OORSPRONKELIJKE BIJDRAGEN

THE EXPERIMENTAL PRODUCTION OF PERFECT AND IMPERFECT TEETH AND THE APPLICATION OF THE RESULTS TO DENTAL DISEASES IN CHILDREN [1])

DOOR

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616.314 : 613.2.

Mr. Chairman, Ladies and Gentlemen; I feel a very proud woman to-day. I am proud to think that you should have invited me to tell you about my work. It is the first time that I have been asked to speak outside the British Isles. Luckily I feel much more at home here than I do in most foreign countries. I have travelled a good deal. I have been in thirteen or fourteen different countries in Europe, and last year we spent a few weeks in Canada and America, but I must admit that I feel more at home here even than I did in these English speaking countries.

Unfortunately I cannot speak your language; I must therefore speak in English. Professor Eykman tells me that we English swallow our words. Should I do this, I hope you will let me know, and I will then try to cough them up again!

My second reason for feeling proud is because Professor Eykman is in the chair to-day. We have a great reverence for Professor Eykman in England. We look upon him as a parent of the vitamin work. There is of course no need for me to say anything here about his important contributions to the study of nutritional diseases as these are so well known. I

[1]) Voordracht met lichtbeelden, gehouden in de vergadering van het Nederlandsch Tandheelkundig Genootschap, 11 October 1925.

feel honoured also to find many other great Dutch scientists here in addition to the members of the Dental Faculty.

Last, but not least, I am very proud to have my husband with me to-day. *(Cheers).* My work, as many of you know, is an offshoot of his work on rickets. He has throughout been my guide, philospher and friend; not only so, but he has provided most of the brains connected with the work. This is the first time I have ever spoken in front of him. *(Laughter).* Previously, I have been very careful to see that he was not present at any meeting at which I was to speak. For this reason I feel perhaps a little more nervous to-day even than usual.

As I have much to say about my work, I will now go straight ahead. I will consider the subject from three points of view. I have written the headings of these divisions on a lantern slide so that, if I swallow my words, you will at least be able to read the subjects I am going to deal with.

The first division is headed:

'The Structure of Teeth in Relation to Caries.'

(By structure I mean the minute detailed structure, the calcification, and not the form or shape of the teeth.)

The second division deals with:

'The Conditions which affect the Structure of Teeth,'

and the third with:

'The part played by Diet in preventing the onset and the spread of Caries in erupted Teeth.'

I shall limit my observations to-day chiefly to the subject of caries.

DIVISION I.

The first division deals with *the relation between the structure of teeth and their liability to decay.* I am also interested

in the problem of other diseases of the teeth and jaws, periodontical diseases, and so on, but will only mention these in passing.

Unfortunately, up to the present I have not succeeded in producing typical dental caries in animals. I hope to do this before long — for it is only when a disease can be produced artificially in animals that the fundamental factors relating to that disease can be satisfactorily investigated. I will take it as a fact that the immediate cause of decay is the action of bacteria on the food sticking to the teeth. In other words, I accept a modified Miller's theory. It seems to me to be common sense that, if this be true, then the better the minute structure of the tooth, the less liable will that tooth be to attacks from the outside. If the tooth is badly formed, the enamel and the whole surface will be rough, and so more food material will stick to the teeth and undergo bacterial decomposition. It is certainly true that this happens in the case of the experimental animals, for the worse the structure the more food is found sticking to the teeth. The enamel of badly formed teeth is also often thinner and softer than normal; this again might be expected to make them more liable to be attacked by caries. Again, a carious process once started would certainly spread more rapidly through defectively calcified dentine. Thus it would be expected that the spread, as well as the onset, of caries would be affected by the structure of the teeth, and especially of the dentine.

Six years ago, when speaking before a British Dental Society, I said that it seemed to me to be common sense to believe that the better the structure of the teeth, the less liable would they be to become carious. I was told that I was quite wrong, that children's deciduous teeth are on the whole very well formed; that only about 3 % of such teeth are hypoplastic, whereas the majority of children's teeth become carious; in fact over 90 % of children were said to have caries.

I looked up the literature on the subject and nowhere could I find any publication which described a close examination of children's deciduous teeth. I therefore started to make a collection of such teeth. I asked my friends to give me the teeth of their children when they were shed or extracted. Many dentists have kindly supplied me with both shed and extracted teeth from private patients, and I have also received many specimens from Mr. J. W. Proud, a school dental surgeon in Sheffield. These teeth have alle been stained, embedded in balsam, ground down on a stone until very thin and examined under the microscope and I find that over 80 % of such teeth, including alle varieties and sizes, incisors, canines, molars, etc., are badly formed, and that over 80 % are carious. On the whole, the worse the structure of the tooth, the more carious is that tooth. The defects seen in these children's teeth are very similar to those I shall talk about later, produced experimentally in puppies' teeth. So that, instead of 3 % of the deciduous teeth being badly formed, there were over 80 % badly formed. The dental surgeons who provided me with the teeth were, in some cases, so kind as to give me their opinion as to the structure of the teeth. On extraction, 3 % were said to be badly formed, (hypoplastic) according to their judgment, whereas I found over 80 percent were really badly calcified.

TABLE 1.

The result of the examination of 636 children's deciduous teeth as regards structure and caries.

Type of Tooth.	Number Examined.	GOOD STRUCTURE.		DEFECTIVE STRUCTURE.	
		No. Caries.	Caries.	No. Caries.	Caries.
I	100	58	11	1	30
C	70	5	0	25	40
M	466	2	11	2	451
Total . .	636	65	22	28	521

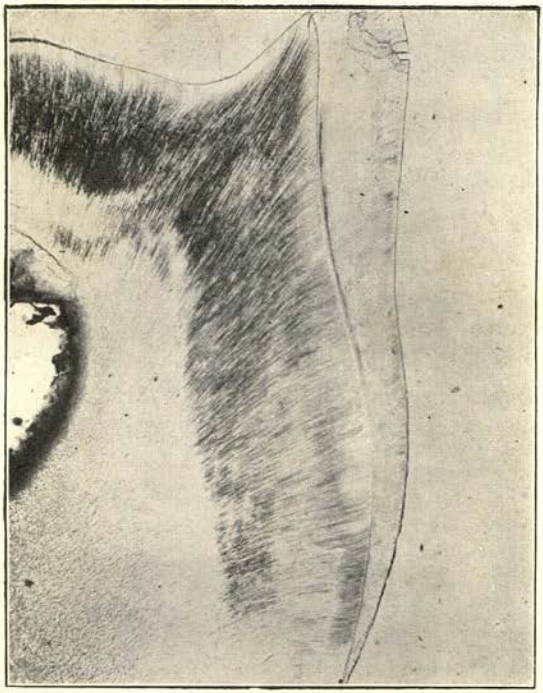

Fig. 1. Ground section of one of the very few well calcified out of many human deciduous molars examined.

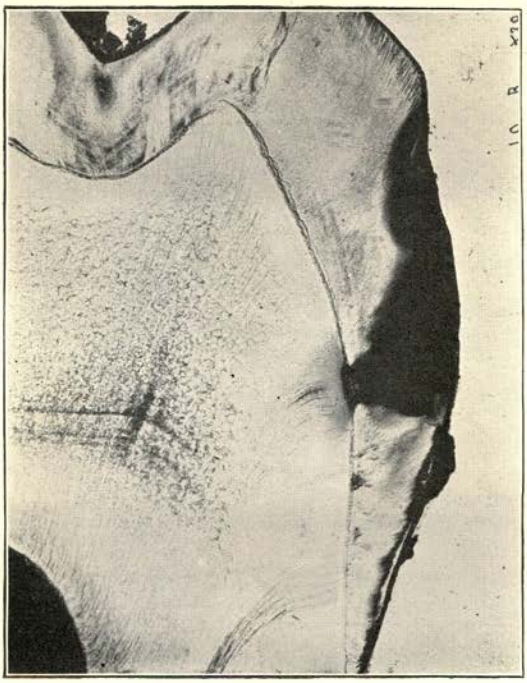

Fig. 2. Ground section of a typical human deciduous molar. (Note interglobular spaces in dentine).

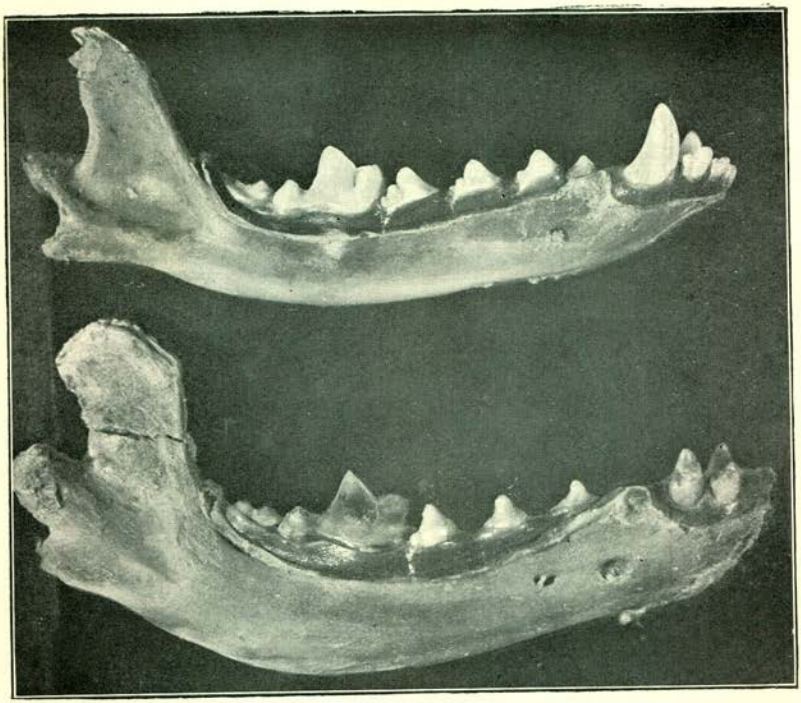

Fig. 3. Side view of the right lower jaws of two puppies — both produced experimentally by small differences in quality of diet — A, with perfectly formed teeth, B, with very defective teeth.

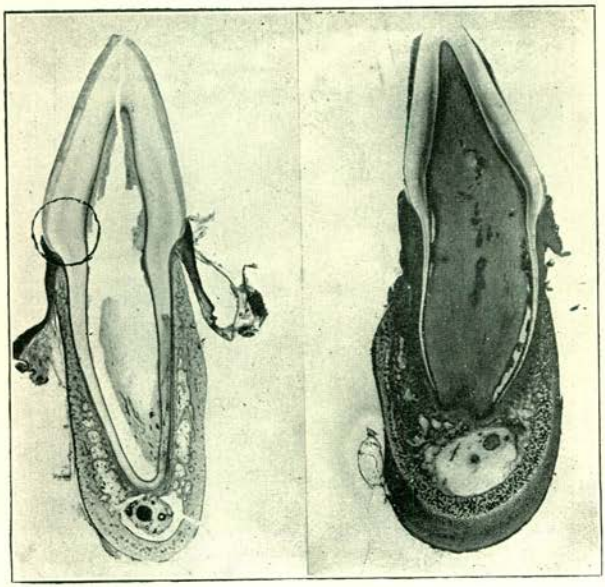

Fig. 4. Sections through the region of the lower left carnassials of A and B in Fig. 3, including tooth, alveolus and jaw bone. (Ring on A represents region from which high power photomicrographs of cross sections are usually taken).

Table 1 shows the result of the examination of 636 children's teeth (deciduous) as regards structure and caries and you will see, that at this time I had examined 100 incisors. Of these, 69 were well calcified and 58 of them were free from caries. Dental surgeons often explain the fact that incisors are less liable to decay than molars by saying that they have no crevices in which food can stagnate, that is to say, the shape of the teeth is such that they are less liable to decay. But there seems to me to be also this other possible explanation, viz., that the incisors are on the whole better calcified and hence less liable to decay than the molars.

Of all the 466 molars examined, very few, in fact only 13, were well formed; the remainder were not only poorly calcified but were also carious. In fact with a few exceptions there is seen all through a direct relationship between structure and caries.

Fig. 1 is a photomicrograph of a ground section of one of the two well formed and non-carious molars. It is better calcified than any other molar I have so far examined. The enamel and dentine are both of normal structure and there are no interglobular spaces in the dentine.

Fig. 2 gives a picture of the structure commonly found in deciduous molars, especially in second molars. Fewer interglobular spaces are seen in some cases than in this specimen and even more in others. This tooth was described by the dental surgeon on the basis of its external appearance as being normal in structure. I am examining a large number of children's teeth first in the mouth and some of these I afterwards examine in the hand after extraction or shedding, and then again microscopically when sections have been made; so that now I am able to judge fairly accurately by a careful macroscopic examination what the structure will be when finally examined microscopically. It seems to me that new

criteria as to the real structure of teeth as judged by the appearance of the enamel will have to be adopted.

(Other typical sections of children's deciduous teeth were thrown on the screen).

On examining microscopically teeth said by the dental surgeon to be hypoplastic, or slightly hypoplastic, I always find at least one group of large interglobular spaces in that line corresponding in development to the brown striae of Retzius in the enamel, namely an Owen's line. Such a tooth is generally found to have small interglobular spaces throughout the dentine, but in at least one region there is a 'line' formed by many large interglobular spaces; my diagnosis in these cases is that the diet of the child was always somewhat defective from the point of view I am about to discuss more fully, but that there has been some serious illness during which the child was not able to absorb the necessary food factors, or that the diet was still more defective at the time this particular dentine and enamel was laid down.

I think you will agree with me from the indirect evidence I have given you that there must be a direct relationship between structure and caries, quite apart from any local conditions in the mouth. I know that some dental surgeons have said that the more hypoplastic the teeth the more liable are they to decay. When they talk about hypoplastic teeth, however, they refer to a gross form of hypoplasia. Other dental surgeons, on the contrary, consider that such hypoplastic teeth are even less liable to decay. The point I wish to insist on now is that, apart from gross hypyplasia as generally accepted, the majority of children's teeth in a country like England, where dental caries in widespread, are badly formed and that, on the whole, badly formed teeth are more susceptible, and well formed teeth are less susceptible to caries.

DIVISION II.

Conditions which affect the structure of the teeth.

I am afraid I have a great deal to say about this part of the subject and it is very difficult for me to know how to summarise it and yet to make it clear to you. Lantern slides will help me greatly, and if there are any questions you would like to ask afterwards, I shall be very glad to try to answer them.

The main point I want to demonstrate is, that by very small variations in the diet which until a few years ago would have been considered negligible, you can cause the production of either good or bad teeth. The most important calcifying factor is a vitamin with a distribution and properties very similar to that called by Mac Collum the vitamin A, a fat soluble vitamin, which is important in growth. Whether or not the calcifying vitamin and vitamin A are one and the same substance I will not discuss here. I shall refer to the vitamin with which I am dealing as the calcifying vitamin.

I want to impress upon you that, apart from the energy bearing foods and the calcifying vitamin, there are other constituents of diet and environment which are also very important from the point of view of calcification; these include the amount of cereal in the diet, the type of cereal in the diet, as well as the amount and the type of salts. These all play a part. Then there is the question of the rate of growth of the animal, the amount of light, especially of ultra violet light to which the animal is exposed. Moreover not only are the ultra violet rays to which the animal is exposed of importance, but also the amount of such light to which the food is itself exposed. All these things must be considered, but my husband and I think that they are all subsidiary to the central factor, namely, the calcifying vitamin. If you have sufficient of this vitamin in the diet, provided that you have also a mini-

mum of salts, of proteins, carbohydrates and fats, then alle will be well. If you have a deficiency of this vitamin, then you must be very careful, for this deficiency can only be compensated for either by reducing in the diet those substances interfering with calcification, and especially cereals, or by increasing the exposure to ultra violet radiation.

I shall now show you some of my experimental results on the screen by means of the lantern.

Fig. 3 is a picture showing you the different appearance of the teeth and jaws of two puppies A. and B. The diet in the first case (A) included separated milk, meat, orange juice, white bread and cod liver oil, and you will see that the enamel is beautifully white and shiny. The diet in the second case (B) included separated milk, meat, orange juice, oatmeal and olive oil; the enamel here is discoloured and rough. These two animals had the same amount of food from the point of view of energy but the cereal and the fat differed. It is interesting to note that as far as salts are concerned, the second puppy had more calcium and phosphorus (oatmeal contains more than white flour), the most important elements in the formed teeth, and yet the teeth are so badly calcified.

Fig. 4 is a low power photomicrograph of a ground section through the chief cusp of the lower carnassial of the puppies A and B (see Fig. 3); it includes the crown and root of the tooth and also the gingival tissues and the alveolar and jaw bones. The great differences between the minute structure in the two sections is very obvious. In the first, A (cod liver oil and white bread), the enamel, dentine, bone and other tissues are all well formed, whereas in the second, B (olive oil and oatmeal), the enamel is thin and it takes up the carmine stain, showing that it is poorly calcified. The dentine of B is also comparatively thin, and that part developing

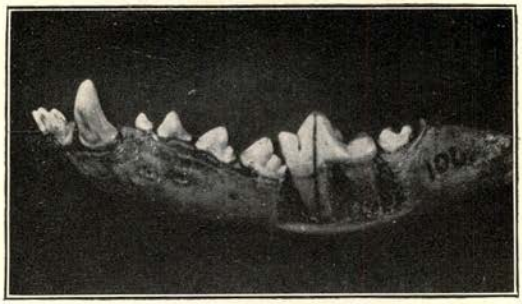

Fig. 5. Photograph of a puppy's left lower jaw with part of the bone in the region of the carnassial cut away. (The line indicates the region from which sections are usually taken).

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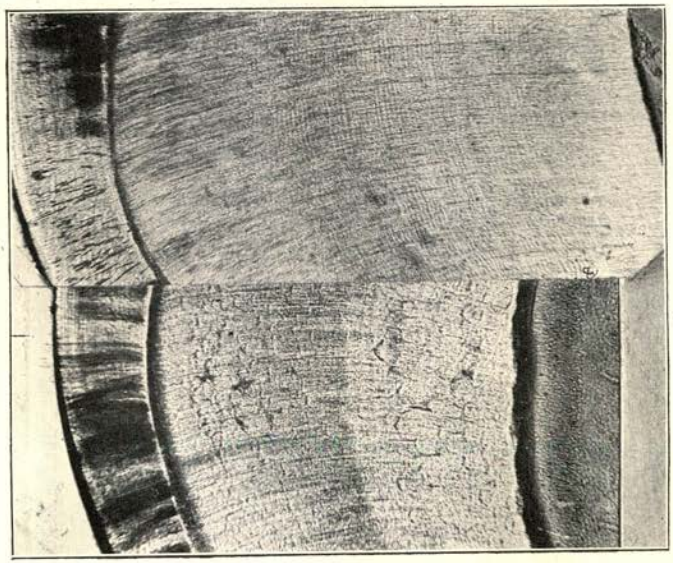

Fig. 6. Photomicrographs from the neck region of the lower carnassials of two puppies of the same litter. The dentine is thick and well formed in 328 (suet), but is thinner and contains interglobular spaces in 329 (lard).

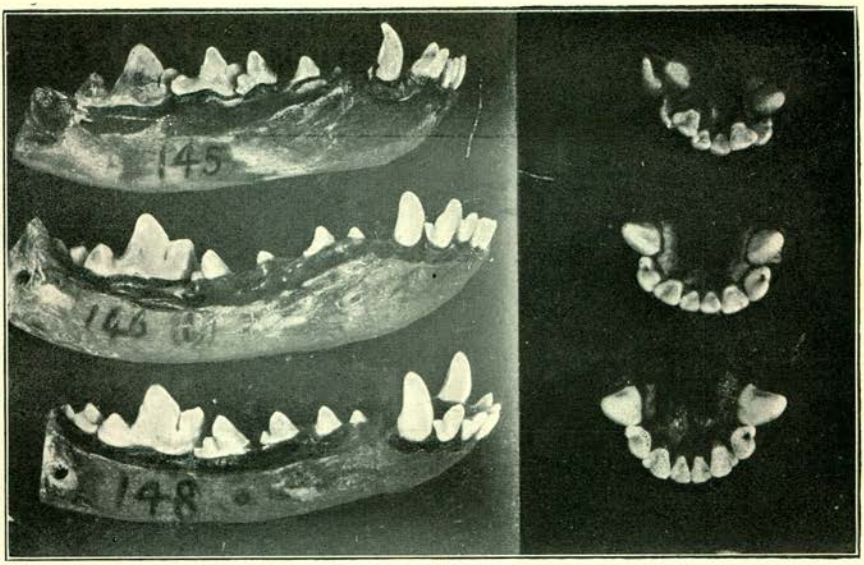

Fig. 7. Photographs of the lower jaws of three puppies of the same litter. The enamel is defective in 145 (linseed oil), slightly so in 146 (butter), but white and shiny in 148 (cod liver oil).

Fig. 8. Photographs of incisor region of the three puppies in Fig. 7. Teeth irregular in 145 (linseed oil), regular in 146 (butter) and 148 (cod liver oil).

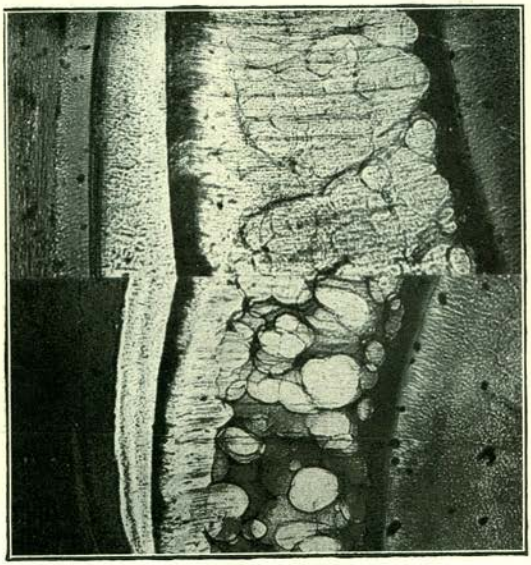

Fig. 9. Photomicrographs of the neck region of two puppies of the same family, fed on diets deficient in calcifying vitamin. The only difference was that 335 had twice as much bread daily as 333. The structure of 335 (more bread) is worse than that of 333 (less bread).

during the special diet is full of interglobular spaces. The root is also well calcified in A but not in B. The jaw and alveolar tissues are obviously hard and normal in every way in A, but contain much osteoid tissue in B. I hope in the course of my talk to-day to prove to you that the reasons for these differences in structure are two-fold. Puppy A received abundant calcifying vitamin in the cod liver oil and less of the anti-calcifying substances present in cereals, especially in oatmeal, whereas puppy B received much less calcifying vitamin, which is very deficient in olive oil, but more of the anti-calcifying substance which is abundant in oatmeal.

Next a few points as to the experimental methods. All the animals mentioned to-day were kept during the whole period in kennels which were cleaned out daily. They all received the same amount of air, sunlight and were under the same hygienic conditions, unless otherwise especially mentioned. In comparing two or more diets, puppies of one litter were used, and, as far as possible, only one factor was varied at a time. The puppies were put on diet at the same time and killed at the same time. A typical basal diet is as follows:

Cereal, 80—200 gms.
Lean Meat, 10—30 gms.
Separated Milk, 150—250 c. cms.
Fat, 10 gms.
Orange Juice (for antiscorbutic vitamin).
Yeast (for anti-beri beri vitamin).
Sodium Chloride, 1—4 gms.

The amount of food given to the individuals of a family was usually regulated by the amount eaten daily by the puppy with the smallest appetite. If one puppy left some food, then the amount of food given to the rest of the family was generally cut down.

Apart from any Vitamin A that may be present in the fat given, it will be seen (Table 2) that the basal diet is defi-

cient in this respect, but that otherwise it contains a good supply of energy bearing foods (proteins, carbohydrates and fats), and of salts. A few years ago, whatever variety of fat had been used, it would have been called a very satisfactory diet.

I will divide these puppy experiments into groups and illustrate each group by lantern slides.

Group 1 showing the importance of a fat soluble vitamin (the calcifying vitamin).

(Many lantern slides were thrown on the screen illustrating this point).

Fig. 6 shows a photomicrograph of the neck region of the carnassials of two puppies (see ring Fig. 4 indicating the region from which high power photomicrographs are usually taken). 328 was fed on the basal diet including white bread for the cereal and suet for the fat; in the case of 329, the fat was lard, otherwise, as far as quality and quantity were concerned, the diet was the same as for 328. The better calcification of 328 (suet) is obvious. The dentine is thicker and there are no interglobular spaces, whereas in 329 (lard) there is less dentine which is crowded with small interglobular spaces. Suet, therefore, contains more calcifying vitamin than lard and brings about better calcification of the teeth.

Fig. 7 shows the side view of the lower jaws of three puppies, and Fig. 8 is the front view of the same jaws. The basal diet for all was: —

White bread (ad lib.)
Separated milk,
Yeast,
Fat, 10 (gms. or c. cms.)

In 145 the fat was linseed oil,
„ 146 „ „ „ butter,
and „ 148 „ „ „ cod liver oil.

It is clear that the addition of cod liver oil to the basal diet has resulted in the formation of white, shiny teeth, well arranged in the jaws. The enamel of 146 (butter) is good, but not quite as perfect as in 148 (cod liver oil), whereas the enamel in 145 (linseed oil) is soft and discoloured and the incisors are crowded in the jaw. Photomicrographs show similar differences in the dentine.

Other experiments showed that egg yolk, large quantities of whole milk, and other substances containing vitamin A also ensure well calcified teeth, whereas most vegetable oils resulted in varied but comparatively poor calcification.

TABLE 2.

Distribution of Vitamin A.

Best Sources.	*Deficient.*
Cod liver oil and fish Oils.	Lard (as a rule).
Egg yolk.	Vegetable oils.
Milk.	Hardened fats.
Cheese (whole milk).	Lean meat.
Butter.	Egg white.
Suet.	White flour.
Green vegetables (cabbage, spinach etc.).	Oatmeal.
	Rice and other cereals.
	Tea, sugar, fruit, yeast.

Distribution of Vitamin Aiding Calcification of Teeth.

Best Action.	*Moderate or Variable Action.*	*Poor Effect.*
Cod liver oil and fish oils.	Lard.	Most vegetable oils, including olive and linseed oils.
Egg yolk.	Cocoanut oil.	Lean meat.
Milk.	Peanut oil (arachis).	Egg white.
Cheese.	Green vegetables.	White flour.
Butter.		Oatmeal, rice and other cereals.
Suet.		Sugar, fruit, yeast.

Group 2. The next point to consider is the action of cereals on the structure of the teeth and the relation of their action to that of other substances in the diet, especially the calcifying vitamin.

Fig. 9 shows sections of the teeth of two puppies which ate the same amount of everything except bread. The fat was in each case olive oil. 335 ate a large amount of bread, and the structure of the teeth is rather worse in every way than in 333, which received only half the amount of bread. The inference from this and other similar experiments is that the more cereal the animals eat the more quickly they put on weight, and the more calcifying vitamin they require for normal growth; but if the vitamin is limited then, under these conditions, worse teeth are produced. Other cereals, such as oatmeal behave in the same way.

Group 3. We must now consider the *type* of cereal. Fig. 10 shows the side view of teeth of four puppies, the basal diet of which was: —.

Cereal, 60—100 gms.,
Separated milk powder, 15 gms.
Lean meat, 10 gms.,
Oil, 10 c. cms.,
Orange juice, 3 c. cms.,
Yeast, 5 gms.,
Sodium chloride, 1—4 gms.

Fig. 11 is a high power photomicrograph of the neck of the carnassials of the four puppies of the same litter.
In 506 the cereal was oatmeal and the oil cod liver,
„ 509 „ „ „ oatmeal „ „ „ linseed,
„ 507 „ „ „ white flour and the oil linseed,
„ 503 „ „ „ white flour germ and the oil linseed.
The puppies were rather slow growing animals.

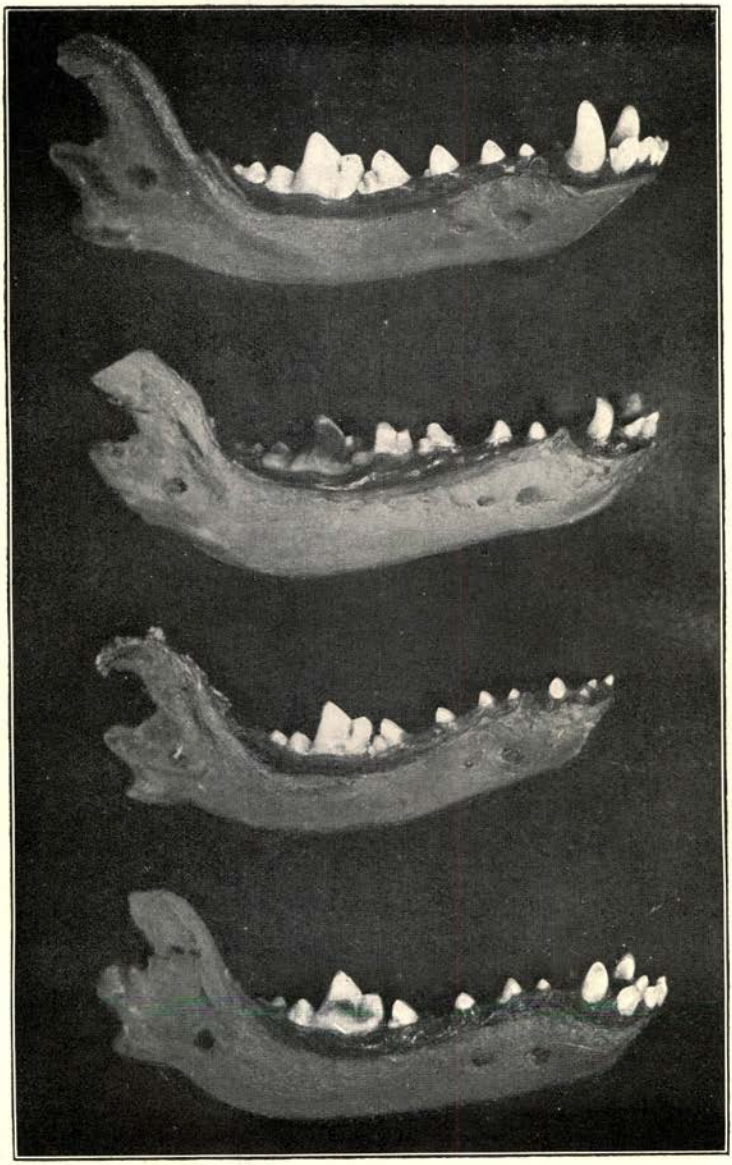

Fig. 10. Photographs of right lower jaws of four puppies of the same family. (see also Fig. 11).

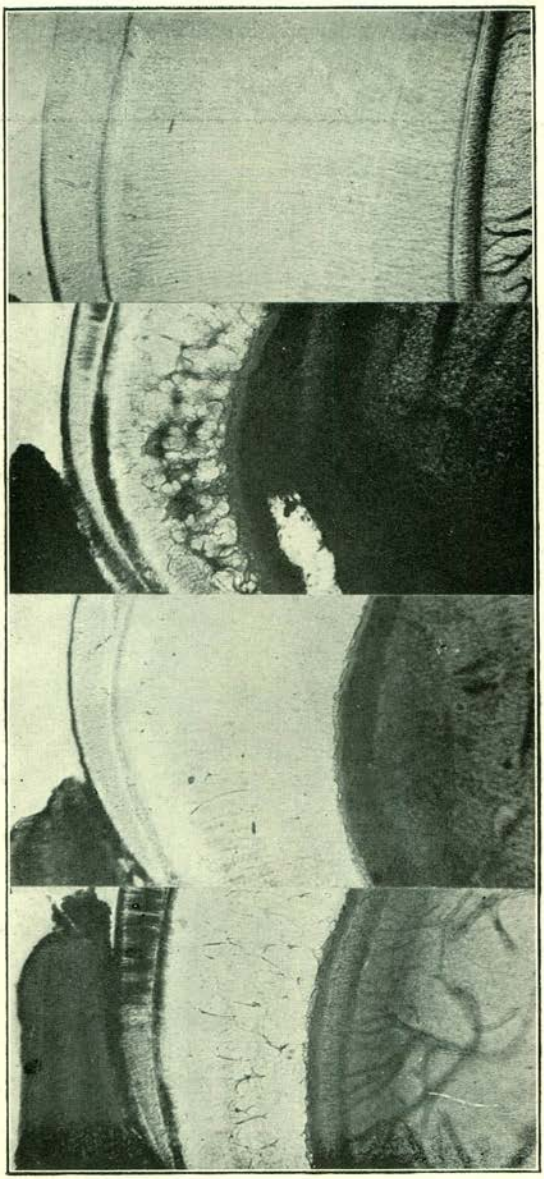

Fig. 11. Photomicrographs of the neck region of the left carnassials of the puppies mentioned in Fig. 10. As regards Figs. 10 and 11, it will be seen that the structure is perfect in 506 (oatmeal and cod liver oil), and that it is very defective in 509 (oatmeal and linseed oil). In 507 (white flour and linseed oil) the dentine is somewhat thin with few interglobular spaces, in 503 (white flour, wheat germ and linseed oil), the defects are greater than in 507 (white flour and linseed), but not as great as in 509 (oatmeal and linseed).

As far as 506 and 509 are concerned, the only difference in the diet was in the type of fat. 506 will be seen to have well formed and well arranged teeth, all of which have erupted, whereas in 509 the teeth are badly formed and irregularly arranged, and the eruption is delayed. Comparing 509, 507 and 503, the oil is the same, namely linseed oil, and the diet is therefore deficient in vitamin A; but the *type* of cereal differs, and it will be noticed that the calcification is very much the worst in 509 (oatmeal) and the best in 507 (white flour). 503 (white flour — germ) is intermediate, but 507 (white flour — linseed oil) the best of these three, is not nearly as good as 506 (oatmeal and cod liver oil), which puppy, like 509, (oatmeal and linseed oil) had oatmeal, but it also had plenty of the calcifying vitamin in the cod liver oil. Other experments with different cereals show that of all those tested, oatmeal has the worst effect if the calcifying vitamin is deficient. I may mention here that it does not appear to matter in what form the oatmeal is given. The results seem to be the same, whether we use fresh oats which we grind down ourselves, or ordinary oatmeal or groats, nor does it seem to matter where the oatmeal comes from.

It will be seen that the addition of wheat germ (10 % of the total cereal) to white flour also tends to produce worse calcified teeth under these experimental conditions. In my experience, when the calcifying vitamin of the diet is deficient, all cereals produce badly formed teeth, but they vary greatly among themselves in this respect. Oatmeal is the worst offender and white flour the least harmful, with maize, rye, and rice (polished or unpolished) occupying intermediate positions.

As you know, Scots are very keen on oatmeal and look upon porridge as their national dish. When my husband published his work on cereals a few months ago, he was severely criticised in papers all over the world. The criticisms

came chiefly from Scotsmen who seemed to miss entirely the points of the work. They said, "Look what fine people we are, and we were brought up on porridge." They forget that if porridge is eaten together with things containing abundant calcifying vitamin, then all is well. The Scots in the old days took much milk with their porridge, and often fat fish such as herrings and salmon. Now all of these substances contain the calcifying vitamin, so that any ill effects which might have been preduced by the oatmeal were counteracted.

Group 4. The question of the part played by *salts,* especially of calcium and phosphorus, in the calcification of teeth, must now be dealt with. As the teeth are largely composed of a combination of calcium and phosphorus, it is natural to believe that these salts play a very important part in the processes of calcification. In America some people have recently said that rickets is due not so much to the lack of a vitamin as to the type of salts and the balancing of these salts. We found that you could reduce the calcium and phosphorus in the diet to a minimum and yet get well formed teeth and bones, provided that the calcifying vitamin was present in abundance. If this vitamin was deficient, then the amount of calcium given in the food played a more important, though still a relatively small part.

Fig. 12 represents photomicrographs of the carnassial (neck region) of four puppies out of a litter of eight (see also Fig. 13). All had the same basal diet, including oatmeal, separated milk, meat, etc. 710, 711, 715, and 713 had olive oil as the fat. 710 had extra sodium phosphate, 711 extra calcium phosphate, and 715 extra calcium carbonate. It must be mentioned here that during the last two or three weeks of the experiment 710 was given cod liver oil instead of olive oil. It will be noticed that the last dentine formed during the taking of cod liver oil contains no interglobular spaces, while previously, during the period in which olive oil was given, large

interglobular spaces are seen. All the teeth in this part of the series dealing with salts are badly formed, although of the four dealt with here, 715 (calcium carbonate) is better than 711 (calcium phosphate) and 710 (sodium phosphate). In fact, the only salt that seems to have assisted matters at all under the experimental conditions is the calcium carbonate, for 710 (sodium phosphate) and 711 (calcium phosphate) are just about as badly calcified as 713 (control). 716 (see Fig. 13) which had the same diet as 713, except that cod liver oil was substituted for olive oil, is normal in every way without the addition of calcium or phosphorus.

Group 5. Now as to the effect of altering the diet after some months. When an animal is given a diet deficient in the calcifying vitamin, it is very easy by adding some substance containing the vitamin at once to produce good calcification. This was seen in Fig. 12 (710) and is also clear from Fig. 14, 250 and 251 were both given a diet deficient in vitamin A; but 251, after about two months, had egg yolk added, whereas the other one remained on the deficient diet. It will be noticed that the egg yolk at once brought about the production of well calcified dentine. On the other hand it is more difficult after a diet containing abundant calcifying vitamin to bring about the production of badly calcified dentine by giving a diet deficient in the vitamin, presumably because during the first period the vitamin is stored and then made use of during the second.

Group 6. Up to this point I have been talking about *diet* in relation to calcification. Now I will deal with the importance of *ultra-violet rays*. The first thing that we noticed was that ultra-violet rays were of distinct assistance in calcification when the calcifying vitamin was deficient in the diet. When there is plenty of the calcifying vitamin present calcification is so good that the ultra-violet rays applied to the skin do not appear to make any difference. Moreover even when the cal-

cifying vitamin is absent or very deficient these rays do not seem to assist calcification, but with a less deficient diet the ultra violet rays certainly help. Fig. 13 shows the jaws of four of a family of eight, (see also Fig. 12) the basal diets of which were oatmeal, separated milk, meat, etc. 713 (Fig. 12), 712, 714, and 717 had olive oil for the fat, 716 cod liver oil. 713, 717 and 716 had a large quantity of oatmeal, while 712 and 714 had half that quantity. In the case of 714 and 717, the animals had ultra-violet rays from a mercury vapour lamp on their bodies three times weekly. 713 compares with 717. It will be noticed that there is very little difference between the two, but in 712 and 714, where the amount of oatmeal containing the anti-calcifying substance is less, because less was eaten, the ultra-violet rays given to 714 have certainly assisted in the calcification. Even so, the calcification is not as good as it is in 716, which received a large quantity of oatmeal with cod liver oil. The point about this and other similar experiments seems to be that if the diet contains abundant calcifying vitamin, it does not matter, within limits, what other things are eaten or whether the body is exposed to ultra-violet radiations, but if this vitamin is deficient, then the balance of the other substances of the diet and the effect of ultra-violet rays on teeth calcification may become much more necessary and important.

I may just mention here in passing that, apart from the structure of the teeth themselves, this vitamin also plays an important part in the normal formation of the tissues surrounding the teeth, the jaws, the alveolus, and so on. But I will not take up your time by discussing this side of the problem to-day. I am, however, convinced in my own mind that the structure of these tissues plays a very important part in periodontal diseases.

(Lantern slides of sections were then shown illustrating by means of radiographs and photomicrographs, the effect of

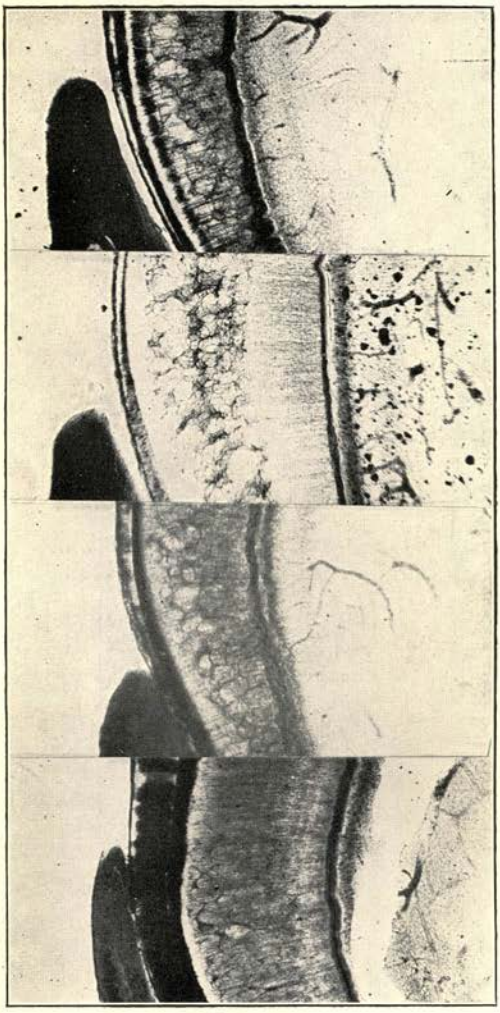

Fig. 12. Photomicrographs of the neck region of the carnassials of four puppies of the same litter. The basal diets of all contained the same amount of oatmeal, olive oil and other substances, and were deficient in calcifying vitamin. 713 had basal diet only, 710 additional sodium acid phosphate (N. B. This puppy had some cod liver oil during last three weeks of experiment. Note: last formed dentine: good), 711 additional calcium phosphate, 715 additional calcium carbonate. The sections show that the addition of the salts containing calcium and phosphorus makes little difference to the calcification under these experimental conditions. Compare with perfect structure of 716 (Fig. 13), a puppy of the same family which had same diet as 713, except that cod liver oil was substituted for linseed oil.

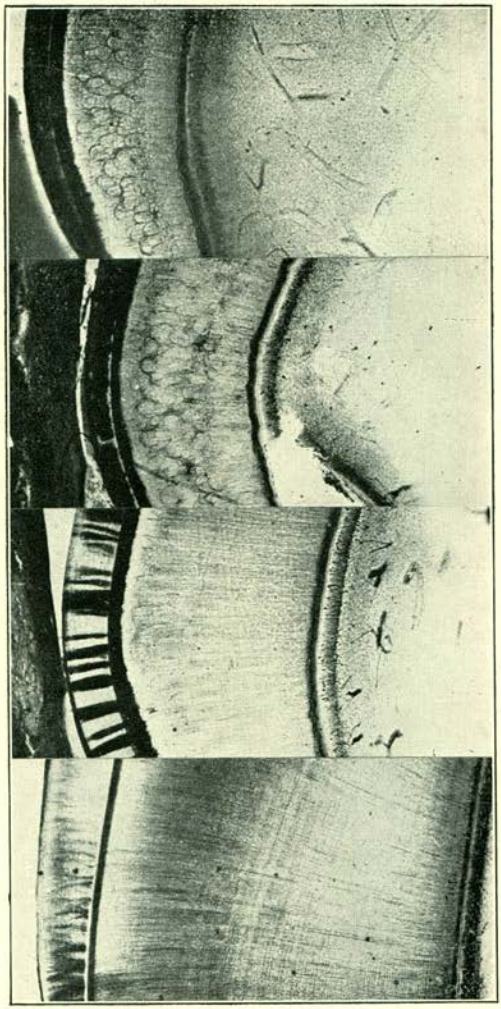

Fig. 13. Same litter as those of Fig. 12. Shows the effect of exposure to ultra-violet radiations compared with the action of cod liver oil. 717 had same diet as 713 (Fig. 12) but animal exposed to ultra-violet radiations — structure very slightly improved. 712 and 714 had half amount of oatmeal taken by rest of series. 714 in addition was exposed to ultra-violet radiations, as the result of which a definite improvement in the structure is seen.

deficient diets from the point of view under consideration, on the structure of the jaws, alveolus, as well as the other periodontal tissues).

DIVISION III.

The part played by diet in preventing the onset and the spread of caries in erupted teeth.

I will now deal with the third division, namely the effect of diet after eruption of the teeth on the onset and spread of decay. As I have already mentioned, I have not yet been able to test many of these points directly on animals. The experimental method of dealing with this question in the first place on animals is most important, for, as you will realize, it is only by such methods that each factor of diet and environment can be tested in turn. It is practically impossible to do the preliminary experimental investigation on human beings. However, although the animal experimental method is of very great value, yet the final test must be made on man. It is possible, at least, to test on him the conditions that have been found from the animal work to give good results.

Apart from structure then, what factors decide whether teeth will decay or not?

I shall not attempt to discuss the part played by the cleaning of the teeth and gums, chewing of hard or soft food, taking of acid or alkaline foods. You know more about these things than I do. I will simply consider this matter from that side in which I personally am interested. In the first place, the teeth are undoubtedly living structures, and as such would be expected to have powers of resistance. Even the enamel appears, according to Dr. J. Howard Mummery, to respond to external stimuli. Dr. Mummery has shown that when caries is tending to spread slowly, a translucent zone is seen in the enamel somewhat analogous to that found so often in the dentine. In the second place, when animals are fed on diets containing, among other things, abundant calcifying vitamin,

they resist to a much greater extent infectious diseases such as distemper, mange, broncho-pneumonia, than when fed on a diet deficient in this factor. May it not be that resistance to the onset of caries also will be greater on a diet containing abundant calcifying vitamin? I am carrying out some experimental work on puppies in this connection. Rat work is also in progress, but I have not succeeded in producing readily the results which Mc Collum and his co-workers claim to have obtained on these animals.

Recently Dr. Lee Pattison and I have been carrying out an investigation on children who are directly under Dr. Pattison's care in a hospital. These children suffer from tuberculosis of the joints and lymphatic glands. During the earlier part of the work, Mr. J. W. Proud, one of the school dental officers in Sheffield, also took part in this investigation. The children were divided into three groups; one group on diet A received a large amount of calcifying vitamin in the form of cod liver oil, egg yolk, milk, etc., but no oatmeal; the second group, on diet B had less of the vitamin and a large amount of oatmeal daily (that is to say, had more of the anti-calcifying substance found in cereals and especially in oatmeal); the third group, on diet C, had a diet intermediate between these two, namely one which contained an intermediate amount of calcifying vitamin and anti-calcifying substance. (See Table 3).

TABLE 3.

Effect of diet on development and extension of caries in children

	A	B	C (control)
Milk	2 to 1½ pints	¾ to 1	1½
Bread	2 to ½ oz.	5 oz.	ad lib.
Fat	1 oz. butter	1 oz. margarine	1 oz. margarine
Sugar	1½ oz.	3 oz.	1½ oz. - jam
Rice.	½ oz.	½ oz.	½ oz.
Oatmeal	0	2 to 4 oz.	occasionally
Meat (cooked) .	1½ oz.	1½ oz.	2½ oz.
Egg	1	v. little	sometimes
Cod liver oil .	3 to 6 drachms	0	2 to 3 dr.
Cocoa	made with milk	made with milk	made with milk.

The condition of the childrens' mouth was very carefully charted as soon after they entered the hospital as possible. We made notes on the external appearance of the teeth, using a slightly blunt probe for examining the surface. We noted the amount of caries and, as far as possible, the under-mining caries, the presence of gingivitis and other conditions. The children were then re-charted before being discharged from the hospital, after, on the average, a period of eight months. A different coloured pencil was used for the re-charting. The extent of the spread of caries was by this means located for each child. We then set to work and analysed our results. This was not an easy matter; but in spite of all the difficulties there appeared an obvious difference in the rate of the spread of caries in the three groups. It was least, namely 1—4 new points of caries per child, in the children on diet A, where the calcifying vitamin was most abundant, and most, namely 5—1 new points of caries per child; in those on diet B, where the calcifying vitamin was least and the anti-calcifying substance was greatest. Group C was intermediate, ie 2—9 new points of caries per child, both as regards diet and the spread of caries. (see Table 4). These differences seem too large to be accounted for by the difficulties of the investigation.

TABLE 4.

Effect of diet on caries in children.

Diet	Main dietetic difference.	Average new carious points per child in about 8 months.
A	Abundant calcifying vitamin and calcium. No oatmeal.	1·4
B	Less vitamin and calcium. Oatmeal.	5·1
C	Intermediate between A and B.	2·9

It may be noted here that the structure, as far as could be seen from external examination, was bad to the same extent in each of the three groups, so that presumably the part played by structure in these results was negligible. The childrens' teeth were regularly cleaned and the hardness of the food, the amount of fruit received, and so on, were about the same.

Unfortunately the number of children dealt with was small, but since that time we have investigated the condition in more than sixty other children, and, as far as we can tell at the moment, these results bear out the figures given in Table 3. You will note from this table the obvious difference; but in work of this nature, in which it is difficult to control all factors, it is necessary to study a large number of cases before definite conclusions can be drawn. When coming to live in Sheffield five years ago, we tried to carry out an investigation of this nature on a large scale, but it was found to be impracticable for one reason and another. It was because of this difficulty that we started an investigation on a small scale at the hospital under Dr. Pattison's control. Now that more interest has been awakened with regard to this subject, it is possible that a larger investigation may be carried out.

How can the teeth after eruption resist the spread of decay under some conditions?

I have mentioned already that even enamel of the teeth appears to react to external stimuli. Undoubtedly dentine and the pulp do so. In the case of the pulp, resistance is attempted by producing secondary dentine, which tends to prevent caries from spreading and so damaging the pulp in which the living cells reside. Now this secondary dentine may be large in bulk and well formed, with no interglobular spaces. On the other hand, it may be small in amount and contain many spaces. It seemed to me that the type of secondary dentine might give an indication of the nature of the diet eaten during the period

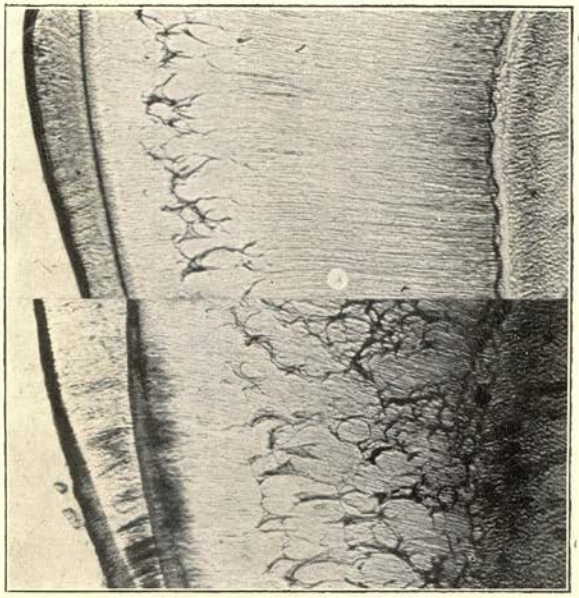

Fig. 14. Photomicrographs of neck region of the lower carnassials of two puppies of the same litter. The result of changing from a diet deficient in the calcifying vitamin to one containing it in abundance is demonstrated. Both had the same vitamin deficient diet at first but later 250 had egg yolk added. Note the great improvement in the dentine of 250.

of its formation. I therefore, by artificial stimulation, produced secondary dentine in puppies fed on different diets. By this means I was able to cause the production of well formed secondary dentine when the diets were good from the point of view under discussion to-day, and either secondary dentine containing many interglobular spaces or very little secondary dentine when the diet was defective.

These experiments seem to indicate that the resistance of the teeth to harmful stimuli such as attrition and caries, is greater on diets which have been found to promote calcification than on the defective diets. (Lantern slides were shown illustrating these conditions).

I have so far considered the resistance of the teeth themselves to caries. Another point of importance must be the conditions of the mouth. I will not discuss to-day the ordinary views held as to the immediate cause of caries, being due, as shown by that great worker Miller, to the action of bacteria on carbohydrates, but will confine myself to a few remarks on the nature of saliva. One point worked out by Dr. Lee Pattison is that when the diet contains abundant calcifying vitamin, then there is, anyway in the children he studied, much more calcium in the saliva than when the diet is deficient in this respect. Probably in other ways too the saliva is affected by a diet which also alters the resistance of teeth to caries. It may be noted here that Dr. Pattison found very small variations in the hydrogen ion concentration and alkalinity of this saliva. Moreover, he found, even as far as the calcium was concerned, that there was little constant variation from day to day or during any one day, provided the diet remained the same.

The main point that I wish then to impress upon you to-day is that by very small variations in a diet, variations which, until recently, would have been considered negligible, you

can cause the production of either good or bad teeth; also that even after eruption, the resistance to noxious influences can be affected by the same variations in the diet. If these results hold for man, as they almost certainly do, we now have at our disposal means for ensuring that, by correct feeding of infants and children, perfectly formed teeth can be produced; also, that, by the same principles of diet and hygiene the resistance of fully erupted teeth to caries can be enhanced.