THE ETIOLOGY OF RICKETS.*

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At the present time there are two different schools of thought on the subject of the etiology of rickets—namely, the dietetic and the hygienic, the former considering that a defective diet is the cause of this disease and that lack of fresh air, sunshine, and exercise is only a contributing factor, which increases the severity of the disease but never causes it if the diet is satisfactory. The latter claim that rickets is directly due to a hygienic defect. Many years before vitamins were thought of, Cheadle¹ brought forward many convincing arguments in favour of the former supposition. He pointed out that the disease was very rare in breast-fed children, and that when it did occur it was almost invariably due to prolonged lactation (with deficient secretion of milk) and the addition of starchy foodstuffs as a supplement. He considered that fat insufficiency was the chief cause of rickets, but also recognized the value of suitable protein in the diet as a preventive and the injurious effects caused by an excess of carbohydrates in young infants. He claimed to have cured rickets by merely correcting the diet of infants, and was of the opinion that cod-liver oil, though useful in some cases, was not an essential form of treatment, and that in other cases—for example, those infants who had indigestion—its use was sometimes harmful.

Findlay, in 1906, expressed the opinion that insufficient exercise was the cause of rickets. His experiments with Paton on pups appeared to have strengthened his views, and recently² he has published a small number of cases which are intended to show that greater curative effects are obtained by massage and electricity than by improved dietary and administration of cod-liver oil, etc. Noël Paton and Watson, as a result of their experiments, came to the conclusion that an unclean environment produced rickets in young animals owing to microbic infection. They suggested that a non-specific microbe such as McCarrison has shown to be the cause of goitre was to blame for the disease. Hess,³ by reason of his dietary experiments on negro children, was opposed to the vitamin theory, and noting, as most other observers of the disease have done, that rickets was most active in winter and tended to improve in spring and summer, suggested that lack of sunshine was the cause of rickets. He demonstrated by a further series of experiments that sun baths in spring and ultra-violet rays in winter had a curative effect on negro infants suffering from this disease.

Rickets in Australasia.

Before discussing the various theories the writer would like to give a short description of rickets as it occurs at the Antipodes, as he considers that it serves to throw a certain amount of light on this vexed question. In former times the existence of rickets in Australasia was denied, and its absence was quoted as an argument against the dietetic origin of this disease. Such, however, is far from the truth, and it is now recognized that rickets, although not nearly as prevalent as it is in Europe and America, is by no means uncommon. It is present, however, in a much milder form—marked deformity of bones is extremely rare, and spasmophilic conditions, such as tetany and laryngismus, which are associated with advanced forms of rickets, are conspicuous by their absence. The common symptoms observed in Australia and New Zealand are slight beading of the ribs, delayed dentition and closure of the fontanelle, a protuberant belly, sweating of the head, and restlessness at night. Anaemia and malnutrition (although occasionally the fat form of rickets is seen) are usual. The muscles are flabby and wanting in tone and the ligaments are relaxed. A tendency to the development of catarrhal conditions of the mucous membranes, such as bronchitis and diarrhoea, is not infrequent. These symptoms are practically identical with those described by Cheadle as existing among the better classes of the population in England.

* Some prefatory matter containing a review of recent experimental work by Hopkins, Osborne, and Mendel, Mellanby, and others (1911-1918), and a few other paragraphs have been omitted owing to exigencies of space.

An analysis of forty cases seen in private practice in Auckland may be of some interest:

Age.	Cases.	Percentage.
Under 6 months	1	2.5
6 to 12 months	21	52.5
1 to 2 years	16	40.0
Over 2 years	2	5.0

The cases seen over the age of 2 years were aged 2 years and 3 months and 3 years and 2 months respectively. The latter case presented marked deformities of the bones of the upper and lower limbs and chest wall, and was the only case of such marked deformity in a New Zealand born child that I have ever seen.

Diet.—In every case the diet was more or less unsatisfactory as regards insufficiency of milk and excess of starchy foods. None of the cases were wholly breast-fed, but 3 (20 per cent.) were partly on the breast, supplemented chiefly with starchy foods. It is noteworthy that 5 (62 per cent.) of these partly breast-fed infants were over the age of 1 year. One infant of 9 months of age from a country district developed rickets on a diet of a patent food and condensed milk, but his twin sister, fed on exactly the same lines, showed no sign of rickets.

Season.—Roughly dividing the year in Auckland into five winter months (which is a very liberal allowance) and seven summer months, it was found that 29 (72.5 per cent.) of these cases were brought for advice during the summer months and 11 (27.5 per cent.) during the winter. These figures tend to show that the increased activity of rickets in winter noticed in other countries does not occur in New Zealand. The probable explanation is that, advice being sought for the catarrhal complications of this disease, diarrhoea in this country is a more marked feature than bronchitis, which, as is well known, tends to occur chiefly during the winter season.

Hygiene.—The hygienic condition of all these patients was good.

Symptoms.—No case was included in the above series of cases in which beading of the ribs, delayed dentition and closure of the fontanelle were absent. Marked sweating of the head and restlessness at night occurred in the majority. All the cases, without exception, were below normal weight. Slight deformity of the chest, such as pigeon-breast or the presence of Harrison's sulcus, occurred in a few of the cases, but bending of the long bones of the limbs, with noticeable enlargement of the epiphyses, was only noted in the one case already mentioned. None of the cases showed signs of laryngismus or tetany.

A comparison between rickets as observed in Europe and Australasia would tend to show that the early symptoms of this disease show great similarity, but that the course is much more benign at the Antipodes than in older parts of the world. A little consideration on the subject should clearly demonstrate why such is the case. There is a wide difference between the environment of the child in Australia and New Zealand and that which exists in the slums and tenement houses of the large cities of Europe.

In Glasgow or London, for instance, among the young children of the poor during the long winter months the hygienic conditions of life of the puppies cooped in a laboratory is almost exactly reproduced. Close confinement, with its attendant evils of insufficient fresh air, sunshine, and exercise, naturally tends to intensify the symptoms of a metabolic disorder. For normal growth and development in an infant we know that a well-balanced diet is an essential, but other factors are also necessary. For proper development of muscles exercise is an important feature. The ceaseless activity during waking hours, which is characteristic of all young animals, is probably a necessity for normal development of both muscles and bones. Similarly the functions of the rapidly developing cerebro-spinal and sympathetic nervous systems of the infant would appear to be stimulated, through the eyesight, by an environment which permits of a variety of objects of interest. Contrast the lot of the infant of the slums with that of the child in the same class of society in Australasia. The former for six months of the year is kept for the greater part of the twenty-four hours in a dingy room, in which sunshine, fresh air, and cleanliness are all lacking. Excessive clothing, hampering the free movement of the child, is not uncommonly applied by the parents to maintain warmth, and, owing to the tendency of these unfortunate children to bronchitis, the mother is often afraid to take them into the open for fear they should "catch cold." In Australasia the winters are short and sunshine is abundant, and in consequence infants are enabled to spend a great deal of their existence in the open air, to exercise their muscles freely, and increase their powers of observation. The occurrence of this disease in Australasia would tend, then, to show that absence of exercise and sunshine have no causal relation.

to rickets. The one feature common to all cases seen by the writer in these countries is some dietary defect, and from this he concludes that rickets is a dietetic disease.

The chief difficulty in accepting a purely dietetic theory for the causation of rickets is the following well-known fact. If a number of children are placed under the same hygienic conditions and fed on a similar ill-balanced artificial diet three things may occur:

- (1) The infant may remain quite healthy and develop normally.
- (2) Malnutrition may occur without signs of rickets.
- (3) Rickets may appear.

None of the theories so far advanced explains the markedly variable susceptibility to the development of rickets possessed by infants placed under similar conditions of life as regards hygiene and diet. Speaking of infants reared in an institution, Hess³ recently said the fact that 25 per cent. of the children fed on the same milk would get rickets and the rest would not, showed that there was some individual idiosyncrasy. If we adopt the dietetic theory of rickets we must go further, and be able to explain why a similar diet given to three infants is capable of producing atrophy in one, rickets in another, while a third child who was similarly treated remained in good health.

Cantley⁴ has endeavoured to explain why the atrophic infant shows no sign of rickets by stating that there is antagonism between the two diseases, the atrophic infant not having "sufficient anabolic vitality for the production of the characteristic proliferative changes in the cartilages and bones." Be that as it may, the fact remains that atrophic infants rarely show signs of rickets. In both these affections, which may be presumed to be due to dietetic causes, as human milk is a specific remedy for one and a prophylactic for the other, there are profound metabolic changes, yet the clinical signs are markedly different. In looking for an explanation which will satisfy all the peculiarities of rickets as shown by laboratory experiments and clinical observation, including the results of therapy, it appears to the writer that one must go further than to assign either a purely dietetic or hygienic etiology for this disease. The profound metabolic changes which occur in a severe case of this disease can, he considers, be only caused by a disturbance of those organs which control metabolism in the young. In the further study of secretions produced by the endocrine organs—"the regulators of metabolism," to quote McCarrison⁵—will perhaps be found the key to open the portals which at present hide the mysteries of this disease. If a deficiency of endocrine secretion is the direct result of an unsuitable artificial diet, and the latter is also the chief factor in the causation of rickets, it would perhaps not be illogical to conclude that this disease is the effect of a deficiency of the secretion produced by one or more of these organs. Of the various endocrine organs a deficiency of whose secretion is responsible for the production of rickets I suggest that the thymus gland is the most probable. McCarrison found that in birds and monkeys experimented on with certain diets "the thymus atrophies intensely," but he has no data with regard to the merely scorbutic diet. A plausible reason for considering that rickets is due to thymic insufficiency is that the age incidence of the active growth of the thymus and of the disease known as rickets is almost identical. The normal thymus does not increase in size after the age of 2 years, and rickets as a progressive disease is usually limited to that age. Surely these significant facts are something more than a mere coincidence. Then again the peculiar idiosyncrasy to rickets may better be explained by this theory than any other. Still⁶ has drawn attention to the wide discrepancy between the weight of the normal thymus gland in infants as noted by different observers, and it is not unreasonable to suggest that Nature has been more lavish in her endowment of some infants with a large and active gland than in others. If such should prove to be the case, we can more easily explain those cases (known to all observers) which, in spite of an ill-balanced diet containing (theoretically) insufficient suitable protein or fat and an excessive amount of carbohydrate, can still remain healthy for a considerable time, because, owing to a highly efficient thymus, a sufficiency of secretion is produced to prevent the onset of this disease. In advanced cases of

rickets the spasmophilic manifestations which occur owing to defective hygiene are possibly due to the atrophy of other endocrine organs such as the parathyroids. The condition of marasmus or infantile atrophy, which is said to be antagonistic to rickets, may possibly be due to a deficiency of the secretion of other endocrine organs.

The writer has endeavoured to show that rickets is primarily a dietetic disease, being due to either an insufficiency of fresh animal food in an artificial diet, or to defective assimilation (as a result of digestive disturbance) of such food. It remains only to express an opinion as to which of the two great classes of foodstuffs, fat or protein, are responsible for such defective metabolism. As a result of clinical study and a consideration of the experiments of others I have come to the conclusion that a deficiency of suitable protein is the probable cause. The experiments of Paton and Watson would appear to show that in pups rickets is not caused by feeding on skimmed milk, and though I have frequently fed infants on fresh and dried separated milk for several weeks I have never seen rickets occur as a result. The beneficial effects of cod-liver oil in this disease, both as a prophylactic and a curative measure, would at first sight appear to be a stumbling-block to the acceptance of this theory. I know, however, of no evidence that the benefit derived from this form of treatment is due to the fat of the cod-liver oil, alone or in part; and further investigation may show that the extractives contained therein possibly serve as metabolic stimulants or regulators.

Summary of Conclusions.

1. That rickets is due to a deficiency of fat-soluble A vitamin in the diet has not been proved.
2. It is primarily due to a diet actually deficient in fresh animal food, probably suitable protein, or to a disturbed digestive condition which prevents the assimilation of the same.
3. The striking metabolic changes in rickets are due secondarily to a deficiency of secretion of one or more of the endocrine organs and probably chiefly of the thymus gland.
4. Confinement in young animals, with its attendant evils of lack of sunshine, exercise, and cleanliness, are important factors in increasing the severity of the disease.

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DIAGNOSIS AND TREATMENT OF PERFORATED DUODENAL ULCER.

FOUNDED ON FORTY-ONE CONSECUTIVE CASES.

BY

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On looking over the records of my work in the Aberdeen Royal Infirmary during the years 1914-1919 I was struck by the number of cases of perforated duodenal ulcer that came under my care—namely, 12 in 1915, 9 in 1916, 8 in 1917, 9 in 1918, and 3 in 1919, or 41 in all. As this seemed much in excess of previous years, I thought it might be of interest to record certain points in the clinical notes of the cases. Owing to the greatly reduced staff in the hospital during that time, the only assistant available for me was a senior student who had many duties to perform, hence the notes are not so detailed as I would have liked.

Perforation is the most serious symptom that can occur with duodenal ulcer; it may take place in both acute and chronic ulcer at practically any age. Perforation has been reported in a child of two months and in a woman aged 77 years, but such cases are exceptional. At the same time the fact must be recognized that duodenal ulcer and its complications is now being found at a much earlier age than was at one time thought to be probable. Perforation is not only the most serious but also the most frequent complication, but the percentage of cases in which it is said to occur varies greatly. Osler, in his *Principles and*