FRACTURES OF THE SHAFT OF THE FEMUR IN CHILDREN AND STUDY OF SPONTANEOUS EQUALISATION IN LENGTH OF BOTH FEMORAE AFTER TRAUMATIC SHORTENING IN ONE SIDE

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THESIS

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C O N T E N T S

INTRODUCTION

Fracture femur in children is increasing due to the modern traffic life and the increased activity of the children. Femoral shaft fractures in children occupy about 10 % of the beds of any traumatology ward. Inspite of the cumulative studies on femoral shaft fractures, still there is no definite line of treatment. The management fluctuates between skin traction in Thomas or Brynts splints; skeletal traction by Kirschner wires and rarely operative reduction is indicated.

Plaster spica was not much attractive in the treatment of femoral shaft fractures on the assumption that shortening and overlap at the fracture site may result; in the absence of continuous traction.

The question which often arises in managing femoral shaft fractures in children, especially those having residual uncorrected shortening, is whether to attempt anatomical correction, inspite of exhausting the child with repeated manipulations and even open reduction, or to leave moderate degrees of uncorrected shortening for the possibly of overgrowth and spontaneous correction, provided the child is in the growing period.

In this work the problem of [illegible]
[illegible] studies of children after [illegible]
the cause of the growth acceleration was also investigated.
This was done by :

1- Clinical and radiological follow up of 28 cases of femoral shaft fractures in children for periods varying from 6 months to 28 months.

2- Experimental surgery : Considerably high percentages of shortening were produced in the femorae of immature rabbits and the maximum amount of shortening that can be compensated was calculated.

The cause of growth acceleration after femoral shaft fractures was also investigated by :

1- Histological and angiographic study : The changes in the epiphyseal growth plates of the fractured and normal sides were studied at different periods after the fracture.

2- Isotope study : Radio-active phosphorous p^{32} was used to study the blood flow in the metaphyseal-epiphyseal areas of both the normal and the fractured sides. The macroautoradiograms of both are developed for comparison of the uptake.

REVIEW OF LITERATURE

Langenbeck (1869) mentioned that diseases which cause irritation and hyperaemia of bones also increase their length and thickness.

Ollier (1921) stated that irritation of the shaft of a long bone causes increase in the rate of growth. The injection of oil of turpentine in the bone marrow or in the knee joint resulted in growth acceleration of approximately 1 %.

Ferguson (1933) reported that the rate of longitudinal growth could be increased by direct trauma to the bone. He postulated that the increased rate of bone growth that frequently occurs after osteotomies or fractures is the result of obliteration of the medullary blood flow to the metaphysis without interruption of the periosteal vessels to the ends of the shaft.

Bisguard (1936) used the tibiae of immature goats. He studied the effect of fracture, bone grafting, and experimental shortening on the rate of growth. He found that (1) lengthening, in excess of normal, occurred in the growing tibiae following fractures and the greatest

[illegible] to the fourth month with an initial period of shortening in the first month. (2) The excess rate was gained at the ends of the shaft through an acceleration of the rate of growth in the epiphysis. (3) The increased rate of growth in the fractured bones occurred irrespective of the length of the bone or of the necessity for equalisation provided intact epiphysis were present. (4) The tibiae in which the epiphysis were destroyed showed no growth proving that the process is not simple compensation from the body for the shortened limb. (5) The increased rate can not be a hormonal stimulation because the other control limb has not shown any excess in the rate of growth.

He concluded that the fracture and its reparative processes produce a local prolonged hyperaemia in the affected bone which is reflected on the growing ends causing growth acceleration.

Miltner (1937) stated that stripping the periostium alone in rabbits femurae produced definite bone lengthening of 3 mm over a period of three months. He repeated periosteal stripping more than one time on the same rabbits but he failed to get more growth.

Jansen (1937) found that simple obliteration of the nutrient canal did not increase the length of the femur in growing rabbits. In his experiment, there was minimal stripping of the periostium and no fracture was produced.

Trueta (1959) emphasised that to obtain growth acceleration in a long bone the intermedullary circulation must be so altered that an increased blood flow is produced in about the metaphyseal junctions. He stressed also that overgrowth in osteomyelitis was found chiefly in cases in which the diaphysis was principally involved with interruption of the medullary canal and that overgrowth stopped when the medullary canal and the nutrient artery were again restored. He postulated that growth acceleration in long bones after fracture involved only those in which the nutrient artery might well be damaged. He gave his conclusion that stimulation of bone growth can be produced by redistribution of the intraosseus circulation to the metaphyseal ends.

Lober and Trueta in the contribution to osteogenesis (1960-1961) established the relation between the increased vascularity and osteogenesis. He attributed

[illegible] overgrowth to increased blood flow in the epiphyseal growth plates.

Venous stasis and bone growth :

Venous stasis has been suggested as a stimulus to bone growth and formation. Kelly (1959) observed that an arterio venous fistula increased bone growth.

DEVELOPMENT OF THE FEMUR

The development of the femur is a good example of endochondral ossification. The first sign in its development is condensation of mesenchymal cells to precartilage. The cells of this condensed mass differentiate into chondrocytes elaborating a hyaline ground substance and shaping a crude model of the femur in the form of a short thick cartilagenous shaft towards the end of the second month of intrauterine life.

The first trabeculae of bone are formed in the connective tissue surrounding the middle portion of the shaft at the 8th week. This perichondrol collar encircles and supports the future shaft during the process of resorbtion and destruction of the cartilage cells. The destruction of the chondrocytes is the result of intercellular oedema. The cells enlarge considerably, the stored glycogen disappears and calcification occurs in the matrix. The calcification of the matrix prevents nutrition to the cartilage cells and causes their degeneration. This is immediately followed by invasion of vascular connective tissue from the periostium covering the diaphyseal collar. This vascular channel will form the future neutrient artery.

The fragments of the degenerated cells are removed by macrophages which accompany and sometimes precede the invasion of vascular supply. Although the blood must transport the materials of osteogenesis. The exact role of the blood and blood vessels in resorption of cartilage model is not clear. There may be close relation between the lysosomal enzymes, oxygen tension and carbon dioxide tension.

Duthie (1955-f) found clusters of mast cells around the degenerating cartilage trabeculae and he suggested an interrelation between the presence of mast cells and the process of resorbtion and demolition of the cartilage model. These cells possibly secrete either a heparin-like precursor, hyaluronic acid or histamine, (McManus 1954). Histamine may have an important role in the demolition phase by altering the local vascularity and the migration of cells.

Once the cartilage cells degenerate and blood vessels invasion occurs, osteogenesis starts. The osteogenic cells (osteoblasts) may either develop from the primitive mesenchymal cells or be carried by the invading blood.

Rutishauser (1950) believed that for deposition of calcium phosphorous complexes there should be in the matrix the sulphated mucopolysaccharide chondritin sulphuric acid and alkaline phosphatase. He postulated also that there was a quantitative balance between the amount of chondroitin sulphuric acid disappearing and the amount of phosphate being deposited.

After the central portion of the cartilage model is resorbed an irregular primary marrow cavity is formed and bone formation commences both in the connective tissue which fills the marrow cavity and in the remaining calcified trabeculae.

The primitive marrow cavity is now bound by slender delicate irregular bony trabeculae. Development proceeds proximally and distally. The process of ossification usually lags behind the growth of the cartilage so that when the bony shaft is formed there remains two cartilagenous ends : the upper and lower epiphysis. At the same time opposition of new bone at the periphery of the perichondral collar, contributes to the growth in thickness.

Vessels from the perichondrium with surrounding connective tissue continue in the cartilagenous ends of

the shaft forms the ossification centres. Later on the ossification centre becomes separated from the epiphyseal cartilage by a bony end plate while it is continuous with the metaphyseal end of the diaphysis. The epiphyseal growth plate is thus formed and further growth in length occurs by :

1- Replacement of cartilage trabeculae developed from the epiphyseal growth plate by bony trabeculae which are added to the metaphysis.

2- Replacement of a deep layer of the epiphysis (articular cartilage) by bone and this lengthens the epiphysis and consequently the femur.

The transverse growth is simply the result of continuous opposition at the outer periosteal surface and resorbtion at the inner or medullary surface. This process is governed by : 1) Morphological growth changes.
2) Lines of stresses.

At birth the femur is formed of a bony shaft. The neck is short while the trochanters and the extremities are in cartilage. In the lower end a centre of ossification is present,(having appeared about the 7th month of intrauterine life). Usually more advanced in girls than in boys.

[illegible] the [illegible] greater trochanter at the age of [illegible], for the lesser trochanter at ten or eleven. Fusion of the head epiphysis and the neck occurs about the age of seventeen or eighteen. The trochanteric epiphysis joins the shaft about the same time. The lower epiphysis is the last to join the shaft at the age of eighteen.

Contribution of the Upper and Lower Epiphyseal Growth Plates to Longitudinal Growth :

The obliquity of the neck of the femur makes it obvious that the proximal epiphyseal plate cannot contribute much to overall elongation of the femur as it serves primarily to lengthen the neck.

In the lower end the direction of the columns is simple, and growth takes place directly in the direction of the bone. It is considered by several authors that the lower epiphyseal growth plate contributes three quarter or two thirds of femoral growth, while the upper contributes only one fourth.