed distribution and in the absence of any nerve lesions. It is of interest that patients with traumatic paraplegia in whom characteristic ectopic bone developed showed no ossification at the site of muscle injury from shrapnel fragments (Soule 1945). If muscle trauma were an important factor it would surely result in an ossified haematoma in these cases.

Histochemical studies of some of our material have already been reported by Curran and Collins (1957). Histological examination has failed to confirm the theory that ossification

FIG. 10

Low-power photomicrograph of tissue from ossified mass about the hip joint. A—atrophic muscle fibres; B—young fibrous tissue surrounding and replacing atrophic muscle fibres; C—young fibrous tissue being converted into woven bone. There is no evidence of multiple haematomata or of spread from distant periosteum. The process is one of multicentric ossification in young fibrous tissue. ($\times 80$.)

FIG. 11

Conversion of woven to lamellar bone. Cortex, Haversian systems and haemopoietic marrow will follow. ($\times 250$.)

occurs in a haematoma, and new bone formation has been found arising multilocally in young fibrous tissue. Figures 10 and 11 illustrate the replacement of atrophic muscle fibres by fibroblasts (far removed from the periosteum), the formation of woven bone and its transformation into lamellar bone.

Apart from any association with paraplegia, pathological calcification and ossification have a large literature, of which authoritative reviews have been published by Barr (1932), Mulligan (1947) and Hass (1956). Attempting a general statement, Hass stated that "perhaps the most important initial change which favours pathological calcification is the modification of a matrix in the direction of reduced viability without an undue disintegration of the structural pattern of the matrix or enhancement of its vascularity." He emphasised that pathological calcification is largely a matter of a variety of microchemical activities associated with calcium salts (deposition, resorption, exchange, displacement, transfer, crystallisation, recrystallisation), all of which can occur in a very small microscopic field of tissue due to the large surface area available in the bone salt. These activities need not be reflected at all in gross measurements like serum calcium and phosphate estimations performed on the peripheral blood. Gross calcium, phosphate, phosphatase and protein estimations were made in most of our cases but no significant results were obtained.

McLean and Urist (1955) summarised their views by stating that ectopic ossification is not to be regarded as metaplasia of connective tissue elements previously in existence in the lesion; nor as a simple sequel to deposition of calcium salts in dead tissue, but as a reaction of perivascular connective tissue cells growing into injured tissue from surrounding areas.

Bridges and Pritchard (1958) suggested a substance, possibly a protein, which when liberated from degenerating muscle may promote ossification in local connective-tissue cells.

Curran and Collins (1957) studied intramembranous ossification in normal and pathological human material (including some from our cases) by autoradiography and selective staining of acid mucopolysaccharides. They found that ossification takes place in a connective tissue rich in acid mucopolysaccharide, much of it sulphated, and that it can be demonstrated in the matrix of woven but not of lamellar bone. However, in spite of all these detailed observations the precipitating factor necessary for the appearance of new bone in muscle in paraplegia remains undetected.

TREATMENT

The prevention of decubitus ulceration in paraplegic patients depends on the disciplined routine of nursing management in which the patient is turned in bed at regular intervals and learns to take meticulous care of his skin. In this way the incidence of skin ulceration and joint sepsis can be greatly reduced and accordingly the formation of ectopic bone or calcified plaques in association with sepsis cannot occur.

The problem of the removal of ectopic bone and calcification associated with sepsis is more related to the treatment of the soft-tissue ulceration and sinus formation than to the removal of new bone itself. In the excision of sinus cavities in decubitus ulceration only sufficient new bone is removed to allow closure of the defect.

On four occasions only have we removed the head and neck of the femur in the treatment of a large ulcer overlying the greater trochanter. New bone arising in and about the ischial tuberosities has been chiselled off piecemeal in the process of excising the scar and infected area. We have never undertaken radical excision of the ischium.

There is no known method of preventing new bone formation of the type which occurs in the absence of sepsis. Moreover, once the change has occurred in any muscle or muscle group no treatment is known which will prevent the progress of the change until ossification is complete. Fortunately it is only in some patients with massive ossification about the hips that new bone formation aggravates the patient's disability. Massive new bone formation in the iliopsoas muscle can cause extra-articular ankylosis of the hip, preventing passive flexion

and greatly interfering with the patient's independence. Moreover, ankylosis of the hip increases the risk of extra pressure on the skin on the back of the thighs and buttocks.

In eight patients in our series the masses of new bone in front of the hip joints were sufficient to cause a hindrance in the activities of daily life and to give a clear indication for operative intervention.

All authors are agreed that operation is rarely indicated. Street (1958) described operations on four patients and emphasised that surgery should not be lightly undertaken and that haematoma formation, wound breakdown, infection, sinus formation and osteomyelitis could be some of the hazards. Miller and O'Neill (1949) reported excision of bone from one patient and Soule and Stiff (1949) reported disarticulation of the lower limb. Armstrong-Ressy, Weiss and Ebel (1959) recorded four cases treated by surgery, one by bilateral femoral head and neck resection, one by a similar operation combined with a Judet arthroplasty, one by excision of ectopic bone and one by high bilateral thigh amputation.

FIG. 12

FIG. 13

FIG. 14

Figure 12—Established new bone in a traumatic paraplegic. Figure 13—The same case after excision of bone. It is not necessary to remove the whole mass, and it may be impossible to do so. Figure 14—The same case three years after excision.

Any operation on a paraplegic patient demands efficient haemostasis in the unduly vascular soft tissues together with gentle handling of the skin and subcutaneous layers and meticulous suture. Blood pressure may be labile and the patient's vital capacity may be reduced. Operation therefore is not to be undertaken lightly, but if capably performed on a precise indication it is rewarding (Figs. 12 to 14).

The timing of operation is important. Removal of bone too early may result in further ossification, haematoma formation, and the recurrence of ankylosis. If operation is too late the new bone may be extremely hard and envelop important structures such as the femoral vessels. We believe that the best time for operation is when the palpable mass ceases to enlarge on clinical and radiological examination, and when increased definition of the edge of the new bone is seen in serial radiographs. This usually takes between three and four months from the time of the first appearance of the thickening.

It is important to make an incision large enough to give good access to the ectopic bone. We have used the Smith-Petersen hip incision which sometimes needs to be extended. Oedema

of the soft tissues has been observed and muscles unaffected by the ossifying process may be unduly vascular.

On exploration of the affected areas we have found the ectopic bone to be curiously circumscribed and limited to certain muscles and muscle groups. The sartorius or rectus femoris muscles may be unaffected whereas the neighbouring muscles are ossified, and we have seen a completely unaffected psoas major remaining in a groove on the medial side of an ossified iliacus muscle. Similar observations have kindly been made available to us by Mr J. D. Wilson and Mr O. J. Vaughan-Jackson. This tendency to affect muscles and only later to traverse tissue planes makes removal less difficult than expected. Although the femoral and profunda vessels and the femoral nerve may appear to be enveloped in new bone, they can usually be dissected free. It is unnecessary to remove the whole mass, and indeed this may be impossible. It is sufficient only to remove enough of the bone to allow flexion of the hip to a right angle. In our patients the central part of the mass of bone was removed as a wedge, leaving the upper and lower ends attached to the ilium and the femur respectively. The capsule of the hip joint and the joint itself were judged to be normal and in no way involved in the process.

Bleeding has not been excessive but the removal of a large block of bone, which may measure as much as five inches by five inches by three inches leaves a large dead space which has to be drained by closed suction. Antibiotic cover has been employed and although wound healing has been delayed no serious sepsis has been encountered. Ossification in any residual haematoma has been slight and insufficient to interfere with a useful range of passive hip movement (Figs. 12 to 14).

Our experience of operation has been satisfactory. All the patients have gained increased functional abilities of practical value.

SUMMARY

1. Ectopic ossification is commonest in, but not confined to, traumatic paraplegia. It occurs also in many other neurological disorders which have in common a gross disturbance of spinal cord reflex activity. It is a true ossification and must be distinguished from calcification.
2. The neurological lesion may lie anywhere from the cerebral cortex to the mixed peripheral nerve. It may involve motor tracts, sensory tracts or a mixture of both.
3. The ossification is localised and self-limiting. It occurs mainly in the lower limbs and is restricted to certain muscles or muscle groups, the nerve supply of which is always below the level of the central neurological lesion.
4. The blood chemistry is usually normal.
5. A true arthropathy is rare except as part of a secondary suppurative arthritis.
6. The resemblance to myositis ossificans progressiva or to ossifying haematoma is only superficial, although the pathological process at cellular level may be the same.
7. The period of onset after paraplegia is variable. The earliest recorded example is in one of our own cases in which ossification occurred nineteen days after injury. Other patients have developed ossification after several years.
8. The condition is commonest in acquired nervous disease rather than in congenital disorders, and so far as we know it has not been described in the myopathies. The presence of muscular spasticity or flaccidity is relevant only in that it indicates a disturbance of reflex activity.
9. Soft-tissue ulceration appears to be frequently associated with ectopic ossification. The type of new bone formation associated with large chronic ulcers is not to be compared with the new bone formation in the muscles of a paraplegic patient in otherwise good general condition.
10. The occurrence of urinary tract infections with calculi and generalised sepsis is not specifically related to the onset of new bone formation.
11. Localised soft-tissue oedema often precedes the formation of new bone. Its appearance is undoubtedly important, but the mechanism of its origin is obscure.

12. It is not yet known what initiates ectopic ossification, what limits its spread and what finally causes it to stop.
13. We have described 100 examples of ectopic ossification in 603 paraplegic patients.
14. Surgery has been required in only eight patients. The only indication for surgery is bony ankylosis of the hip in an unacceptable position.

REFERENCES

- ABRAMSON, A. S. (1948): Bone Disturbances in Injuries to the Spinal Cord and Cauda Equina (Paraplegia). *Journal of Bone and Joint Surgery*, **30-A**, 982.
- ABRAMSON, D. J., and KAMBERG, S. (1949): Spondylitis, Pathological Ossification, and Calcification Associated with Spinal-Cord Injury. *Journal of Bone and Joint Surgery*, **31-A**, 275.
- ARMSTRONG-RESSY, C. T., WEISS, A. A., and EBEL, A. (1959): Results of Surgical Treatment of Extrasosseous Ossification in Paraplegia. *New York State Journal of Medicine*, **59**, 2,548.
- BARR, D. P. (1932): Pathological Calcification. *Physiological Reviews*, **12**, 593.
- BENASSY, J. (1957): Ostéomes des Paraplégiques. *Revue du Rhumatisme et des Maladies Ostéo-articulaires*, **24**, 457.
- BRIDGES, J. B., and PRITCHARD, J. J. (1958): Bone and Cartilage Induction in the Rabbit. *Journal of Anatomy* (London), **92**, 28.
- CEILLIER, A. (1920): Para-ostéo-arthropathies des paraplégiques par lésion de la moelle épinière et de la queue de cheval. *Thèse de Paris*. Paris: Imprimerie Générale Lahure.
- COSTE, F., GALMICHE, P., and BRION, S. (1950): A propos d'un cas curieux de myosite ossifiante bloquant les hanches; réflexions sur les ossifications ectopiques. *Revue du Rhumatisme et des Maladies Ostéo-articulaires*, **17**, 180.
- CURRAN, R. C., and COLLINS, D. H. (1957): Mucopolysaccharides in Fields of Intramembranous Ossification in Man. *Journal of Pathology and Bacteriology*, **74**, 207.
- DAMANSKI, M. (1961): Heterotopic Ossification in Paraplegia. A Clinical Study. *Journal of Bone and Joint Surgery*, **43-B**, 286.
- DEJERINE, Mme, and CEILLIER, A. (1918a): Trois cas d'ostéomes-ossifications périostés juxta-musculaires et intrafasciculaires, chez des paraplégiques par lésion traumatique de la moelle épinière. *Revue Neurologique*, **25**, 159.
- DEJERINE, Mme, and CEILLIER, A. (1918b): Neoformations osseuses para-articulaires et parostales chez un paraplégique par lésion haute de la queue de cheval. *Revue Neurologique*, **25**, 207.
- DEJERINE, Mme, and CEILLIER, A. (1918c): Para-arthropathies du genou chez les paraplégiques. *Revue Neurologique*, **25ii**, 348.
- DEJERINE, Mme, and CEILLIER, A. (1919): Para-ostéo-arthropathies des paraplégiques par lésions médullaires: Étude clinique et radiographique. *Annales de Médecine*, **5**, 497.
- DEJERINE, Mme, CEILLIER, A., and DEJERINE, Y. (1919): Para-ostéo-arthropathies des paraplégiques par lésion médullaire: Étude anatomique et histologique. *Revue Neurologique*, **26**, 399.
- EICHHORST, H. (1895): Ueber die Beziehungen zwischen Myositis ossificans und Rückenmarkskrankheiten. *Virchows Archiv*, **139**, 193.
- HASS, G. M. (1956): Pathological Calcification. In *The Biochemistry and Physiology of Bone*, pp. 767-810. New York: Academic Press, Inc.
- HEILBRUN, N., and KUHN, W. G., Jun. (1947): Erosive Bone Lesions and Soft-tissue Ossifications Associated with Spinal Cord Injuries (Paraplegia). *Radiology*, **48**, 579.
- IRVING, J., and LE BRUN, H. (1954): Myositis Ossificans in Hemiplegia. *Journal of Bone and Joint Surgery*, **36-B**, 440.
- JOHNSON, J. T. H. (1957): Atypical Myositis Ossificans. *Journal of Bone and Joint Surgery*, **39-A**, 189.
- KÖNIG, F. (1906): Ueber traumatische Osteome, frakturlose Callusgeschwülste. *Archiv für Klinische Chirurgie*, **80**, 64.
- KÜTTNER, H. (1908): Beiträge zur Chirurgie des Gehirns und Rückenmarks. 2. *Berliner Klinische Wochenschrift*, **45**, 654.
- LIBERSON, M. (1953): Soft Tissue Calcifications in Cord Lesions. *Journal of the American Medical Association*, **152**, 1,010.
- LORBER, J. (1953): Ectopic Ossification in Tuberculous Meningitis. *Archives of Disease in Childhood*, **28**, 98.
- MCLEAN, F. C., and URIST, M. R. (1955): *Bone: an Introduction to the Study of Skeletal Tissue*. Chicago: University of Chicago Press.
- MCNEUR, J. C. (1954): Calcification Round the Hips in Paraplegia (Three Cases). *Proceedings of the Royal Society of Medicine* (Section of Orthopaedics), **47**, 14.

- MILLER, L. F., and O'NEILL, C. J. (1949): Myositis Ossificans in Paraplegics. *Journal of Bone and Joint Surgery*, **31-A**, 283.
- MULLIGAN, R. M. (1947): Metastatic Calcification. *Archives of Pathology*, **43**, 177.
- PÖTZL, O. (1908): Demonstration. Verein für Psychiatrie und Neurologie in Wien. Sitzung vom 10 März. *Wiener Klinische Wochenschrift*, **21**, 999.
- RIEDEL, B. (1883): Demonstration eines durch achttägiges Umhergehen total destruierten Kniegelenkes von einem Patienten mit Stichverletzung des Rückens. *Verhandlungen der Deutschen Gesellschaft für Chirurgie*, **12**, 93.
- ROCHE, M. B., and JOSTES, F. A. (1948): Ectopic Bone Deposits: a Paraplegic Complication. *American Journal of Surgery*, **75**, 633.
- SCHLESINGER, H. (1917): Beiträge zur Kenntnis der neurotischen Myositis ossificans circumscripta. *Jahresbericht auf dem Gebiete der Neurologie und Psychiatrie*, **20**, xxiv.
- SOULE, A. B., Jun. (1945): Neurogenic Ossifying Fibromyopathies: a Preliminary Report. *Journal of Neurosurgery*, **2**, 485.
- SOULE, A. B., Jun., and STIFF, D. W. S. (1949): Changes in the Bones and Soft Tissues in Paraplegia. *Bulletin of the U.S. Army Medical Department*, **9**, 1,018.
- SPIELE, W. (1959): Heterotopie beenvorming na mazelen-encephalomyelitis. *Nederlands Tijdschrift voor Geneeskunde*, **103i**, 1,037.
- STANGER, J. K. (1947): Fracture-Dislocation of the Thoracolumbar Spine. With Special Reference to Reduction by Open and Closed Operations. *Journal of Bone and Joint Surgery*, **29**, 107.
- STEINERT, H., and VERSÉ (1910): Myopathologische Beiträge 2. Dystrophia musculorum progressiva retrahens. Kasuistisches und Kritisches zur Lehre von den hereditären Krankheiten. *Mitteilungen aus den Grenzgebieten der Medizin und Chirurgie*, **21**, 105.
- STOREY, G., and TEGNER, W. S. (1955): Paraplegic Para-articular Calcification. *Annals of the Rheumatic Diseases*, **14**, 176.
- STREET, D. M. (1958): Para Articular Bone Formation. *Proceedings of Seventh Annual Clinical Paraplegia Conference*, p. 31. United States Veterans Administration.