

cartilage cells, the proliferating zone, and then the bone formation.

(An Epidiascopic demonstration of the microscopical and naked eye appearances of rachitic tissues was then given.)

I have here a rabbit's bone, part of which was placed in hydrochloric acid to remove the lime, so that this end is quite soft. The fact which these specimens illustrate has led to a very curious error in regard to the causation of rickets.

It is perfectly true that in rickets the mineral matter is much less than is normally present in bone, and that has led to the idea that the chief cause of rickets is due to an absence of lime or calcium in the food, so much so that in the case of the Glasgow babies they were supposed to be suffering from rickets because the water from Loch Katrine, which is a very pure water, had not enough lime. Now, as a matter of fact, a deficiency of lime is very seldom found in babies' diet. You who have attended these lectures have heard me dwell upon the serious defects of patent foods, but you have never heard me say they do not contain enough lime—they all contain a large amount of lime. Not only that, but when you dilute ordinary milk with two parts of water, there is as much lime in that mixture as there is in mother's milk of good quality. Consequently we cannot attribute any of the defects as we see them in rickets to a deficiency in the supply of lime; very often the rachitic baby has had much more lime than it could possibly require. So that, though the removal of lime shows how soft the bone becomes, and how useless it would be but for the lime, yet the growth of the bone depends on the production of the material in the shape and character that you see in that bone which is quite independent of the lime. It is the deposition of lime in the perfectly formed structures which makes good bone. If those structures cannot be formed, and lime is deposited anyhow, then you get the rachitic bone. I may remind you that as a result of late rickets, the bone contains more lime than is normally the case, and the bone is much denser, and more brittle.

THE CHIEF CHARACTERISTICS AND RESULTS OF RICKETS.

Let me now summarise some of the chief characteristics and results of rickets. In the first place, it has a most serious influence on the nervous system. Very many infants die from convulsions at about 12 months of age—practically all of these deaths are due to rickets. There is not the smallest doubt that

it is a very common cause in later life of epilepsy, and it is one of the great causes of insanity. The injuries done to the nervous system in rickets are appreciated at the present time by very few, but the more you study infantile conditions, and the more you study the conditions as you see them in lunatic asylums, the more you will realise that the preventable causes of insanity lie chiefly in the disease which we are discussing this afternoon. The whole structure of the nervous system is wrong; the performance of its functions becomes quite unbalanced, and you have sometimes the epileptic condition, and sometimes the condition of insanity.

OTHER RESULTS.

To take you from that to a quite different aspect of affairs, there is very little doubt at the present time that the chief cause of adenoids is rickets; the condition which we know as the scrofulous condition with enlarged glands is one of the common sequelæ; while pulmonary tuberculosis practically would very largely cease to exist if it were not for this disease. If you go to any chest hospital, and put on one side the patients who do not exhibit signs of having suffered from rickets, you will not find many patients left who are there for tuberculosis. The whole life history of pulmonary tuberculosis as it affects the young adult is simply the destruction of the resistance of the body by rickets, assisted by the deformities of the chest which are such a marked feature of an advanced case of rickets. Let me remind you of the spinal deformities which are seen in practically all classes of society as the result of the same disease. Dr. Clement Dukes has stated that of the boys at Rugby School a very large proportion exhibit signs of defects due to infantile malnutrition. Any competent physician can tell you the same thing in reference to the spinal deformities one sees in practice. In lying-in hospitals we see rare forms of deformity of the pelvis which cannot be etiologically accounted for; but if we put all these rare forms together, they would not bear comparison in number with the deformities of the pelvis caused by rickets. And throughout this list, and I might easily add to it, there is nothing surprising, because, as I explained at the outset of my lecture, rickets is a disease of growing structure, and if you have conditions in which such structural defects can manifest themselves, owing to a defective supply of building material, the lesions must be widespread, and as extensive as the organs of the body. Thus we have all these conditions due to rickets.

(To be concluded.)

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