OORSPRONKELIJKE BIJDRAGEN

A RESUME OF RECENT RESEARCH [1]) WORK ON THE DENTINE.

(From the John Hampton Hale Research Laboratory of the Royal Dental Hospital and School of Dental Surgery)

by

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From time to time in the last six years reports have been published in the English and American Journals of the work which has been done in the John Hampton Hale Research Laboratory of the Royal Dental Hospital of Londen. The result of this work has been gradually to unfold a new conception not only of the physiology of the dentine but also of its Pathology and of its reaction to injury.

The starting point of the investigation was an attempt to ascertain how the nerve fibres which were described by the late J. Howard Mummery were distributed in the dentine. According to Mr. Mummery the fibres lay between the fibril of Tomes and the wall of the tubule. If this assumption were correct it seemed to postulate a lymph space in which they could be accommodated and if so the lymph must be moving freely in such spaces since otherwise it would become exhausted of oxygen and nutriment and die.

Several attempts were made to investigate this matter. Trypan blue was injected into rabbits and cats and the dentine became tinged with the dye but it was extremely dilute and quite unrecognisable microscopically so that it

[1]) Lecture read at Utrecht 12th October 1929.

might just as easily have diffused along inside Tome's fibril as outside it.

Nevertheless the experiment showed that foreign bodies injected into the tissues got into the dentine and very soon a method was devised of introducing Indian Ink into the pulp tissue of a live dog or monkey [1]) and observing its fate after a few hours. Indian Ink is a suspension of finely divided particles of carbon, and decalcified sections of the dentine showed that these minute solid paticles were being carried up the tubules after even one hour, though better after twenty-four by a mass movement of body fluid between the fibril and the wall of the tubule. (Figs. 1 and 2).

This experiment proved two things: first that the fibril does not completely fill the tubule as some dental anatomists believed and secondly that there is a movement of lymph in the dentine. This tissue now appears as an intensely alive structure, extremely hard in order to fulfil its special function but at the same time tunnelled by an almost infinite number of channels — the tubules — along which there moved freely a supply of body fluids whereby metabolic changes can take place in the dentine.

We have moreover, some indication of what these changes are, lime salts are constantly being added to the dentine especially in the early years of the life history of the tooth. [2]). The salts as we later found are desposited on the walls of the tubule sometimes in such quantity as to occlude the tubule completely and render the dentine translucent.

More recently still cases have occurred in Dogs suffering from a disease as yet undescribed in which the lime salts have been withdrawn from the dentine to the extent of 14% of their total amount compared with normal dogs. Finally

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[1]) Fish E. W. The Circulation of Lymph in Dentine and Enamel. Journal of American Dental Association, May 1927.

[2]) Fish E. W. Circulation of Lymph in the Dentinal tubules with some observations on the Metabolism of the Dentine. Proc. Roy. Soc. Med. 1926. Vol. XIX. Section of Odontology.

the fibril of Tomes which is also present in the tubules appeared to be there for the purpose of enabling the odontoblast to retain control of these activities.

Following upon the observation of an actual mass movement of lymph along the tubules, the question naturally arose as to what outlet there was for the lymph at the peripheral end of the tubules and some preliminary experiments suggested that there was a communication across the cementum between the dentine and the periodontal membrane [1]). Later experiments throw considerable doubt upon this observation but the existence of a marginal lymph plexus consisting of the terminal branches of the tubules anastomosing and communicating not only with each other but with the lymph spaces called the Granular layer of Tomes was confirmed and completes a lymph path in the dentine. by which the fluids can travel from one part of the pulp along the tubules and the marginal lymph plexus to return to another part of the pulp down other tubules. The *vis a tergo* in this movement which is not direct and continuous but of an ebb and flow character is of course the pulsatile variations in the blood pressure of the pulp.

The movement of these fluids becomes strikingly illustrated when solid dye stuff is placed in the pulp tissue of a dog's tooth during life. Twelve to twenty-four hours later the whole zone of dentine near the point at which the dye was introduced is full of the dye (Fig. 3) the marginal lymph plexus becomes clearly defined by the dye and the tooth looks as though it were made of blue Bristol glass. This occurs if the dye be introduced into the pulp tissues Post Mortem but very much more slowly since there is no circulation to help it and the process of diffusion takes from one to four weeks. The ultimate result is however exactly the same since if a fluid from the pulp can reach a given point in the dentine

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[1]) Fish E. W. The Circulation of Lymph in Dentine and Enamel. Journal of American Dental Association, May 1927.

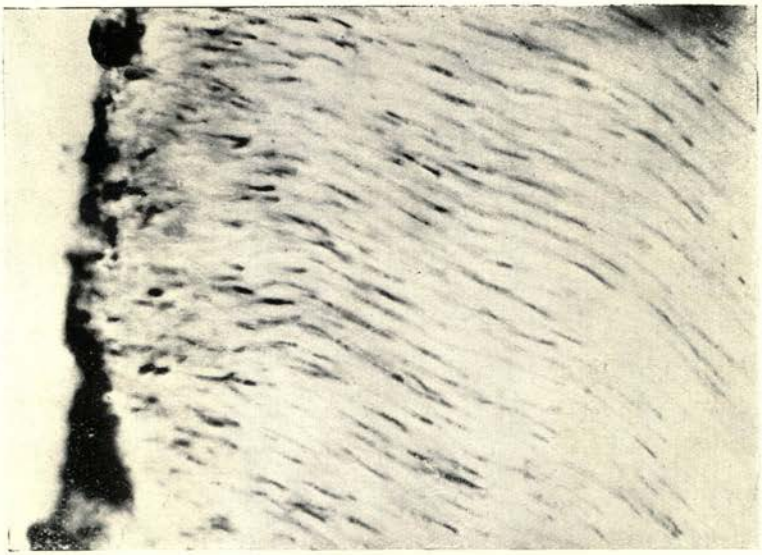

Fig. 1.

Longitudinal section of dentinal tubules of dentine of monkey. Indian ink was placed in the pulp tissue of the living tooth in situ and left for twelve hours. The solid particles of pigment have been carried along the tubules by the movements of the lymph stream in them.

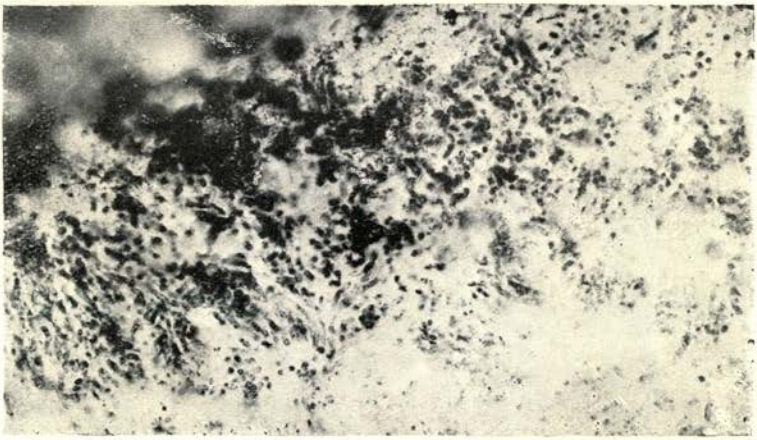

Fig. 2.

Transverse section of the dentinal tubules of a tooth treated as in Fig. 1. The pigment shows as rings in the tubules. The clear centre of each ring is the Fibril of Tomes.

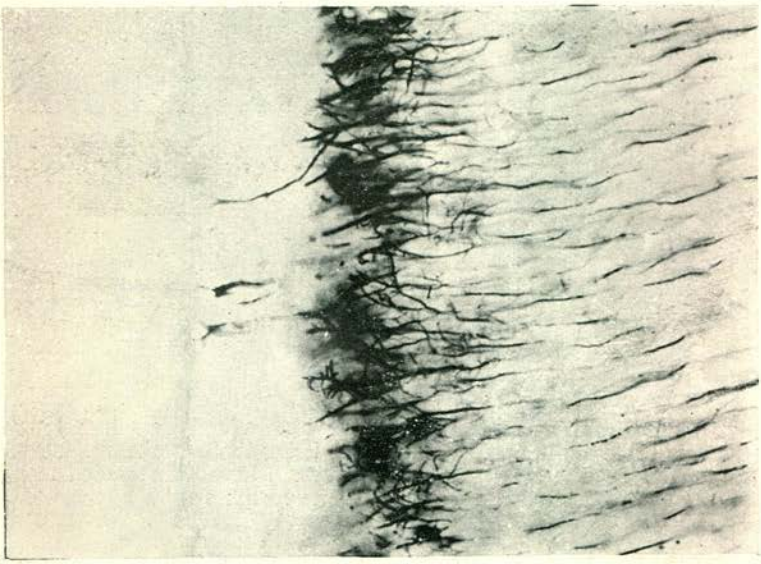

Fig. 3.

The marginal lymph plexus of the dentine of a dog. Solid dye was placed in the pulp tissue of the living tooth and sealed there for twelve hours. The anastomosis of the terminal branches is seen to form a Marginal Lymph Plexus for the exchange of lymph between the tubules.

post mortem by diffusion, that is if there is a continuous fluid path between the two points, it can do so antemortem, and since all the fluids of the body are in a state of movement more or less rapid and systematic the process of dissemination of a dye or of normal constituents of the lymph will be more rapid during life than in the laboratory after death.

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The technique of sealing a solid dye in the pulp tissue of a tooth either alive or dead has a further interest since it is a method which has proved very valuable and reliable in indicating whether any given tissue or part of a tissue of the tooth is within the pale of nutrition or not. It will be immediately obvious that if body fluids cannot reach and enter a tissue it is not vital and similarly that if they can, it is vital. As an example of this it was found by placing dye in the pulp of the dog's tooth during life that the coloured fluids of the pulp ultimately reached the enamel and permeated the prism sheaths. This is not the case in either the monkey or man except to some very slight extent and some interesting deductions can be drawn from these experiments [1]).

For our present purpose, however, the value of the method is confined to its application to the dentine. It will be at once apparent from what has been said about the comprehensive irrigation system of the dentine that any injury to the periphery of the dentine even of quite a trivial nature provided only that it opens up the tubules must interfere with the system both by blocking the movements of lymph in that particular tract of the system and also by permitting the entrance of mouth fluids to the tubules which would inevitably cause coagulation of the stagnant lymph and death of the fibril.

Some attempt will be made in the short space at our dispo-

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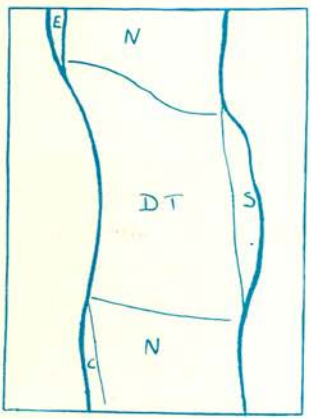
[1]) Fish E. W. A Physiological Suvey of the Nature of Enamel in Carnivora and Primates. The Dental Record, June 1928.

sal to give an account of this interesting sequel to injuries of the dentine and we shall find that a peripheral injury to the dentine does not only cause the death of the tract of dentine through to the pulp but also kills some of the odontoblasts associated with that dentine.

Injuries to the dentine however occur which are not so severe as to involve actual opening of the tubules so that the circulation may be maintained despite the injury. This condition obtains for instance at the surface of the dentine when the neck of the tooth has become denuded of gum but is still covered by the keratine cuticle. The dentine is then very sensitive-extremely sofor it is undergoing a reaction which in any other tissue would be inflammation. The result of this irritation is calcification of the tubules until the lymph of the tubule is completely obliterated and we have a translucent zone. The moment however that the injury to the dentine becomes more severe and actually opens up the tubules they die completely and right through to the pulp so that we then have the injury surrounded by a translucent zone which however was formed before the injury actually destroyed any of the dentine. This explains the presence of translucent zones under caries and shows how their presence is not incompatible with the fact that the whole tract is dead under such an injury since the translucent zone was formed before the dentine was actually opened and killed. The point will be further elucidated at a later stage in the argument.

It is first necessary to give our reasons for assuming that a tract of dentine that is injured at the surface dies. The evidence falls into two groups. Firstly we find that such a tract is cut off from the body fluids or lymph which irrigates normal dentine and which is derived from the pulp. Secondly that the Tomes fibril in the tubule is dead.

Taking the lymph irrigation first we must provide a tooth having an experimental injury of say two or three months standing or more conveniently to those who have no laboratory at their disposal any adult lower incisor which has attrition



Key to fig. 4.

Dead tract under erosion cavity at the neck of the tooth (human)

E. Enamel; N. Normal dentin

D.T. Dead tract.

S. Secondary dentin

C. Cement.

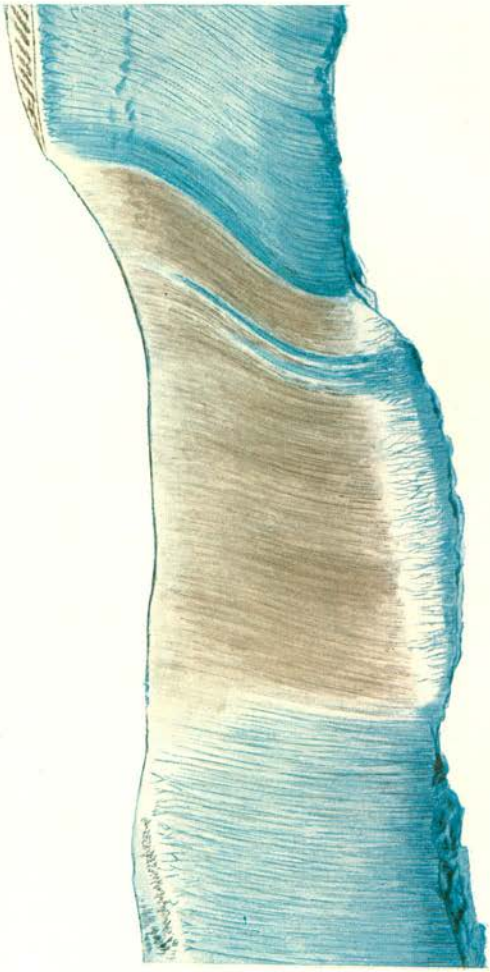


Fig. 4.

Colour drawing of human tooth having an exposed area of dentine at the neck of the tooth due to erosion. Blue dye was placed in the pulp tissue after extraction through a hole made in the dentine of the root. The dye diffused into all the normal dentine, but not into the injured tubules at the exposed neck of the tooth. These are seen to be sealed off from the pulp by secondary dentine and are therefore dead. NOTE the homogeneous layer of lime salts laid down between the primary and secondary tubules and that while the tubules of the secondary dentine are full of dye the injured primary ones are devoid of it being sealed off by the homogeneous layer.

at the biting edge will serve for the demonstration. This tooth either before extraction, or more conveniently after, has a hole drilled into the pulp and a trace of dye — solid Methyl blue is the most convenient — is introduced into the pulp tissue. The opening into the pulp is sealed with hard wax and the tooth put into dilute formalin for a few weeks.

At the end of this time it is quite blue, almost black in fact. A section is cut passing through both the peripheral lesion and the pulp when a very striking appearance at once presents. The dentine is deep blue throughout; the dye has diffused from the pulp along all the tubules and reached their terminal branches. Below the injury however and exactly co-extensive with it is a perfectly white unstained tract of dentine leading to a patch of secondary dentine which is interposed between it and the pulp. Microscopical examination of the specimen shows why the dye has failed to reach this tract of injured dentine. The secondary dentine exactly covers the injured tubules — no more and no less and the first formed part of the secondary dentine is a homogeneeus layer of lime salts (Fig. 4). This layer clearly forms the barrier for the tubules of the secondary dentine contain the dye while those of the injured dentine on the other side of the barrier do not. If this barrier be examined under higher magnification we see that the primary tubules end abruptly and that the secondary tubules start in fine branches *de novo* (Fig. 5). Despite this it has been urged that the injured tubules have failed to stain because they are undergoing fatty degeneration. It is difficult to see how fatty degeneration could take place in a dentinal tubule but a simple experiment disposes of the suggestion for if the section be immersed in Methyl Blue solution *after in has been ground* the injured tract stains just as easily as the normal dentine, the only part remaining unstained is the fully calcified barrier between primary and secondary dentine. The object of mentioning this experiment is that it shows a further point very clearly namely that often a few tubules on each side of the dead tract do not

stain even by this method and they are seen to be fully calcified up — these are tubules which have not been opened by the injury and so their circulation of lymph has not been interrupted. On the other hand the terminal and lateral branches of these tubules are in communication with the dead tubules and hence the contents of these branches will coagulate and irritate the main tubule. Hyper-calcification follows and serves as a lateral barrier between the dead tubules and the normal dentine.

To return from this digression, the previous experiment has shown that the injured tract of dentine is cut off from its source of nourishment which is the pulp but we have a second experiment which shows with equal clearness that the tract is dead. If declacified sections be prepared of such a tooth we find that there are very few odontoblasts left under the secondary dentine the majority having died when the dentine was injured. If the cavity be an experimental one and only four weeks be allowed to elapse between cutting the cavity and extracting the tooth these odontoblasts will be seen to be degenerating, they will not yet have disappeared but appear shrunken, distorted and granular. About eight weeks must elapse before the cells entirely disappear and by this time a considerable layer of secondary dentine will have formed-evidently not produced by the dying odontoblasts but by the pulp tissue or by cells carried there by the blood. The role of the odontoblasts therefore appears to be merely the laying down of tubules in the secondary dentine. If many odontoblasts escape destruction when the primary dentine is injured the secondary dentine has many tubules: if few, then it has few tubules, if none then the secondary dentine is as often happens a mass of laminated lime salts with no tubules at all. (Fig. 6).

The argument is therefore as follows: the tract of dentine under a superficial lesion which opens up the tubules is a dead tract because its odontoblasts are dead and they could not have died without the intervening tissue sharing their

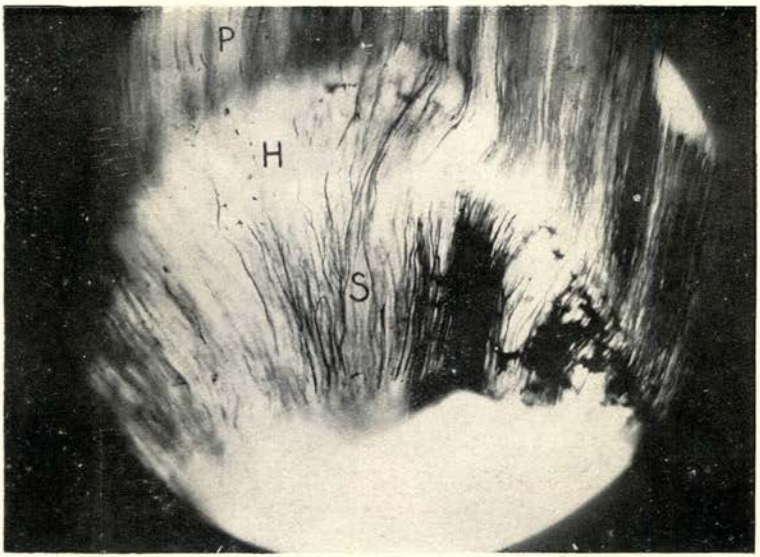


Fig. 5.
Higher magnification of primary and secondary dentine showing the homogeneous layer (H) and injured primary dentine (P) and the new tubules of the secondary dentine (S).

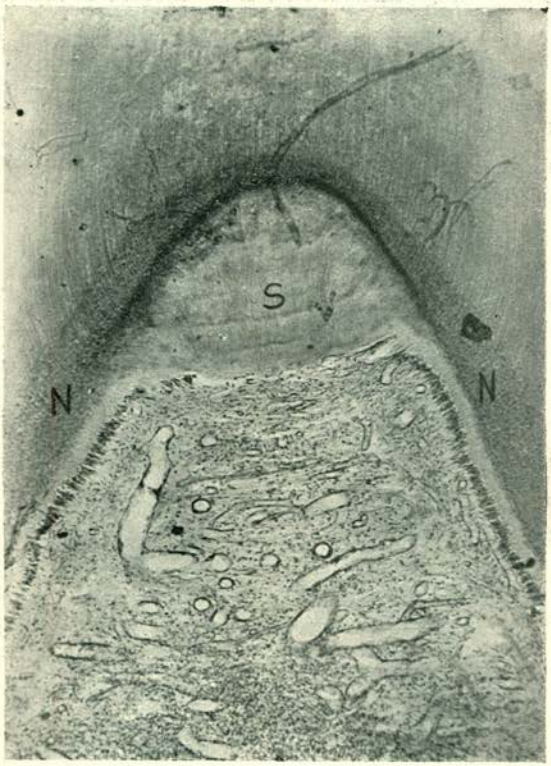


Fig. 6.

Secondary dentine sealing off the tubules injured by attrition in a human tooth. The section. Shows the absence of odontoblasts under the secondary dentine (S) while the normal row of odontoblasts is present under the uninjured dentine (N).

fate. Therefore the fibrils within the injured tubules are dead and moreover the tract is shut off from the pulp by an impervious layer of lime salts so that its tubules are open to the mouth at one end and closed from their source of nourishment, the body fluids, of the pulp at the other.

Some short reference may be made to the bearing all this has on caries for therein lies our chief clinical interest in such purely academic discussions as the foregoing.

Perhaps the most obvious point is that since the tissue between a carious cavity and the pulp is dead, caries itself is not a disease but merely the putrefaction of dead tissue. Fig. 4 shows the dead dentine at the neck of a tooth under a superficial injury and we know well that this is one of the commonest sites in which caries can occur. It follows that if and when caries supervenes in this dead tract it is merely a putrefaction. Similar dead tracts occur under lamellae and hypoplastic spots in the enamel provided these constitute a leak in the enamel, and these are just the spots which are known clinically to decay.

It may be of interest to describe the history of a case of caries as we have come to view it:

First we have a leak in the enamel at the bottom of a fissure owing to a lamella in this situation. Not all lamellae are permeable from the mouth but many of them are and cause dead tracts in the dentine beneath them. The first sign of caries is a small decalcified patch of enamel at the bottom of the lamella. The infection spreads from here directly into the dead tract of the dentine and meeting with very little opposition grows down the tubules towards the secondary dentine which walls off the caries from the pulp. Now we realise the importance of the seal of secondary dentine and the obvious confirmation of our evidence that the injured dentine is dead; for if a corner is broken off a tooth the diffusion of mouth fluids down the canals often kills the pulp unless we seal up the fractured surface with cement.

In the same way the diffusion of toxic fluids from a carious cavity into the pulp would assuredly kill it at a very early stage in the proceedings if the dentine below the cavity were not sealed off from the pulp quite effectively by the secondary dentine.

We have considered carefully the way in which this tract is sealed from the pulp and we have referred to the hyper calcification of the tubules each side the tract walling off the dead from the live tubules. Now this hyper-calcified zone round, not under, but round the dead area pays an important part in the spread of caries for not only does the infection spread towards the pulp but it also spreads laterally opening into and killing the hyper-calcified tubules which were at first only irritated. We now have included in the dead tract patches of hyper-calcified tubules showing as translucent areas and these can often be shown in ground sections which have been stained with methylene blue after grinding. The lateral reaction may occasionally be so intense that a considerable area of fully calcified tubules occurs all round the dead tract and the caries may spread slowly into it without opening up any fresh tubules since they are solidly calcified up. The result presents a somewhat atypical picture, but it is not common.

The spread of the caries towards the pulp continues until the infection is held up at the barrier between the primary and secondary dentine. How long this barrier holds out will depend on the virulence of the attack, the efficiency of the barrier and the environment of the tooth but sooner or later it breaks down and the secondary dentine is invaded. We then get an exact repetition of the earlier story. The tubules of the secondary dentine together with their odontoblasts die also and the secondary dentine is walled off from the pulp with a mass of tertiary laminated dentine which in its turn may become attacked by the caries. Barrier after barrier may form and the process may be repeated a number of times but ultimately the pulp becomes exposed and dies.

In conclusion the author wishes to express his sense of the great honour which the Members of Netherlands Dental Society have extended to him in inviting him to address them and in returning thanks to them he wishes also to express his gratitude to the Medical Research Council and the Dental Board of Great Britain for their generosity in defraying the expenses of these researches.