OUT OF THE PAST

RICKETS AND OTHER DEFICIENCY DISEASES OF THE OSSEOUS SYSTEM

(*Last Lecture given by Sir Arnold Theiler to B V Sc V Students, Onderstepoort, 1936)

The causes of these diseases may be divided into 3 main groups, namely

1. Malnutrition — protein deficiency
2. Mineral deficiency or abnormalities
3. Vitamin deficiency esp. Vit. D

They will thus be considered accordingly.

**Malnutrition**

In this case it is mainly a protein deficiency or even an amino acid deficiency e.g. the protein of maize is deficient in certain amino acids. In plain words one may say that if the osteoblasts are not "fed" properly they cannot produce bone. Instances of this may be seen in Dr Quin's physiological experiments in connection with the Alimentary System where improper or insufficient absorption takes place due to fistulae, anastomoses and other derangements.

Again, in Mönig's *Oesophagostomum* experiments the same conditions may be seen with heavy worm infestations — in this case probably due to toxin formation, but not impossibly due to digestive and absorptive derangement. Probably in practice the same will be seen in cattle in certain areas of South Africa in winter or during periods of drought.

**Mineral Abnormalities**

1. Phosphorus deficiency
   (a) **Rickets**

   Definition:— A superabundance of osteoid tissue is formed which transgresses the normal limits due to a phosphorus deficiency or perhaps an excess of Ca and normal amount of phosphorus (experiments still in progress), in young animals before the epiphyseal line is closed. Whereas normally the width of the epiphyseal line is 0.1 cm, in rickets it becomes wider, perhaps 1 cm, not due to increased formation of bone but due to decreased absorption.

   Produced in pigs experimentally: Clinically the animal's growth is greatly retarded. The joints are abnormal in shape and size and the animal has pain due to the fact that the newly formed osteoid matrix does not ossify and consequently the nerves are not protected against pressure etc.

   (b) **Osteomalacia** this is the so-called rickets in adult animals especially cattle. The cause, pathology, etc. is exactly the same as in young animals except that it occurs after the epiphyseal line has closed. It is the so-called "stywesiekte" (stiffness) of cattle in South Africa — "True Stywesiekte" (Crotalariosis of cattle is not true stywesiekte — a laminitis) is a phosphorus deficiency in old animals. The condition may be cured by giving the animals phosphorus in the form of bone meal — that is in **cattle and sheep**. In **pigs** — feed a normal ration with correct Ca:P ratio (1:1) — cure. Vitamin deficiency almost impossible to produce in South Africa, due to its abundance of sunlight, except under extremely artificial conditions, where all the food is boiled etc.

   In horses only one case of rickets is on record in a foal. It cannot be produced in the horse, or perhaps only with extreme difficulty.

   NB A Ca deficiency does not produce rickets.

2. **Calcium Deficiency**

   **Osteoporosis** is produced where there is an insufficiency of calcium with a sufficiency of phosphorus.

   **Definition**:— It is a resorptive atrophy of the osseous tissue i.e. the bone deposited is reabsorbed leaving a porous, easily fractured bone, with an absence of osteoid tissue.

   In pigs in this condition one sees a paralysis of the back.

3. **Abnormal Calcium-Phosphorus Ratio**

   If an excess of P over Ca is fed a condition of **Osteodystrophia fibrosa** develops.

   **Definition**:— Excessive breakdown of bone replaced by fibrous bone tissue.

   This condition can be produced in pigs and horses. At one time it was thought to be an infectious disease in horses. Often seen in "old days" in horses in cities and towns (Johannesburg) due to the fact that the animals were fed exclusively on a bran ration which is rich in P but poor in Ca. When fed on hay as well, the condition did not appear.

   It affects the whole skeletal system, but is clinically only seen in those parts where continuous and great mechanical stress (or trauma) is applied such as in the jaw bones. These swell up to a great extent, giving the face a puffed appearance, due to excessive formation of fibrous bone, to combat the continual strain placed upon it. The animals become progressively weaker until they are unable to stand any longer and have to be placed in slings until death supervenes. The condition responds readily to feeding with a normal Ca:P ratio and complete recovery takes place. Whether the swelling of the jaw bones will disappear...
completely, perhaps with contraction or absorption, is not known but indications are present to show that it may perhaps reduce to some extent.

4. **Calcium and Phosphorus Deficiency**

Experiments in pigs show that the animal grows well if both minerals are deficient as long as they are given in a normal ratio.

**Osteodystrophia Fibrosa in Human beings.**

This disease in humans is always associated with a tumour in the parathyroids which causes a derangement in the Ca metabolism etc. This is never seen in animals.

**Vitamin deficiency**

This is commonly known to be the cause of rickets in humans in Europe. The theory is that Vitamin D is necessary to fix the Ca and P in the bones. However, in S.A. with our abundance of sunlight the animal is able to produce its own Vit. D as well as the plants on which such an animal is fed. This is therefore of little practical importance except perhaps in instances where animals are kept under extremely artificial conditions such as where all foods are boiled etc.

In all above-named conditions the skeletal system is weakened to such an extent that deformities and fractures are frequent. In rickets with fractures a superabundance of osteoid tissue is formed — no callus is formed due to P deficiency and the absence of Vit. D.