Acta orthop. scand. 45, 883–897, 1974

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FRACTURES IN CHILDREN WITH MYELOMENINGOCELE

Report of 15 Cases and a Review of the Literature

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Accepted 21.v.74

In recent years orthopaedic surgeons have seen increasing numbers of children with paralysed lower limbs due to spinal dysraphism. The problem is not new, but the advances of neurosurgery and urology have given a much better prospect of life to these children. Therefore, the correction of the limb deformities is a challenge that has to be accepted in order to help them to stand and walk.

However, parallel with the better general outlook and the improved management of the lower limb deformities in these children, an increasing number of fractures has occurred and some of the fractures show an unusual pattern of repair.

This paper reports a study of 130 children with myelomeningocele, fifteen of whom sustained fifty-five fractures.

REVIEW OF THE LITERATURE

Gillies & Hartung (1938) were the first to describe undisplaced fractures of the upper metaphysis of the tibia in children with myelomeningocele. The fractures in the two cases they described healed with excessive callus formation; each involved some diagnostic difficulty demanding in one case bone biopsy to exclude sarcoma and, in the other, blood investigations to exclude syphilis.

Alliaume (1950) reported two cases with myelomeningocele of the lumbosacral region. In both cases there were diaphyseal fractures of the femora: the second case showed excessive callus formation.

Katz (1953) described a patient with myelomeningocele who sustained four fractures of the same leg; there was no apparent trauma

and they healed with "excellent callus formation". He stated that spontaneous fractures do not occur in the paralysed lower limbs of children who have previously suffered from poliomyelitis.

Jeannopoulos (1954) described bone changes in six children with lower limb mono- or paraplegia. He postulated the existence of trophic nerves to bone, seeking support in the histological findings of de Castro (1925) and Hurrell (1938) each of whom had shown nerve fibres in close contact with osteoblasts.

Carr (1956) found among 100 cases of spina bifida four that had sustained fractures. Two of these formed excessive callus, and one sustained so many fractures that the limb was eventually amputated. Histologic examination of the callus from the last case did not show the characteristic "chondroid tissue", which Fairbank & Baker (1949) observed in the hyperplastic callus of osteogenesis imperfecta.

Golding (1960) showed and commented on the radiological appearances of two cases with involvement of the metaphyseal region of the knee and of the distal tibial metaphysis. The changes regressed with conservative treatment and the lesions "may resemble sarcomata or Charcot's arthropathy".

In the same year Exner reported two examples of excessive calcification in limbs of a patient with myelodysplasia, perhaps due to spontaneous fractures (Exner 1960). Oehme (1961) described two cases with metaphyseal changes and a periosteal reaction similar to the lesions of scurvy, but the ascorbic acid excreted in the urine in the 24 hours hours after a massive dose was normal.

Soutter (1962) described lesions of the metaphyseal and epiphyseal regions of the knee and ankle, and pointed out the diagnostic difficulties with osteomyelitis and bone sarcoma.

Komprda (1964) described a case with thickened sclerosed and fragmented new bone formation.

Thompson et al. (1964) reported three children, out of twenty-two with myelomeningocele, with fractures of the lower femoral metaphysis, and one with a fracture-separation of the lower femoral epiphysis. All presented excessive callus formation. He pointed out that cases of this type could be confused with the "battered child syndrome".

Gyepes et al. (1965) described seven children with changes in the metaphysio-epiphyseal region of the lower femora and tibiae.

Robin (1965) described four cases that sustained fractures of the lower limbs at different levels. He pointed out that not all were spontaneous fractures.

PATIENTS

In the last fifteen years 130 patients with myelomeningocele, suffering from partial or total paralysis of the lower limbs, have received orthopaedic treatment in this and the associated hospital. Among these patients there are 15 who sustained a total of 55 fractures. Only one was born before 1960.

The degree of callus formation was assessed by comparing with normal children and grouping the results as more, equal or less.

Clinical details of the patients sustaining fractures

Incidence. The percentage of children with myelomeningocele sustaining fractures is approximately 11.5. This is clearly higher than in normal children of the same age group. The number of fractures that occurred in each child is seen in Table 1.

Age. Twenty-six per cent of the fractures occurred in the 4 to 5 year-old group, and 20 per cent in the 2 to 3 year-old group.

Table 1.

No. of fractures	No. of children
1	3
2	3
3	4
4	1
5	1
6	1
7	1
12	1

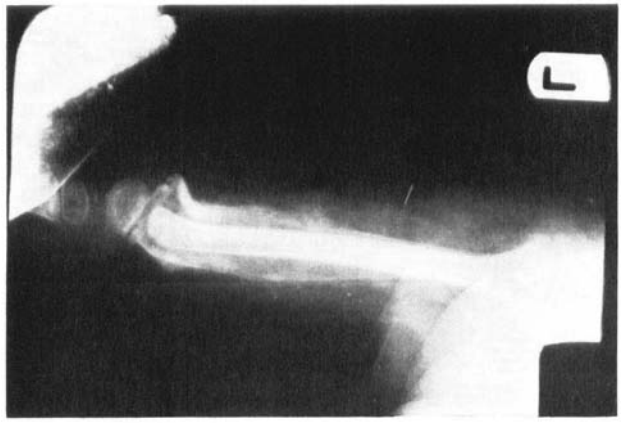

Figure 1. Fracture separation of the left lower femoral epiphysis with large periosteal reaction. Lateral view.

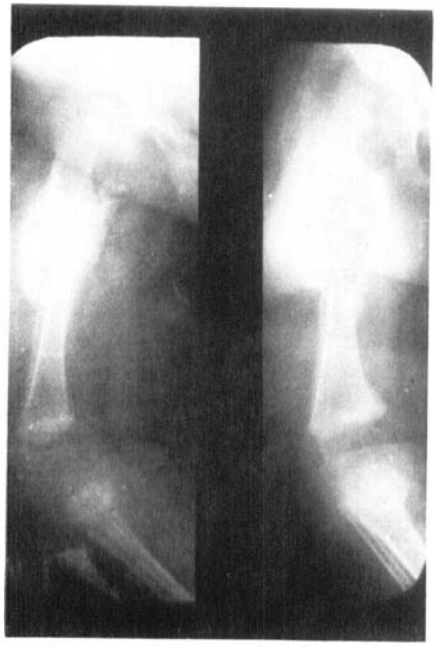

Figure 2. Showing the large amount of callus formation and the patchy, irregular appearance ("lumpy or woolly") together with the extensive periosteal reaction. No comment is made on the suspicious appearance of the upper tibial metaphysis.

Bone fractured. Sixty-two per cent of the fractures occurred near the knee joint, an uncommon site for fracture in normal children (Blount 1955). The greatest number of fractures occurred at the junction of the middle and lower third (24 per cent).

Callus formation. Nineteen of the fractures healed with a large amount of callus formation (Figure 1), 23 with a moderate amount and 10 with a minimal amount. In some instances (Figure 2) the callus had an irregular radiographic appearance giving a "lumpy" or "woolly" picture. Similar changes were observed by Gillies & Hartung (1938) in the form of lack of trabeculation.

Level of paralysis. Fifty-one per cent of the fractures occurred in children paralysed below L-2—L-3 levels. It is noticeable also that 16 out of the 19 fractures that healed with excessive callus formation belonged to this group.

Causes of the fractures. The causes of the fractures are shown in Table 2.

Table 2. Relationship of the skeletal findings to injury, surgical intervention, plaster immobilization and passive mobilization.

Fractures caused by passive mobilization or manipulation of stiff joints	8	30
Fractures caused in theatre (operation, plaster wedging, etc)	10	
Fractures occurring after removal of plaster casts	12	
Fractures due to a fall	4	
Fractures occurring when walking or in bed without apparent cause	16	21
Fractures without known cause	5	

It can be seen from the table that 30 fractures (55 per cent) have an association with treatment received in the way of physiotherapy, surgical intervention, and plaster immobilization; 21 fractures (38 per cent) occurred without any apparent reason ("spontaneously"). Occasionally it has been reported by the nursing staff that the legs of these patients have been trapped between the side-bars of the cot.

Fracture-separation of the epiphysis. This lesion occurred in three cases (Figures 2 and 3). In one there was radiological evidence of closure of the epiphyseal cartilage (Figure 4).

Unusual presentation. In two cases the presenting symptoms of the fracture were raised temperature and malaise. In one an operation was performed to exclude osteomyelitis.

Vitamin C excretion

Vitamin C saturation tests were performed in seven of the patients studied and in three other children being treated for other conditions.

Test procedure. Three hours after a meal, 15 mg per kg/body weight of vitamin C is administered orally. The urine passed in the first four hours is discarded and the urine passed during the next two hours is collected in a bottle with 10 ml of glacial acetic acid. Quantities of vitamin C lower than 15 mg are considered abnormal.

Great difficulty was experienced in collecting some of the specimens from the children with myelomeningocele, due to incontinence and leakage of the collecting bags.

Table 3.

	Case No.	Amount of vitamin C excreted
Myelomeningocele patients	2	< 1 mg
	3	2.5 mg
	4	1.5 mg 5.6 mg
	6	< 1 mg
	8	< 1 mg
	10	< 1 mg
	13	18.5 mg 3.8 mg
Other patients*	1	< 1 mg
	2	4.2 mg
	3	7.1 mg

* 1) Boy, 3 years old. Mongoloid. Fracture of the right femur four weeks prior to test.
2) Girl, four years old. Fracture of the left femur, three weeks prior to test.
3) Boy, three years old. Congenital talipes equinovarus, surgical correction of the foot one week prior to test.

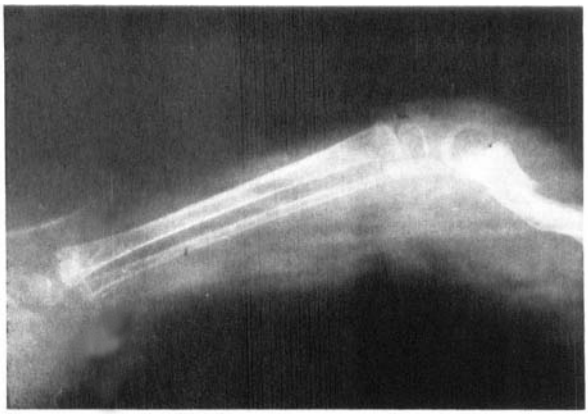

Figure 3. Shows a fracture of the lower femoral metaphysis and a fracture-separation of the upper tibial epiphysis.

Four of the myelomeningocele patients with low vitamin C excretion had their wrists examined radiographically but showed no signs of ascorbic changes.

DISCUSSION

Surely the most striking thing is the high incidence of fractures. The fact that 38 per cent occurred spontaneously and 55 per cent occurred in association with treatment received indicates that there is a strong predisposition to sustain fractures.

The cause of the fractures

Neither the number nor the type of fractures is peculiar to children with myelomeningocele; it also occurs in children with paraplegia from other causes such as subdural haematoma (Caffey 1946), fractures of spine (Katz 1953, Robin 1965), avulsion of lumbosacral roots, transverse myelitis (Jeannopoulos 1954), and tumours of the cord (Robin 1965). However, in children with atrophic limbs due to poliomyelitis, in which sensation is intact, spontaneous fractures are rarely found (Katz 1953, Jeannopoulos 1954).

Absence of active movements in the limbs, due to paralysis, produces bone atrophy*, and the prevention of passive movements caused by brace treatment, enhances it still further. This could explain the 55 per cent of "per-therapeutic" fractures that occurred in the patients under study. However, fractures occur in atrophic bone much more

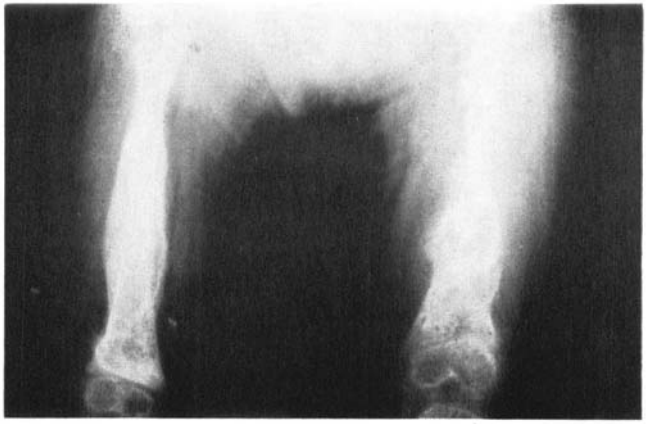

Figure 4. Multiple fractures: Right and left shafts of femora and right lower femoral metaphysis. Notice left femoral epiphyseal line is closed.

frequently when it is deprived of sensation. This may be because strains applied to the legs are not checked by these patients with absence of normal sensation.

In conclusion then, the predisposition to fractures in these children seems to be due:

a) to atrophy of the bones of the lower limbs; or
b) to sensory deprivation of the limbs.

The cause of bone atrophy

Is the atrophy of the bones of the lower limbs of these children due to disuse, i.e. to lack of stimuli provided by weightbearing and by muscle action; or alternatively are "trophic impulses" necessary for proper bone growth and maintenance?

Each of these possibilities will be discussed in turn:

1) *Disuse bone atrophy:* Nothing is known of the precise nature of the bone atrophy in these children, but in experimentally denervated limbs it has been found that the cause of the tissue atrophy is an

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* We have adopted the terminology used by McLean & Urist (1961): "In our opinion atrophy is the term that most nearly describes the condition characterized by deficient formation of new matrix, and we have used this as a general designation."
"The use of the term osteoporosis usually refers to a systemic condition in contrast to the local atrophy of disuse."

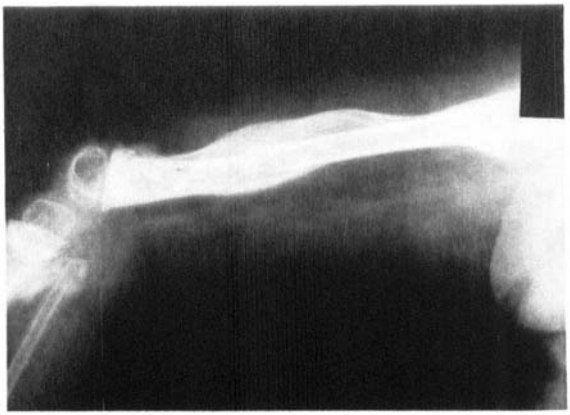

Figure 5. Lateral view of Figure 4 showing the fracture of the upper tibial metaphysis.

increase in the rate of resorption in the presence of normal or subnormal formation, both in bone (Landry & Fleisch 1964) and in muscle and bone (Slack 1954). Geiser & Trueta (1958) arrived at the same conclusions in bone atrophy caused by section of the tendo calcaneus in the rabbit.

Heaney (1962) studying the bone atrophy of patients who suffered from poliomyelitis also stressed the increased bone resorption and bone formation in the early stages and the lower turnover at later stages when the bone atrophy did not increase. Jowsey et al. (1965) studying "senile osteoporosis" reached the same conclusion. All these views are opposed to the classical belief that bone atrophy is caused by diminished bone formation only (Albright & Reifenstein 1948).

In disuse atrophy an increased excretion of calcium has been reported. This gives the patients a calcium balance on the negative side (McLean & Urist 1961, Rose 1966).

2) *"Trophic bone atrophy":* The view that bone maintenance could be due to trophic impulses to the osteoblasts has had its adherents (de Castro 1930, Hurrell 1938, Jeannopoulos 1954). But Eloesser (1917) did not find any morphological changes in the ribs of cats deprived of their posterior root innervation. He reported no physico-chemical changes in deafferented limb bones, although the joints of such cats showed traumatic changes resembling arthritis. Corbin & Hinsey (1939) found that cats whose hind limb had been deafferented and sympathectomized for as long as three years developed neither macroscopic nor microscopic changes in their bones. If the animals were kept

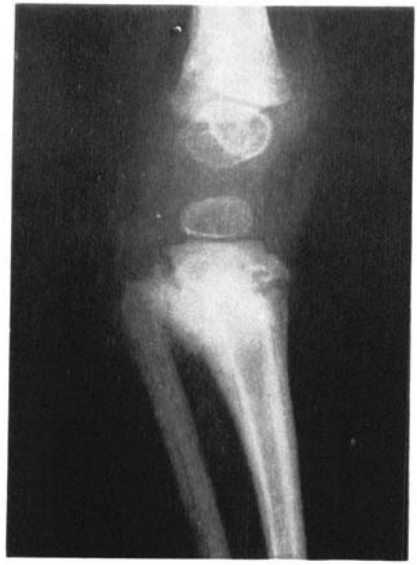

Figure 6. Two months after a fracture of the upper tibial metaphysis. Continued walking. Notice the irregular callus formation.

in small cages, no joint changes were observed, but if their mobility was unrestricted they developed arthritis of the hip joint. Corbin & Hinsey concluded, therefore, that bones and joints are not supplied with nerves with a specific trophic function: "Deafferentation results in a loss of the protection afforded by the propioceptive mechanism and, therefore, may lead to attrition arthritides in those joints subjected to constant trauma".

Gillespie (1954) denervated kittens' limbs and concluded that the bone changes in weight and strength which follow paralysis are due to the secondary loss of muscular activity. No evidence could be obtained that nerves exert any specific trophic influence on bone. This view has been supported by Ring (1961), and by Kharmosh & Saville (1965).

The pattern of healing

The amount of callus formed round the fractures in the present study has been shown to be very variable; some cases formed a minimal amount of callus while others showed exuberant callus formation (Figures 1, 4 and 5). In normal children callus formation is sometimes very marked (Aegerter & Kirkpatrick 1963).

The reason why a third of the fractures in these children show exuberant callus is unknown, but it may be an expression of a high

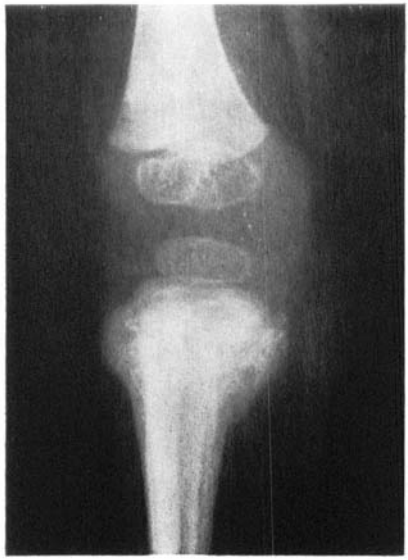

Figure 7. The same as above, oblique view.

level of "metabolic flux" in the bone at the time of the fracture or it may be that such fractures are difficult to immobilize due to the lack of sensation.

The exuberant callus formation of some of the fractures described in the present work is not peculiar to paraplegic children; it has also been reported in patients with osteogenesis imperfecta (Fairbank & Baker 1948, Strach 1953, Schwarz 1961). But in osteogenesis imperfecta the maximum amount of callus occurs two to six months after the fracture. In contrast, the maximum amount of callus formed in fractures in children with myelomeningocele is reached within a few weeks. Some of the cases of osteogenesis imperfecta with exuberant callus formation are accompanied by pyrexia as in two of the present cases.

The metaphyseo-epiphyseal lesions (Figures 6 and 7) have the radiographic appearance of a neurotrophic joint (Brailsford 1948). Eichenholtz (1966) reports that the changes present in paraplegic joints are not of the same nature as Charcot's joints.

Regarding the type of callus in these fractures, Eichenholtz (1963) says: "It seems never to progress to mature compact bone, but instead seems to hover in limbo between the coarse fibrous bone of recent osteoid deposition and adult cortical bone". I cannot support this statement because microscopically the callus removed from one of the cases has a normal appearance.

Experimental work concerning the healing of fractures in denervated limbs is scanty and confusing (Smith & Dunsford 1955, Hulth & Olerud 1965, Reyes-Cunningham et al. 1971). Our own experimental material shows that the hyperplastic callus depends on the mobility of the fracture, the type of bone involved and the period of bone atrophy (Navarro & Peiró 1974, Navarro et al. in preparation).

Does hypo-vitaminosis Ċ contribute?

The possibility exists that the exuberant callus is an expression of a subclinical deficiency of vitamin C, in view of the low excretion of this vitamin in seven of the patients studied. Against this are the following facts:

a) In some cases of exuberant callus in children with myelomeningocele, normal levels of vitamin C have been found in blood and urine (Oehme 1961, Thompson et al. 1964).
b) Not all the fractures in the seven children studied healed with excessive callus formation.
c) Three other children who underwent the test also showed low excretion (see Table 3).

Experimentally it has been shown that fracture healing in vitamin C deficient animals is low and accompanied by severely altered callus formation (Strauch & Geiler 1963). In chronic scurvy the osteoblastic activity is markedly suppressed (Banks 1943, Silvermann 1953).

Two further facts suggest that the exuberant callus of these fractures is not due to a deficiency of vitamin C, viz:

a) The rapidity of union of the fractures in these children in contrast to the slow union of fractures in scurvy, and
b) There is no radiological evidence of scurvy in the metaphyseo-epiphyseal areas.

Suggestions for prophylaxis

It seems very likely that insensitive limbs are easy to strain and damage between the side-bars of the cots used in children's wards in this country. It would be better to use transparent plastic planks for the sides of the cots for these children. As an additional aid to prophylaxis, it has been suggested that the strength of the bones in these children might be greater if splints were used instead of plaster (Strach 1967).

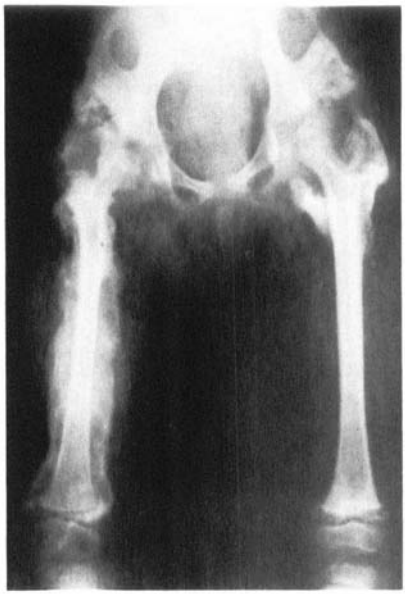

Figure 8. Shows extensive new bone formation around the operated hips, but especially surrounding the right femoral shaft; the epiphysis is slightly displaced and all the callus seems to come from the physeal region.
On the left knee the metaphysis shows some sclerosis and fragmentation but without callus formation.

CONCLUSIONS

1) Among 130 cases of children with myelomeningocele, 15 sustained 55 fractures.
2) More than half (55 per cent) of the fractures occurred pertherapeutically having a clear association with manipulation and immobilization in plaster, which leads to further rarefaction.
3) Sixty per cent of the fractures occurred "spontaneously"; in most cases the patients were in bed and their legs being trapped in the side-bars of the cot was suspected as the cause.
4) Sixty-two per cent of the fractures occurred near the knee joint.
5) The cause of the bone rarefaction and of the fracture is discussed.
6) Pyrexia and malaise can be the presenting signs of a fracture.
7) Only 36.5 per cent of the fractures healed with excessive callus formation. The reason for this exuberant callus is unknown but it may be an expression of the state of the metabolic flux in the bone at the time of the fracture or of excessive movement in the absence of sensation.
8) An excessive amount of heterotopic bone occasionally forms after operations on the hips of these children (Figure 8).
9) The clinical and radiological picture of a fracture in these children can be confused with osteomyelitis, bone sarcoma, bone syphilis,

bone lesions of scurvy, neurotrophic joints and the "battered child syndrome".

10) As prophylaxis against fractures, the following measures are suggested:
   a) The use of beds in which the side-bars are blocked by transparent plastic planks, and
   b) The use of splints rather than plaster for immobilization wherever this is possible.

SUMMARY

The fractures suffered by 130 children affected with myelomeningocele are studied.

The relation to the level of neurological involvement, site of the fracture, age of the patients, treatment received and the possible deficiency of vitamin C is examined.

The healing process in denervated limbs is discussed, and prophylactic conclusions are drawn.

ACKNOWLEDGEMENTS

I am indebted to Mr. G. R. Burwell, Mr. R. Owen and Mr. N. Nisbet for help and advice. Also to Miss Catto for the histological study of one of the cases.

I am grateful for the secretarial and photographic help from the hospital.

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