THE ETIOLOGY OF DENTAL CARIES.

(Thesis contains some original observations on the incidence of dental caries in breast-fed children contrasted with the condition of the teeth in hand-fed children, and a general review of the subject.)

"The condition of the teeth of the inhabitants of this country is a disgrace to her civilization and a grave menace to the public health, and it is imperative that the medical profession should take up the question of the prevention of dental caries seriously and apply to the greatest advantage the knowledge that has been laboriously accumulated by the dental profession during the last twenty years. So far this knowledge is almost unutilised by us."

These words of the Medical Officer of health for Shropshire (British Med. Journal ii P1510-1912) appear to me a suitable introduction for a thesis on the etiology of dental caries.

No one engaged in the medical inspection of school children, can fail to be impressed with the vast amount of disease of the teeth found.

The prevalence of dental caries among school children has been a subject for special consideration for many years now by dental practitioners. The School Committee of the British Dental Association collected statistics quoted by Tomes showing condition of the teeth of children attending English and Scottish Schools.
These are shown in the following table.

Number of children examined, 10,517, - the average age was twelve years.

<table>
<thead>
<tr>
<th>Temporary teeth requiring filling</th>
<th>...</th>
<th>9,573</th>
</tr>
</thead>
<tbody>
<tr>
<td>Temporary teeth requiring extraction</td>
<td>...</td>
<td>8,436</td>
</tr>
<tr>
<td>Permanent teeth requiring filling</td>
<td>...</td>
<td>13,017</td>
</tr>
<tr>
<td>Permanent teeth requiring extraction</td>
<td>...</td>
<td>6,079</td>
</tr>
</tbody>
</table>

Sets of teeth free from decay amounted to 1,508, or only 14.2 per cent.

An examination of the Reports of School Medical Officers from all over Great Britain shows that the number of school children with sound teeth varies in the different districts from 5 per cent to 30 per cent. For example, in Glasgow (1911) among 14,869 children attending Board Schools including boys and girls, and whose ages ranged from 4 to 15 years, the percentage of children found with sound teeth was 16.7%. Among 6,247 boys and girls, ages 4 to 15 attending Voluntary Schools 19.4% were found with sound teeth.

In Edinburgh (1911) of 13,349 children if classed together as Infants, 9 year olds, and Leavers on same plan as for Glasgow, about 10% are found with sound teeth.

In Bradford, those with sound teeth amounted to 4.7% in better class districts, and 7.6% in poor class districts.

In Berkshire, 33% were found with sound teeth.

In Northamptonshire, about 30% of children in the urban districts had sound teeth, and in the rural districts the number found was 37%.
In Hungary, investigations have shown that in some districts, at all events, only 13% to 17% of the children have sound teeth.

In Baden, the conditions of the teeth appears even worse.

Figures for other districts could be given, but they all tell the same tale.

It will be of interest to compare these figures with figures furnished by an investigation on the part of J.R. Mummery (Trans. Odont. Soc. Vol. ii, New Series) into the prevalence of dental caries among ancient and existing uncivilized races. Mummery, in his paper, gives an account of an examination of 1,658 skulls found in "The Ancient Tumuli of Wiltshire and other parts".

In 68 skulls, dating back to the Stone Age, only two cases of caries were found.

Among 44 skulls of a similar race, inhabiting more northern parts of England, 9 cases of caries were found.

In 32 skulls of a later race - Bronze Period - there were 7 cases of caries.

In 143 skulls of Romans, found in Great Britain, there were 32% with carious teeth.

In 76 Anglo-Saxon skulls there were 12 showing caries.
Mummery adds - the proportionate amount of caries is greatly diminished in these Anglo-Saxon skulls, compared with Romano-British skulls, and we may reasonably assume that the simple habits of the Anglo-Saxons, together with their nourishing food, tended to maintain a higher standard of health, and consequently a better condition of the teeth than their predecessors enjoyed.

In 36 Egyptian mummies there were 11 cases of caries.

In 9 skulls of Esquimaux - a people of nomadic habits, living almost entirely on meat he found one case of caries and one with two molars removed.

In 51 skulls of Indians of North-West America - a people whose diet is chiefly dried fish, he found two cases of caries; and among North-American Indians of the interior, 21 skulls showed two cases of caries. Their diet was meat, with occasional addition of roots, and they were often subject to great privation. Among the Gauchos - a mixed race of Indians and Spanish blood, inhabiting Argentine, and whose diet was entirely roast beef with Paraguay tea without sugar, only one case of tooth-ache could be traced.

Among Indians of same race, inhabiting towns and indulging in artificial diet - acid confectionery and inferior wines - caries was prevalent.

The Arabs of the Nubian Desert - whose diet is chiefly milk and the flesh of camels - possess sound and well formed teeth.
In 38 skulls of Fiji Islanders, two showed caries.

In 66 skulls of New Zealand Natives two showed caries.

In 70 skulls of Polynesians, a people whose diet is chiefly vegetables and fish - there were 8 with caries.

In 21 skulls of Sandwich Islanders, diet mainly vegetable with small amount of meat, - there were 4 with caries.

In 132 skulls of Australian natives, there were 27 with caries, in some, caries was extensive.

In 33 Tasmanian skulls (native) 9 showed caries; in the majority the caries was extensive.

In 49 skulls of Zulu Kaffirs there were 7 with caries.

In 268 skulls of Africans of the tribes which supply the Slave Market, there were 66 with caries. In 16 of these cases the whole of the molars and premolars were carious. The tribes are the most feeble of African tribes; they live in unhealthy districts and live on a mixed diet, but mainly vegetable.

Of natives of Southern India - whose diet is varied, but frequently unwholesome, and among whom, sweetmeats are much indulged in, 71 skulls showed 10 with caries; in 3 cases six teeth were involved.

Of natives of Northern India - whose diet is chiefly vegetable and of wholesome nature, and
whose staple food is wheat, 153 sculls showed 9 with caries. In none were there more than 2 teeth carious.

Pickerill, Prevention of Dental Caries, (1912) P 9. in an examination of 260 Maori sculls, all from an uncivilized age, found caries present in only 2 cases, or 0.76%.

In descendents of Britons and Anglo-Saxons he found 86% to 98% of caries.

In 50 Maori children living under European conditions entirely, 95% had caries. He shows "the Maori to have been the most immune race to caries, for which statistics are available".

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The foregoing details may be tabulated thus:

### MODERN RACES

<table>
<thead>
<tr>
<th>CLASS of PERSON EXAMINED</th>
<th>Number of Persons Examin en</th>
<th>Percentage with Carious Teeth</th>
<th>AUTHORITY</th>
</tr>
</thead>
<tbody>
<tr>
<td>English &amp; Scottish Schools, Boys &amp; Girls...</td>
<td>10,517</td>
<td>86.8</td>
<td>British Dentist Assoc. (Tomes).</td>
</tr>
<tr>
<td>Glasgow Board School Children ...</td>
<td>14,869</td>
<td>83.0</td>
<td>S.M.O Report 1912.</td>
</tr>
<tr>
<td>Glasgow Voluntary School Children...</td>
<td>6,247</td>
<td>80.6</td>
<td>S.M.O Report 1912.</td>
</tr>
<tr>
<td>Edinburgh School Children...</td>
<td>13,349</td>
<td>90.0</td>
<td>S.M.O. Report 1912</td>
</tr>
<tr>
<td>Bradford School Children...</td>
<td>...</td>
<td>95.3</td>
<td>S.M.O Report 1912.</td>
</tr>
<tr>
<td>... ... ...</td>
<td>...</td>
<td>92.4</td>
<td></td>
</tr>
<tr>
<td>Berkshire School Children...</td>
<td>67.0</td>
<td>67.0</td>
<td>S.M.O Report 1911.</td>
</tr>
<tr>
<td>Northampton School, Urban...</td>
<td>70.0</td>
<td>70.0</td>
<td>S.M.O Report 1911.</td>
</tr>
<tr>
<td>Northampton School, Rural...</td>
<td>63.0</td>
<td>63.0</td>
<td>S.M.O Report 1911.</td>
</tr>
<tr>
<td>North Germany School Children</td>
<td>19,725</td>
<td>95.0</td>
<td>Schleswig Holstein Dent. Assoc.</td>
</tr>
</tbody>
</table>
## ANCIENT and UNCIVILIZED RACES

<table>
<thead>
<tr>
<th>CLASS of PERSON or SKULLS EXAMINED</th>
<th>Number Examined</th>
<th>Percentage with Carious Teeth</th>
<th>AUTHORITY</th>
</tr>
</thead>
<tbody>
<tr>
<td>British skulls (Stone Period)...</td>
<td>68</td>
<td>2.9</td>
<td>Mummery</td>
</tr>
<tr>
<td>British skulls (probably later)</td>
<td>44</td>
<td>20.4</td>
<td>&quot;</td>
</tr>
<tr>
<td>British skulls (Bronze Period)...</td>
<td>32</td>
<td>21.8</td>
<td>&quot;</td>
</tr>
<tr>
<td>Romano-British skulls...</td>
<td>143</td>
<td>32.0</td>
<td>&quot;</td>
</tr>
<tr>
<td>Anglo-Saxon Skulls</td>
<td>76</td>
<td>15.7</td>
<td>&quot;</td>
</tr>
<tr>
<td>Egyptian Mummies</td>
<td>36</td>
<td>30.5</td>
<td>&quot;</td>
</tr>
<tr>
<td>Esquimaux Skulls</td>
<td>9</td>
<td>11.0</td>
<td>&quot;</td>
</tr>
<tr>
<td>R.W. American-Indian skulls...</td>
<td>51</td>
<td>4.0</td>
<td>&quot;</td>
</tr>
<tr>
<td>Fiji Islanders skulls...</td>
<td>38</td>
<td>5.2</td>
<td>&quot;</td>
</tr>
<tr>
<td>Macri skulls</td>
<td>66</td>
<td>3.0</td>
<td>&quot;</td>
</tr>
<tr>
<td>Polynesian skulls</td>
<td>70</td>
<td>11.4</td>
<td>&quot;</td>
</tr>
<tr>
<td>Sandwich-Islander Skulls...</td>
<td>21</td>
<td>19.0</td>
<td>&quot;</td>
</tr>
<tr>
<td>Australian native Skulls...</td>
<td>132</td>
<td>20.4</td>
<td>&quot;</td>
</tr>
<tr>
<td>Tasmanian Skulls</td>
<td>33</td>
<td>27.2</td>
<td>&quot;</td>
</tr>
<tr>
<td>Zulu Kaffir &quot;</td>
<td>49</td>
<td>14.3</td>
<td>&quot;</td>
</tr>
<tr>
<td>African (Slaves) Skulls...</td>
<td>268</td>
<td>24.2</td>
<td>&quot;</td>
</tr>
<tr>
<td>Northern Indian Skulls...</td>
<td>153</td>
<td>5.8</td>
<td>&quot;</td>
</tr>
<tr>
<td>S. Indian Skulls...</td>
<td>71</td>
<td>14.0</td>
<td>&quot;</td>
</tr>
<tr>
<td>Macri Skulls Unciv.</td>
<td>260</td>
<td>0.76</td>
<td>Pickerill</td>
</tr>
</tbody>
</table>
To these may be added the table quoted by Pickerill, showing results of Patrick's Examination of pre-historic crania found in certain American museums, thus: -

<table>
<thead>
<tr>
<th>RACE.</th>
<th>Number of Teeth examined.</th>
<th>Percentage Carious.</th>
</tr>
</thead>
<tbody>
<tr>
<td>Asiatics (including Chinese, Japs, Malays, Armenians, Hindoos and Burmese)</td>
<td>2,180</td>
<td>2.064</td>
</tr>
<tr>
<td>Egyptians and Africans</td>
<td>3,306</td>
<td>3.418</td>
</tr>
<tr>
<td>Polynesians and Australians</td>
<td>2,738</td>
<td>4.309</td>
</tr>
<tr>
<td>Central Americans</td>
<td>930</td>
<td>4.872</td>
</tr>
<tr>
<td>North Americans - including Esquimaux</td>
<td>27,362</td>
<td>5.093</td>
</tr>
<tr>
<td>South Americans</td>
<td>6,719</td>
<td>5.804</td>
</tr>
<tr>
<td>Europeans</td>
<td>3,422</td>
<td>7.079</td>
</tr>
</tbody>
</table>

From these statistics it is evident that dental caries is enormously more prevalent among modern peoples than it was among the ancients.

**INFLUENCE of SOCIAL CONDITIONS on DENTAL CARIES**

There is reason to believe that even among these ancient races there were differences in favour of the less civilized. Among the Romans and Egyptians - whose civilisation approximated more closely to our own than was the case with the other races mentioned - one can see a greater tendency to decay of the teeth.
There must have been some differences in the habits, or diet, among those who, presumably, were living in greater luxury, which made them more susceptible to decay of the teeth. This is borne out by what is observed among modern school-children, for it has been remarked that, speaking generally, the children of poorer class districts have better teeth than children of better class districts. For example, Tomes, speaking of children in poor and high class districts says, the children in these latter "compared by no means favourably with their less fortunately placed brethren" so far as the condition of the teeth was concerned. The School Med. Officer for Kilmarnock district of Ayrshire says (Report 1910) "The poor, ill-cared-for children have the best teeth".

S.M.O for Borough of Bootle (Report 1910) says "Speaking generally, the poorer children possess the best teeth". The M.O. for Liverpool says (Report 1910) "As a matter of interest, it was noticeable that, generally speaking, the amount of sepsis and number of teeth decayed were in inverse proportion to the cleanliness and better social condition of the children, in other words, the poorest children often had the best teeth".

The reports of some other School Medical Officers bear similar testimony to the relatively better condition of the teeth in poorer class children.
INFLUENCE of SEX.

Tomes says: "After puberty the female sex is distinctly more liable to dental caries than the male" Röse in an examination of the teeth of 6,280 children, the number of boys and girls being about equal, found that caries was present in about equal amounts.

At Liverpool (S.M.O Report) practically no difference was found between condition of teeth in boys and in girls. In a series of 274 girls and 145 boys all in their twelfth (12th) year of age, I found the proportion of caries was 100 for girls to 93 for boys.

From other reports it would appear that on the whole there is very little difference in the amount of caries in boys and girls, but perhaps a slight preponderance in the case of girls.

INFLUENCE of AGE.

Tomes says: "If it (caries) has not occurred before the age of five and twenty, there is a strong probability, especially in males, of immunity till about the fiftieth year, when, coincidently with the manifestations of bodily decline, the teeth again become liable to be extensively attacked with caries"

Hopewell-Smith, says: (Transaction School Dentists Soc., vol 4. No. 4., 1910) "We are all, I think, agreed that dental caries more or less stops after the period of adolescence is over, that immunity sets in, and that, speaking broadly, men and
women consult dental surgeons more frequently for the
results of the changes in the sockets of their teeth,
and for the repair of pre-existing dental defects".

Black - "Caries of the teeth, in most of its
practical aspects, is a desease of youth".

**INFLUENCE of HEREDITY.**

Hereditary influence does not seem to have any
close relationship to dental caries, at least directly.
The influence of heredity is seen, however, in irreg­
ularities in position of the teeth, and in this way may
be a predisposing cause of caries.

**RELATIVE LIABILITY of the INDIVIDUAL TEETH to CARIES**

J. F. Flagg, in Dental Cosmos, 1874, p. 401, gives
two tables, one his own, and the other by W. C. Head,
showing liability of the different teeth to caries:-
thus,

<table>
<thead>
<tr>
<th>W.C. Head's Table (Age 10-12)</th>
<th>J.F. Flagg's Table</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Upper Central Incisor</td>
<td>1. Lower first molar</td>
</tr>
<tr>
<td>2. Lower First Molar</td>
<td>2. Upper first molar</td>
</tr>
<tr>
<td>3. Upper first molar</td>
<td>3. Lower second molar</td>
</tr>
<tr>
<td>4. Lower second molar</td>
<td>4. Upper second molar</td>
</tr>
<tr>
<td>5. Upper lateral incisor</td>
<td>5. Upper lateral incisor</td>
</tr>
<tr>
<td>6. Upper second molar</td>
<td>6. Upper second pre-molar</td>
</tr>
<tr>
<td>7. Upper second pre-molar</td>
<td>7. Upper central incisor</td>
</tr>
<tr>
<td>8. Upper first pre-molar</td>
<td>8. Upper first pre-molar</td>
</tr>
<tr>
<td>9. Lower second pre-molar</td>
<td>9. Lower second pre-molar</td>
</tr>
<tr>
<td>10. Lower third molar</td>
<td>10. Lower third molar</td>
</tr>
<tr>
<td>11. Upper third molar</td>
<td>11. Upper third molar</td>
</tr>
<tr>
<td>12. Upper canine</td>
<td>12. Upper canine</td>
</tr>
<tr>
<td>13. Lower first pre-molar</td>
<td>13. Lower first pre-molar</td>
</tr>
<tr>
<td>14. Lower lateral incisor</td>
<td>14. Lower lateral incisor</td>
</tr>
<tr>
<td>15. Lower central incisor</td>
<td>15. Lower central incisor</td>
</tr>
<tr>
<td>16. Lower canine.</td>
<td>16. Lower canine.</td>
</tr>
</tbody>
</table>

Flagg adds that, as a whole, the upper teeth are
more liable to decay than lower, the proportion being
eight to five.
Pickerill gives the following table showing the relative liability of the individual teeth to caries

<table>
<thead>
<tr>
<th>TEETH</th>
<th>Average percentage of each carious</th>
</tr>
</thead>
<tbody>
<tr>
<td>First Molars</td>
<td>6.5</td>
</tr>
<tr>
<td>Second Molars</td>
<td>5.1</td>
</tr>
<tr>
<td>Upper Cent. Incisors</td>
<td>3.9</td>
</tr>
<tr>
<td>Premolars</td>
<td>2.8</td>
</tr>
<tr>
<td>Third Molars</td>
<td>2.0</td>
</tr>
<tr>
<td>Upper Canines</td>
<td>1.7</td>
</tr>
<tr>
<td>Lower Incisors</td>
<td>0.7</td>
</tr>
<tr>
<td>Lower Canines</td>
<td>0.5</td>
</tr>
</tbody>
</table>

The Surgeon General reports (quoted by Pickerill) of United States Army for years 1901-3 show a great preponderance of upper teeth requiring treatment compared with lower, the figures are 43,000 upper teeth and 26,000 lower teeth.

Hitchcock (quoted by Tomes) found in a series of 20,000 carious teeth 13,136 upper and 6,864 lower.

Magitot, in 12,000 carious teeth found 7,029 upper teeth and 4,971 lower.

Pare and Wallis (Guy's Hospital Report, 1893) from records of extractions, at Guy's Hospital found 16,410 upper teeth and 13,602 lower in a total of 31,012 carious teeth.

These observations show a much greater liability to caries in the upper teeth than in the lower, but if the Molar teeth only be considered, it is usually found that the preponderance of caries is in the lower ones.
Magitot, for example, gives in his table:

- **First Molars**
  - Upper 1,791
  - Lower 2,127

- **Second Molars**
  - Upper 829
  - Lower 1,282

- **Third Molars**
  - Upper 282
  - Lower 207

or, 3,616 lower and 2,902 upper, an excess of 714 in the case of the lower Molars.

In Pare and Wallis's tables there are:

- **First Molars**
  - Upper 5,259
  - Lower 5,632

- **Second Molars**
  - Upper 2,415
  - Lower 3,489

- **Third Molars**
  - Upper 1,317
  - Lower 1,322

or, 10,443 lower Molars and 8,991 upper, i.e., an excess of 1,452 lower Molars.

In a series of observations made by myself I found in 321 boys (age 12) 237 lower molars and 166 upper molars, and in 313 girls (age 12) 237 lower molars and 145 upper molars.

A study of these figures shows that some of the teeth are much more liable to caries than others, the first Molars are in fact, about twelve times more liable than the lower canines, - according to
Pickerill's figures.

The lower canines and lower incisors are relatively immune to caries, and it is no doubt due to this that the upper teeth as a whole show a great preponderance of caries, for the upper incisors come next the molars in their liability to caries.

Tomes, speaking of the protective factor which helps the lower teeth says, "it is not efficacious at the back of the mouth" and that "this would seem to confirm the idea that it is the secretion of the submaxillary glands that is the protective agency".

CARIES IN ANIMALS.

There is apparently not much information available as to the frequency of caries in animals, but Golzer says "Caries is not rare in the horse, and that it is met with in the dog, and in certain animals kept in captivity, for examples, monkeys and rodents".

Chemical Composition of the Teeth.

As it will be shown that caries of the teeth is essentially a chemical disintegration of the tooth structures, a brief note on the chemical composition of the tissues chiefly concerned - namely the Enamel and the Dentine may not be out of place.

THE ENAMEL, which, of course forms a cap for the portion of the tooth above the gum, has been estimated to consist of:-
Calcium Phosphate ... 69.82 per cent
Calc. Carb. ... ... 4.37 " "
Magnes. Phosph. ... ... 1.34 " "
Calc. fluoride
and other salts ... ... 0.68 " "
Organic Matter. ... ... 3.6 " "

Tomes, however, says that what has been considered Organic Matter is simply water of crystallisation, so that the enamel is practically an inorganic substance. The enamel is covered by a very delicate membrane, called Nasymth's membrane. This membrane will be considered later.

THE DENTINE, is composed of:

( Calc. Phosphate)
Inorganic Matter ( Calc. Carbonate) 62.0 per cent
( Magnes. Phosph. )
Organic Matter ... ... ... ... 28.0 " "
Water of Crystallisation... ... ... 10.0 " "

CHANGES WHICH OCCUR IN THE TOOTH STRUCTURE IN CARIES.

Caries first shows itself on the enamel surface and the appearance varies according to the nature of the surface affected. If it begins on a free surface, the enamel loses its normal polish, then a white or chalky coloured spot appears. If the disease begins in a fissure or depression on the crown of a tooth, a dark coloured spot will be the first indication of its presence. Soon after commencement of decay a more or less pronounced discolouration sets in but according to Miller, discolouration of intact smooth enamel does not occur. Gradual disintegration of the enamel goes on until the Dentine is reached, and as the disintegrating process advances there the dentine is changed into a tough cartilaginous substance.
A translucent zone appears in the dentine between the carious focus and the tooth pulp. Dental pathologists are divided in their opinion as to the cause of this translucency. Some consider it to be due to a vital process - a vital reaction on the part of the dentinal fibrils, causing calcification in the dentinal tubules. Others consider it due simply to decalcification of the matrix surrounding the dentinal tubules.

The following facts cited by Colder are in favour of the translucency being due to changes in the tubule contents, and presumably a vital reaction on the part of the dentinal fibrils.

1. Injury to the dentinal fibrils causes reaction in the pulp leading to formation of adventitious dentine at a point corresponding to commencement of such fibrils on the surface of the pulp.

2. The diameter of the tubules in the translucent zone is distinctly diminished.

3. Stains do not readily affect the translucent zone. Partially decalcified dentine is readily stained with eosine, but the stain has little effect on the translucent zone.

4. Coloured collodion does not penetrate the tubules in the translucent zone so readily as in the normal parts.
If the translucent zone were the result of decalcification one would expect to find it on all sides of the carious cavity, but this is not the case, the only portion of dentine affected being that containing fibrils which have been injured by the carious process.

The translucent zone is absent in artificial caries.

It is not present where caries is progressing in pulpless teeth.

In reference to this translucency Tomes says: "on the whole, then, it remains unexplained; the evidence for decalcification and for calcification are alike weak, that against decalcification being somewhat the stronger".

MICROSCOPIC CHANGES in DENTAL CARIES.

The enamel cuticle (Nasymth's membrane) is found discoloured and cleft in all directions, and closely packed with bacteria, and loosened from the enamel at the decayed points.

The enamel prisms are found loosened, and the spaces between the prisms filled with micro-organisms (these do not penetrate between the prisms of normal enamel). When once the enamel is perforated by decay the destructive process is continued chiefly on its inner surface.
Micro-organisms are found invading the softened dentine, but the softening of the dentine extends further than the invasion of the micro-organisms. The micro-organisms travel more rapidly in the direction of the dentinal tubules than at right angles to them, since in the latter direction they must advance through the fine lateral branches of the tubules. Miller says:

"The action of acid always precedes the invasion of the decay".

**ARTIFICIAL DECAY**

Miller and Underwood attempted to produce artificial decay by incubating a mixture of bread, milk, meat, saliva, teeth, at blood temperature, but found no change in the teeth resembling caries. The mixture was, however, allowed to become putrid. Miller considered this putridity was due to Alkaline decomposition, and therefore, likely to interfere with the success of the experiment. By avoiding certain errors of technique, he was able to produce artificially, destruction of tooth substance "which the most practised microscopist will not be able to distinguish from real decay as it occurs in the human tooth". His experiments were frequently interfered with by the growth of yeast fungi in the bread + saliva mixture. This fungus used up the acid which had been formed by acid producing micro-organisms present in the mixture, and as a result, alkaline decomposition set in.

Miller concludes that his experiments show, among other things, to what an extent the resistance which the tooth opposes to the destroying factors depends upon the structure of the tooth, further, it furnishes an answer to the question why all teeth under the same conditions do not become decayed in the same degree.
A tooth of sound structure, protected by sound enamel, will resist the action of acid many years, whereas a soft, imperfectly developed tooth, under the same conditions, would show decay in the course of a few weeks.

The experiments of Miller appear to leave no room for doubt that caries of the teeth is directly due to the effects of acids on their structure, and that these acids are developed in the mouth, chiefly from the fermentation by micro-organisms of particles of carbohydrate material retained in the mouth.

Miller found that starch paste, and sugar, when treated with saliva, reacts about equally strongly in regard to formation of acid; and that nascent sugar is more rapidly transformed into lactic acid than sugar in its ordinary state.

Glucose, laevulose, and maltose ferment directly into lactic acid, e.g.,

$$C_6 H_{12}O_6 \rightarrow 2 C_3 H_6 O_3$$

Glucose Lactic Acid

Cane sugar is ultimately fermented into lactic acid thus:

$$C_{12} H_{22} O_{11} + H_2 O \rightarrow C_6 H_{12} O_6 + C_6 H_{12} O_6$$

Dextrose Laevulose.

and $$C_6 H_{12} O_6 \rightarrow 2 C_3 H_6 O_3$$ as above

The following table shows the results of one of Miller's experiments.
<table>
<thead>
<tr>
<th>MATERIAL USED</th>
<th>DURATION OF EXPERIMENT</th>
<th>AMOUNT OF ACID UNITS FORMED</th>
</tr>
</thead>
<tbody>
<tr>
<td>Bread, 1 gram</td>
<td>12 and 30 hours</td>
<td>35 and 110</td>
</tr>
<tr>
<td>Sugar, 2.0 g</td>
<td>&quot; &quot;</td>
<td>20 &quot; 41</td>
</tr>
<tr>
<td>Rice, 0.5 g</td>
<td>&quot; &quot;</td>
<td>25 &quot; 72</td>
</tr>
<tr>
<td>Macaroni, 0.5 g</td>
<td>&quot; &quot;</td>
<td>20 &quot; 76</td>
</tr>
<tr>
<td>Meat</td>
<td>&quot; &quot;</td>
<td>0 &quot; 3</td>
</tr>
<tr>
<td>Meat (raw)</td>
<td>&quot; &quot;</td>
<td>-- &quot; 5</td>
</tr>
<tr>
<td>Fish</td>
<td>&quot; &quot;</td>
<td>0 &quot; 5</td>
</tr>
<tr>
<td>Eggs</td>
<td>&quot; &quot;</td>
<td>0 &quot; --</td>
</tr>
<tr>
<td>Cheese</td>
<td>&quot; &quot;</td>
<td>0 &quot; (?)</td>
</tr>
<tr>
<td>Spinach</td>
<td>&quot; &quot;</td>
<td>0 &quot; 0</td>
</tr>
<tr>
<td>Potato (raw)</td>
<td>&quot; &quot;</td>
<td>0 &quot; 0</td>
</tr>
<tr>
<td>Salad (raw)</td>
<td>&quot; &quot;</td>
<td>-- &quot; (?)</td>
</tr>
<tr>
<td>Fat</td>
<td>&quot; &quot;</td>
<td></td>
</tr>
</tbody>
</table>

4.0 c of fresh human saliva used in each mixture.

An "Acid Unit" = quantity of acid necessary to neutralize.

\[ 0.1 \text{ c of a 0.5 per cent solution of KOH.} \]

**ACTION OF MOUTH BACTERIA UPON CARBOHYDRATES.**

Of 22 kinds of mouth bacteria examined by Miller, 16 soon brought about an acid reaction when cultivated in beef extract—peptone—sugar solution. Four produced an alkaline reaction under the same conditions, and with 2 the reaction remained neutral. It was found impossible to draw a sharp line between those bacteria which produced an acid reaction, and such as cause an alkaline reaction; because, in some cases, the reaction was very weak, and changed in course of the experiment; while in others it was materially influenced by change in the amount of sugar.

In the case of 10 out of 18 different bacteria it was found that the acid formed was lactic acid.
Batyric acid, formic acid, acetic acid, etc., are (of-ten) produced in lactic acid fermentation of carbohydrate, but these are probably formed in very small quantities in the mouth, so that they can play no very important part in the various processes going on in the mouth.

Albuminous material exposed to the action of mouth bacteria undergoes putrefaction. The process is accompanied by an alkaline reaction. Mixtures of albuminous material will yield, under the influence of mouth bacteria, an alkaline, or an acid reaction, according as the albuminous material or sugar is in excess.

Goadby, in section dealing with the Bacteriology of Dental Caries in J.F. Colyer's Dental Surgery Pathology, classifies the organisms concerned in dental caries, thus:-

(a) Acid-forming Bacteria - capable of producing acid by the fermentation of carbohydrates.

(b) Liquefying Bacteria, which either by their own action, or by the production of proteolytic enzymes, cause digestion of the decalcified dentine.

Goadby, has also shown that other organisms, which are neither proteolytic, nor acid producing, form colonies of a gelatinous consistency which adhere firmly to the surface of the tooth. Where these colonies are allowed to accumulate, gelatinous plaques may be formed.
The carbohydrate fermenting organisms can grow symbiotically with these plaque forming organisms, and the acid which the former produce diffuses through these plaques, which thus act as an osmotic membrane.

The part, however, played by these plaques in the production of caries is very much debated by those who have carefully studied the subject, some denying that the plaques are at all an essential feature of caries. Pickerill believes that the source of some of these plaques found on the teeth is to be found in the precipitation of mucin by acid substances taken into the mouth, and also by the evaporation of carbon dioxide from stagnant saliva.
Having shown that, according to Miller, caries of the teeth is a chemico-parasitic process, it seems to me this is a proper stage to give a brief history of the development of knowledge of the subject, as one is now more able to appreciate the work of those who have given consideration to it, although, on other grounds, it would have been more orderly to have begun with the history of the subject.

Morsman, in Dental Cosmos, 1885 p.530 etc., gives an interesting contribution to our knowledge of the history of the etiology of Dental Caries.

The following is based chiefly on his paper.

Hippocrates and Herophilus ascribed toothache to a bad condition of the humours.

A.D. 40 Celeus gives no description of caries, but prescribes the application of aromatic substances to the tooth, and emollients for external use. He also advises systemic treatment, and gives directions as to diet.

A.D. 131 Galen gave a formula for a mixture to relieve pain in hollow teeth. Galen observed that there was some existing connection between the stomach and the teeth, for he said, "A lack of nutrition made the teeth thin, brittle and weak" (Harvey. D. Cos. 1899, p949)

A.D. 636 Paul of Aegina said "Indigestion is the cause of tooth decay". He treated it from a clinical standpoint, believing the acid from the vomit caused the destruction of the teeth. (Harvey. D. Cos. 1899).
Giovanni of Arcoli drew up a decalogue for the prevention of tooth decay. Avoid eating indigestible substances, vomiting, sweet and sticky food, hard substances, food which sets teeth on edge, food too hot or too cold, etc., (Pickerill).

E. C. Kirk, in British Dent. Journal, Dec. 15th 1913 quotes from an old German work published anonymously in 1530, the original work being in his own possession. The translation given is: "Caries is a disease and defect of the teeth in which they become full of holes and hollow, which most often affects the molars, especially if one eats and does not clean them of the adhering food, which decomposes, producing a bad acid moisture which eats them out, increasing continually little by little so that it destroys the teeth entirely".

A book entitled "Zahnmedizin" published in Frankfort.

In Dent. Cosmos. 1887, p 1. there is an account of a book published in Frankfort thus:

(Frankfort - Chr. Egenolff. 1541)

"Chapter II. The various causes of bad teeth. Care should be taken that no fragments of the victuals remain within them, but also to wash and cleanse them with pure water.

Indigestion produces vapours, these rising from stomach injure the teeth materially.

Slimy and sticky food should be avoided, for it adheres to the teeth, and causes much detriment."
Hot victuals burn the teeth, over cold food or drink also injure the teeth; likewise beware of quicksilver, or salves in which there is quicksilver.

Neither should we go to sleep soon after a heavy meal for this will also damage the teeth."

Chapter XIII. - How to save the Teeth.

Tooth powders recommended, myrrh, alum, etc., and "finally, always after eating wash the mouth with wine or beer".

1548 1548, Ryff, wrote a treatise on the Eye, Eyesight and Teeth - teaching unknown.

1561 1561, Ambrose Pare - regarded teeth as bones and caries in both identical.

1678 1678, Leuwenhoeck made discoveries as to the anatomy of the teeth - a great stimulus to study of caries.

1728 1728 Fauchard distinguished several forms of caries, and assumed an internal and an external cause. He supposed the internal cause to be acrid lymph.

1757 1757 Pourdet stated that when fluids in the dental vessels are too thick they putrefy and act on the teeth.

1778 1778 John Hunter thought that decay deserved the name mortification, but said "I am apt to suspect that during life there is some operation going on which produces a change in the diseased parts.

1796 1796 S. K. Mitchell demonstrated the existence of an acid in the mouth capable of decomposing the teeth.
1806. Fox propounded the **Inflammatory Theory**, and says "Caries differs from the same disease in bone only in that the teeth do not possess sufficient living power to effect the process of exfoliation."

1821. L. S. and E. Parmby first proposed (in U.S.A. at least) the **Chemical Theory**.

1835. Robertson of Birmingham says "The only cause capable of explaining the partial operation, and the particular situations, of decay, is the corrosive or chemical action of the solid particles of food which have been retained and have undergone a process of putrefaction, or fermentation, in the several parts of the teeth best adapted for their reception". This is really the **first definite enunciation** of the chemical theory Morsman adds.

1838. Regnard held same views and issued a pamphlet on the subject.

1840. Koecker advanced a theory of contagion.

1843. Westcott demonstrated experimentally the action of citric and malic acids and also sugar upon the teeth.

1846. Desirabode maintained the dual character of Caries - a cause acting from within and one from without.

1846. Linderer scouts idea of inflammation and advances the purely chemical theory.

1846. Fichimus considered caries had its origin in the cuticle of the enamel. He distinguished a putrefactive process caused by infusoria which he called
denticolae; described process as extending from the enamel membrane to the enamel and thence to the dentine.

1859. Tomes in first edition of Dental Surgery maintained a chemico-vital theory. The translucent zone in the dentine he believed to be protective, demonstrated also the formation of secondary dentine.

1860. Neuman agrees with Tomes as to resistive power of dentines, but regarded consolidation of dentinal tubules to be thickening of the sheaths at the expense of basic substance. He considered caries to be a veritable "odontitis", and thought, the distinction between caries of bone and caries of teeth purely formal.

1862. Cudet advocated theory of internal caries in which the dentine was the tissue primarily affected, but believed an external cause might also exist.

1867. Magitot elaborated a theory of resistance and consolidation of dentine, but eliminated the idea of inflammation. Cause he said was strictly chemical, made some experiments on artificial caries.

1868. Leber and Rottenstein demonstrated the presence of leptothrix granules in the dentinal tubules. They denied existence of any reaction in the dentine, believed fungi to proliferate in the tubules and to expand them, thus aiding the disease; and that caries can be produced artificially out of the mouth. Considered caries of enamel to be due largely to a chemical cause.
1868 Bridgeman propounded an electro-chemical theory. He was awarded a prize for his essay.

1870 Wedl considers caries due to abnormal secretions of oral membranes and salivary glands, and in consequence of fermentation of these secretions, acids develop and act upon the teeth. He verified in part experiments of Leber and Rettenstein and said: "There are no grounds for ascribing changes in the dentinal fibrils to an inflammatory reaction in the pulp".

1871 Mummery made an elaborate investigation as to frequency of caries in different races.

1873 Tomes in 2nd edition of his Dent. Surgery abandons all idea of vital action as part of caries, and regards calcification of the fibrils as doubtful.

" 1873 Spence Bates attributes caries to carbonic acid in abnormal quantities acting in a nascent state.

1878 Frank Abbott believes cement, dentine, and enamel, to be traversed by a network of living matter and that there is a reaction against advance of caries.

1880 Weil taught that leptothrix bores its way directly through Nasymth's membrane and then enters the enamel.

1881 Underwood and Mills believe that caries depends on micro-organisms.
1882 Miller, W.B. enunciated the parasitico-chemical theory. This is the theory now generally accepted, though it is criticized by Von Beust (see below).

1910 Hopewell-Smith's theory of abrasions of enamel cuticle plus a specific micro-organism.


This observer considers Miller's theory is based chiefly on test-tube experiments. In some of his own experiments he has shown that freshly extracted tooth, placed with its apex in a solution of fuchsin, can carry this solution to its periphery by means of its capillaries - and in a short time. He says his experiments in demonstrating the conductability of the smaller enamel tubuli, have given tangible shape to the long suspected assumption that a process of metabolism is possible in the enamel. He further says "Excluding a few unimportant reservations, we must assume that caries is a disease which has its origin within the teeth."

1913 Kirk advances theory of a dissolved carbohydrate in the saliva as a cause of caries. Had also referred to it in 1902.
PREDISPOSING CAUSES OF DENTAL CARIES

Since the work of Miller and others has been made known, there is general agreement as to the direct causes of dental caries. The predisposing causes have now to be considered. These are conveniently studied under three heads, viz: -

1. STRUCTURE of the TEETH.
2. IRREGULARITY in POSITION of the TEETH.
3. CAUSES LYING OUTSIDE the TEETH.

1. Influence of the structure of the teeth as a predisposing cause of caries. Sandill says: "The one dark spot in our knowledge of the remote causation of caries, is the inherent structural weakness of the tissues."

A tooth, consisting as it does, chiefly of dentine, covered for the most part with a layer of enamel, which in its turn is covered with the enamel cuticle, - a thin but tough membrane, called Nasymth's membrane, it would be, perhaps, the most logical method to begin the consideration of the structure of the teeth by a study of Nasymth's membrane. It is, however, more in accordance with the historical development of the study of the subject of caries to begin with the Dentine.

Black appears to have been the first to make anything like an elaborate examination of the dentine. Considering that the dentine is the third line of
defence against caries - counting Nasymth's membrane as the first - Black's work is relatively unimportant. He says, however, "we cannot gain anything by the determination of the specific gravity of the tooth as a whole, for the reason that it is made up of four tissues - enamel, cement, dentine, and pulp, the relative amounts of which are different in different teeth. It therefore becomes imperative that each tissue be examined separately, or that a single tissue be made the basis of the examination. I have chosen to confine the examination for density, proportion of water, lime salts, and organic matter, to the dentine, as best expressing the character of the tooth as a whole."

Small blocks of dentine were prepared by special apparatus. These were subjected to pressure, and crushing stress noted, and also the strain - or amount of change of form.

Black sums up the results of his observations, thus:

1. The teeth are strongest in youth and early adult age, diminishing somewhat in strength with advancing age.

2. Teeth that have lost their pulps and have become discoloured, lose strength in a marked degree, apparently from a deterioration of the organic matrix.

3. Teeth that have become badly worn from mastication, and in which the pulps become so much calcified as to cut off the nutrition of the crown portions of the dentine, lose strength apparently from deterioration of the organic matrix.
4. Teeth of old people, and especially those in which much calcification of the pulp occurs, deteriorate in strength.

5. Differences in density, or in the percentage of lime salts in the teeth is not the controlling factor in the strength of the teeth, nor their hardness, this seeming to depend on the condition of the organic matrix.

6. Differences in strength of the teeth have no influence as to their liability to caries. Differences in the density, or in the percentage of lime salts in the teeth, have no influence as to their liability to caries.

7. Imperfections of the teeth, such as pits, fissures, rough or uneven surface, and bad forms of interproximate contact, are causes of caries only in the sense of giving opportunity for the action of the causes that induce caries.

Miller (Dent.Cos.1903, p 696) found that there is a very great difference in the resistance which dentine from the teeth of different animals offers to the action of acids, and that the difference was in favour of hard dentine, but that hard dentine is not immune, only relatively so.

It is, however, in the enamel that the chief resistance is to be found; the physical features of the enamel are therefore most important.

THE ENAMEL IN RELATION TO CARIES.

In a series of experiments undertaken by Miller to determine whether defects in the form and structure of the tooth influenced the process of caries, he found that abrasions, bruises, cracks, and other defects of the enamel, very sensibly diminished its resistance. He also found that the external intact smooth enamel surface offers a most stubborn resist-
ance to the action of weak acids, and far greater resistance than the internal surface of a cavity in the enamel. The most elaborate investigations dealing with the structure of the enamel are those of Pickerill, and described in his book - The Prevention of Dental Caries. Over 1,000 teeth of civilised and savage races were examined minutely, microscopically, and physically. The teeth were divided into three kinds, namely

1. Native Teeth - but little if at all afflicted with caries.

2. Sclerotic Teeth - characterised clinically by hardness, yellowish colour, and relative immunity to caries.

3. Malacotic (malakos = soft) Teeth - characterized clinically by softness, whiteness, and susceptibility to caries.

He allows that Black's and Tome's researches appear to go to negative such classification as having any relation to caries, but considers the classification convenient.

The enamel surface was cleaned with xyIol and alcohol or ether, and afterwards "stained" by gently rubbing powdered charcoal over the surface. In this way any elevations or depressions on the enamelled surface would be easily identified on examining the surface microscopically in reflected light, for the elevations would be left white and the depressions black. Such examination shows that the enamel surface is normally furrowed at right angles to the axis of the tooth, thus:
Only one tooth of those examined failed to show these ridges and furrows.

The ridges are widest and the furrows deepest at about 2/3 of the distance between the neck and occlusal surface of the tooth. They are less frequent towards the occlusal surface than at the neck, where they are also finer and less marked. Microphotographs show that there is a very decided difference in the surface markings of Maori (native) teeth and clinically soft teeth. Diagramatically the ridges and furrows may be shown thus:

<table>
<thead>
<tr>
<th>Malacotic (soft) tooth</th>
<th>Maori Tooth</th>
</tr>
</thead>
<tbody>
<tr>
<td><img src="image1" alt="Diagram of Malacotic Tooth" /></td>
<td><img src="image2" alt="Diagram of Maori Tooth" /></td>
</tr>
</tbody>
</table>

In addition to the ridges and furrow, fissures of different types are found on the enamel. These may be triangular, sinuous in shape, and they may be deep. Some of the fissures may be traumatic in origin, but others are no doubt due to developmental defects. These features are all more marked in soft teeth.

**Permeability of Enamel**

Experiments were made to ascertain the permeability of the enamel surface by exposing teeth to the action of silver nitrate in 10% solution for 24 hours in the dark. It was found that the penetration was about 4 times greater in malacotic teeth than it was in the Maori teeth, the sclerotic teeth coming about midway in permeability.

The following table shows that the degree of
penetration varies with the time the tooth has been erupted: - (P.42)

<table>
<thead>
<tr>
<th>TEETH</th>
<th>AMOUNT of PENETRATION of 10% AgNO₃</th>
</tr>
</thead>
<tbody>
<tr>
<td>Unerupted 3rd Molar</td>
<td>0.83 mm</td>
</tr>
<tr>
<td>Erupted 3rd Molar (about 2 years use)</td>
<td>0.42 mm</td>
</tr>
<tr>
<td>Premolar sclerotic type, 11 years use</td>
<td>0.18 mm</td>
</tr>
</tbody>
</table>

There is comparative equality of penetration in the unerupted molars of both Maori and European teeth. Pickerill considers that this would seem to point to the fact that the cause of subsequent differences in permeability of various enamels is not entirely a developmental one, but rather a property acquired largely after eruption.

He compares a second permanent molar of a Maori with a premolar of a European, and shows that in two years, the Maori tooth has become as impermeable as the European has in eleven years.

Since the permeability of the teeth diminishes inversely with the time they have been erupted, he considers that the logical conclusion is, that the opinion that enamel once formed, cannot possibly undergo any physiological alteration is untenable.

In view of these results obtained by Pickerill, it would seem that Wallace's statements (J. S. Wallace, Prevention of D. Caries, 1906, P.91) would require to be modified. In speaking of the physical nature of the food stuffs of the present day, Wallace says "One
seldom sees (in American literature) any reference whatever to the physical nature of the food, except perhaps, to maintain the old delusion that it is on account of the soft food of the present day that the teeth are not so perfectly calcified and resistant to the carious process as was the case in the time of our primitive ancestors. We, of course, as odontologists, know, that the crown of the tooth is formed under the gum and quite independently of functional activity, that the enamel is not strengthened by wear." He also says (P.18)"The vulnerable part of the crown is invariably formed before the tooth has cut the gum and before the child has reached the modern schoolroom. They thus form an exception to the rule that an unused member will be less perfectly developed than one constantly used. Moreover, assuming that the pressure and strain of mastication did cause a more lively circulation of the blood in the periosteum and pulp and on that account induced an increased deposition of lime salts, this could have absolutely no effect on the enamel, which is an inorganic substance, formed once for all in early childhood."

**EFFECT OF HEAT ON THE ENAMEL.**

Pickerill found that heating the enamel in an electric furnace very appreciably diminished its permeability to the silver nitrate. This was probably due to the destruction of organic matter in the enamel,
since silver nitrate is a selective stain for organic intercellular substance, and, therefore, had stained organic matter in the enamel. He adds, however, that adsorption and precipitation may account for the appearances given with the silver nitrate.

In regard to the effect of heat on the enamel, it may be mentioned here, that the drinking of hot fluids has been cited by some as an important cause of caries.

In regard to the presence of organic matter in the enamel, attention may be drawn to a paper by C. F. Boedecker in Dental Cosmos. 1911 p.1000, on Nutrition of the enamel of the Human Tooth. Boedecker decalcified specimens of enamel in 5 to 10 per cent nitric acid in celloidin, the celloidin in the mixture had as nearly as possible the same specific gravity as that of organic matter assumed to be present. The celloidin was found to support the delicate organic matter when robbed of its calcium support. Microphotographs were taken in ultra-violet light. In these, organic matter is seen penetrating the enamel from the dentine and continuous with the dentinal fibres. Lamellae are also seen connecting Nasmyth's membrane on one side with the dentine on the other. Boedecker also refers to the aid of cataphoresis in demonstrating the presence of delicate "protoplasmic channels" in adult enamel and that "these roads of transportation" are more numerous and larger in young enamel than in older specimens.
The same observer says that the greatest amount of organic matter is found near the dentinal margin, the outer portion has less, while the middle portion has still less. If the permeability to silver nitrate is due to the presence of organic matter in the enamel, it is difficult to explain why the internal enamel surface, at the amelo-dentinal junction, is apparently not permeable, according to Pickerill, if the greatest amount of organic matter is found near the dentinal margin of the enamel. However, the permeability experiments shows that the enamel surface is permeable to solutions of silver nitrate, and therefore presumably to solutions of acid substances.

THE DENSITY OF ENAMEL.

On investigating the density of the enamel, it was also found that Native enamel was the most dense, and the enamel of Malacotic teeth the least dense, and there was evidence that the density of healthy enamel bears a direct ratio to the time which has elapsed since its formation.

OTHER PROPERTIES OF ENAMEL.

Pickerill made other investigations and the conclusion he arrived at was, that the enamel of teeth varies appreciably as to the surface structure, density, hardness, permeability, and solubility in acid, and that these variations bear a distinct relationship to the clinical classification of teeth into sclerotic and malacotic (soft) types.
We have seen (v a P.36) that Miller found that defects in the structure of the enamel, such as bruises and cracks, etc., very sensibly diminished the resistance of the enamel to decay. One must add, however, that some observers attach comparatively minor importance to these defects, for example, Leon Williams (Dent. Cos. 1898 P528) says: "In connection with the supposed relation between the presence of bacteria at the commencement of decay and congenital defects in the enamel I may call attention to the fact that bacteria are most frequently found to be acting upon enamel where this tissue is usually most perfect. . . . . . Bacteria undoubtedly avail themselves of defects where they exist, but at the same time, they act quite independently of all defects." The sheltered situation of an approximal surface, with enamel of perfect structure, is evidently a more favourable condition for decay than almost any form of defect upon the lingual or buccal surface" and that "although there are marked variations in the resistance of different enamels to cutting instruments, these variations bear no necessary relation to decay or immunity from decay"

In view of Miller's work and also the work of Pickering, there can, however, be no doubt that structural defects favour the incidence of caries, although these defects may be only one of many factors concerned in the production of caries. It would, therefore, be an advantage if any cause could be shown which favours
structural defects in the teeth so that steps could be taken with a view to its elimination. On this point I have no positive evidence to being forward, but it occurred to me that the method of infant feeding might have some definite relation to dental caries, and that probably the teeth of children who have been fed during infancy, that is, during the formative period of the teeth, on breastmilk might have teeth of a sounder character than have those children who had been fed on substitutes for mothers' milk.

I was not aware that the subject of Infant Feeding had previously been considered in relation to the incidence of dental caries. I have, however, found some references to the subject in dental literature.

In the Medical Press and Circular Vol.II. 1899, there is an abstract of a paper read by Dr. Kingston-Barton in which Dr. Barton gives the results of his observations - extending over twenty years - on Early Decay of the Teeth. He says:-

1. Breast-fed children have always the best teeth.

2. Children fed on cow's milk, asses' milk, goats' milk had the second best.

3. Children brought up on patent foods, have the worst teeth.

In 100 cases which he examined, he found that 75% of those with good teeth had had some form of breast feeding. He adds that if a person between 20 and 50 years of age have good teeth it will nearly always be found that he has had a period of good infant suckling, followed by very simple feeding in the first few years
of life. More figures would have made Barton's statements more convincing, but it is evident that he has no doubt whatever that children who have been breast-fed have, as a rule, the best teeth.

Frick, in a paper read at the Third International Dental Congress, and quoted in Dental Cosmos, 1901, p. 57, says "The frequency of Dental caries has increased considerably since fifty years ago, especially in certain localities. As it has not been demonstrated that the immediate exciting causes of dental caries are more active, and as it is probable they are not, judging from the way in which the hygiene of the mouth is observed, we must conclude that it is the predisposition that is more frequent." He then goes on to give his statistics which comprise "more than 200 cases." The patients selected were all under 25 years of age, and were drawn from Swiss, Germans, French and English. The average amount of bad teeth in the breast-fed was 1.58, that for the hand-fed was 4.16. He says "if we separate the children under ten years of age, the difference is greatly increased. Out of 12 breast-fed children only one had some of the deciduous and the first permanent molars decayed. Out of 21 artificially fed children 19 had decayed deciduous teeth and first permanent molars." He deduces that the quality of the teeth is improved in direct relation to the period of time that the infant enjoys natural nourishment.
In the discussion which followed the reading of this paper Dr. Vian said that he had observed similar facts to those mentioned by Dr. Frick.

Dr. Pouisot said that the changes in the teeth and the disturbances in general health originate in lactation.

Other speakers supported Frick's observations.

Michael (British Journal of Dental Science, Vol.47, p391 - 1904) gives an account of his examinations in public schools of Wurzburg in the preceding five years. He examined the teeth of 11,762 children (6,116 females and 5,646 males). He noted the total number of teeth examined together with the total number of carious teeth found, and the amount of carious teeth was 19.02 per cent. Of these, 11,762 children, 7,763 had been fed on the breast. These showed caries to the extent of 11.46 per cent, that is, about 7.5 per cent less than the average frequency.

The average time of nursing was 6.5 months.

He found only 122 children who had been nursed for more than ten months. These showed only 9 per cent of caries.

Wheatley studied the condition of the teeth of children attending schools in Salop in reference to breast-feeding and hand-feeding.

In year 1909 he found the average amount of carious teeth in breast-fed children, age 12-13, was 4.5 per cent, and in the bottle-fed 4.9 per cent.
The subject was further studied in following year and following table drawn up:

**BOYS and GIRLS AGE 5 - 6.**

<table>
<thead>
<tr>
<th>BREAST-FED CHILDREN</th>
<th>Up to 9 months</th>
<th>10 -12 months</th>
<th>13 -18 months</th>
<th>19 Mons</th>
<th>TOTAL</th>
</tr>
</thead>
<tbody>
<tr>
<td>Number examined ...</td>
<td>132</td>
<td>373</td>
<td>467</td>
<td>276</td>
<td>1248</td>
</tr>
<tr>
<td>Average Number of decayed teeth per child ...</td>
<td>6.2</td>
<td>5.7</td>
<td>5.9</td>
<td>7.0</td>
<td>6.2</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>Number examined ...</td>
</tr>
<tr>
<td>Average Number of decayed teeth per child ...</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>BOTTLE-FED CHILDREN</th>
<th>Up to 9 months</th>
<th>10 -12 months</th>
<th>13 -16 months</th>
<th>19 Mons</th>
<th>TOTAL</th>
</tr>
</thead>
<tbody>
<tr>
<td>Number examined ...</td>
<td>80</td>
<td>298</td>
<td>267</td>
<td>126</td>
<td>771</td>
</tr>
<tr>
<td>Average Number of decayed teeth per child ...</td>
<td>6.3</td>
<td>6.4</td>
<td>6.8</td>
<td>8.1</td>
<td>6.9</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>Number examined ...</td>
</tr>
<tr>
<td>Average Number of decayed teeth per child ...</td>
</tr>
</tbody>
</table>
Wheatley also compared the children of Town Schools with Country Schools and found results shown thus:-

<table>
<thead>
<tr>
<th>TOWN SCHOOLS</th>
<th>COUNTRY SCHOOLS.</th>
</tr>
</thead>
<tbody>
<tr>
<td>Breast-fed</td>
<td>Bottle-fed</td>
</tr>
<tr>
<td>Number of decayed teeth per child ...</td>
<td>6.82</td>
</tr>
<tr>
<td></td>
<td>5.38 &quot; 12</td>
</tr>
</tbody>
</table>

These figures show a difference in favour of the breast-fed children, but the difference is not very great.

**OBSERVATIONS MADE BY MYSELF ON THE CONDITION OF THE TEETH IN BREAST-FED, AND IN HAND-FED SCHOOL CHILDREN.**

As the scheme of medical inspection under which I was working included only those children who were either in their eighth, or their twelfth year of age, I confined my attention at first to children at the older age period, in order to minimise, as far as possible, the effects of changes in the condition of the teeth due to the second dentition. Later on, however, I noted the condition of the teeth at the eighth year also. It was only in those cases where a parent attended at the inspection that the necessary information could be obtained. A large number of parents don't attend the inspection, so that the number of children on whom observations were made was not so large as it would have been had a parent attended in all cases. The observations extended over a period of nearly two years.
The following table summarises my results:

### BOYS (in TWELFTH YEAR of AGE).

<table>
<thead>
<tr>
<th>BREAST-FED</th>
<th>BOTTLE-FED</th>
</tr>
</thead>
<tbody>
<tr>
<td>Number examined</td>
<td>Average number of decayed teeth per child.</td>
</tr>
<tr>
<td>118</td>
<td>1</td>
</tr>
</tbody>
</table>

### GIRLS (in TWELFTH YEAR of AGE).

<table>
<thead>
<tr>
<th>BREAST-FED</th>
</tr>
</thead>
<tbody>
<tr>
<td>Number examined</td>
</tr>
<tr>
<td>209</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>BREAST-FED</th>
<th>BOTTLE-FED</th>
</tr>
</thead>
<tbody>
<tr>
<td>Number examined</td>
<td>Average number of decayed teeth per child.</td>
</tr>
<tr>
<td>65</td>
<td>1.55</td>
</tr>
</tbody>
</table>

### CHILDREN IN EIGHTH YEAR of AGE.

<table>
<thead>
<tr>
<th>BREAST-FED</th>
<th>BOTTLE-FED</th>
</tr>
</thead>
<tbody>
<tr>
<td>Number examined</td>
<td>Boys</td>
</tr>
<tr>
<td>80</td>
<td>157</td>
</tr>
<tr>
<td>Number of decayed teeth per child</td>
<td>2.42</td>
</tr>
</tbody>
</table>

that is, a ratio of 84.2 for breast-fed to 100 for bottle-fed in boys.

and, a ratio of 75.1 for breast-fed to 100 for bottle-fed in girls.

Taking the children of both sexes and of the two age groups together, there are 564 who were breast-fed and 178 who were bottle-fed.
The total number of decayed teeth in the breast-fed was 965, or 1.71 per child; the corresponding figures for the bottle-fed were 434 and 2.43.

This gives a ratio of about 70 for the breast-fed children to 100 for the bottle-fed children.

I think, therefore, there is sufficient evidence to warrant the belief that children who have been fed on the breast are not so liable to caries of the teeth as those who have been bottle-fed. This may be because breast-fed children have as a rule better health than hand-fed children, due to their comparative freedom from gastro-intestinal disturbances, so that their body development — including of course the teeth — is likely to proceed more evenly and not be so subject as hand-fed children are, to periods during which it is interfered with. This diminished incidence of dental caries in breast-fed children may, however, be due to better assimilation of mothers' milk and hence better bones and teeth be the result. It is well-known that breast-fed children are not nearly so subject to rickets as hand-fed children are, and although it is not quite clear if there is any relation between rickets and dental caries, it is reasonable to suppose that the structure of the teeth will be affected, as the bones are in rickets, by the character of the food, quite apart from any digestive disturbances the food may give rise to.
If there is an increase in the defects on the surface of the enamel, there would naturally be a greater predisposition to caries. No positive evidence is at hand to support the suspicion that the enamel surface is less perfect in hand-fed children, but it seems likely that this is so, or at any rate that the quality of the enamel is in some way less resistant to the attacks of the exciting causes of caries.

THE ENAMEL CUTICLE IN RELATION TO CARIES.

So far the structure of the teeth has been considered mainly with reference to the dentine and the enamel, but to complete the consideration, attention must now be given to the enamel cuticle or Nasmyth's membrane. Nasmyth's membrane is a delicate membrane which covers to a greater or lesser extent, the enamel of all teeth. It is apparently resistant to the action of acids to an extraordinary degree. Miller found that a tooth may remain for months in fermenting solutions and enamel undergo considerable decalcification, without losing its enamel cuticle. He considered the resistance of the enamel was due in part to the protective action of its cuticle.

Hopewell-Smith (Transactions of School Dentists Soc. Vol IV. No IV.) assigns a very important rôle to this membrane in its determining the commencement of caries at points of the tooth surface where it is difficult, if not impossible, for the lodgment of food-stuffs to take place.
He says it is present in teeth even in persons over 70 years of age. It exists as a thick covering over the crowns, passes over the cusps, and into the pits and fissures, and is attached firmly to the neck of the teeth.

It is, however, lost in parts over the cusps or cutting edges early in the life history of each tooth. The cusps, however, are the most automatically cleansed of all the surfaces of the crown. After a breach of the membrane micro-organisms pass beneath its torn edges to a suitable site, where, under suitable conditions, a nidus is produced. If they are ordinary bacteria, nothing happens, but if a specific micro-organism or group of micro-organisms enters under this torn edge, then caries results.

Hopewell-Smith's contentions are practically summed up in these words: - "If you will admit the protective action of Nasmyth's membrane as the first line of defence; if you will admit that it exists for a definite purpose as a tangible structure, only yielding to the action of mechanical agencies; if you admit that a special micro-organism - an X bacterium - exists also, which at present is unrecognisable and uncultivable - although it may be Bac-necrodentalis or Streptococcus brevis or staphylococcus albus - all the problems connected with the etiology of caries vanish. All predisposing causes go by the board, heredity, structural defects, etc., only producing a greater rapidity
in the progress of the attack, and not its initiation."

The comment one might make on this is that though it may be true that caries owes its initiation to a special micro-organism, or group of micro-organisms, this does not explain why caries is more prevalent among modern civilised races than it was, apparently, among ancient and certain uncivilised races, as has been previously shown.

2. IRREGULARITIES IN POSITION OF THE TEETH AS A PREDISPOSING CAUSE OF CARIES.

When teeth are irregular in position, food debris is more likely to collect about them, and, for the same reason, is more likely to remain longer in contact with them, owing to the difficulties in the way of the self-cleansing forces at work in the mouth. Such teeth will be more exposed to the action of acid produced by the fermentation of carbo-hydrate debris by the micro-organisms of the mouth. Mouths free from caries, even where the teeth are irregularly placed, are not uncommon, but speaking broadly, teeth, irregular in position are more liable to caries. In regard to this irregularity it would seem there is a tendency for it to increase in modern civilised races. Carter (quoted by Colyer) says: "There is a general concensus of opinion in favour of the view that the jaws of modern races are becoming smaller, the diminution in
size being greater in the jaws than in the teeth.

Talbot (quoted by Tomes and Nowell), says: "On the whole the higher the intellectuality the more numerous the deformities, and that these deformities are more numerous among the better classes than among the poorer."

Mummery also commented on the rarity of irregularities of the teeth of savage races. As Tomes and others have pointed out, Natural Selection may have to do with the frequent occurrence of undersized jaws in well-to-do people, for in the human female the oval, tapering face, with small mouth is the beautiful type, and this type of face does not afford much room for ample dental arches. On the other hand the face with prominent jaw development is less attractive.

It is also reasonable to suppose that the character of modern food stuffs may have something to do with these irregularities, since, owing to its softness, but little demand is made on the masticatory muscles, with a consequent want of full development of the jaws.

METHOD OF INFANT FEEDING IN RELATION TO IRREGULARITIES IN POSITION OF THE TEETH.

In order to determine whether the method of infant feeding had anything to do with maldevelopment of the jaws J. F. Colyer carried out a series of measurements in children free from adenoids, and
obtained the following data:

<table>
<thead>
<tr>
<th></th>
<th>Distance between fixed points on the 1st premolar or the 1st deciduous molar.</th>
<th>Distance between fixed points on the first permanent molar.</th>
</tr>
</thead>
<tbody>
<tr>
<td>Breast-fed</td>
<td>26.31 mm</td>
<td>38.85 mm</td>
</tr>
<tr>
<td>Hand-fed</td>
<td>25.61 mm</td>
<td>34.1 mm</td>
</tr>
</tbody>
</table>

He also compared the width of the palate with the height and found figures for breast-fed and hand-fed as follows:

Breast-fed ... 2.63
Hand-fed ....... 2.52

These figures were taken from patients in private practice, and Colyer considers Hospital patients would show a greater difference in favour of the breast-fed children.

In 41 patients where the old-fashioned tube bottle had been used the average measurements were:

23.05 mm between fixed points on 1st premolar or 1st deciduous molar.
32.14 mm between fixed points on 1st permanent molar.

It seems probable that the character of the teat is mainly responsible for this difference in measurements of the jaws in hand-fed and breast-fed children. A teat shorter made of harder rubber, and broadening towards the base would seem to be a better imitation of Nature. Such a teat would necessitate more action of the mandible than the type of teat in general use. With a long and soft teat, much sucking action goes on in which the cheek muscles are constantly...
acting towards the median line, and not away from it as is the case when the child is feeding on the breast. The breast-feeding child gets the milk as much by expression, by movements of the lower jaw, as by suction.

Nasal obstruction arising from presence of adenoids may influence the development of the maxilla, but on the other hand, the faulty development on the maxilla due to insufficient exercise of the masticatory muscles may predispose to mouth breathing and adenoids. A vicious circle may be established.

Colyer made a series of observations on breast-fed and hand-fed children to determine whether the presence of adenoids affected the width of the palate or not.

In case of breast-fed children he found:

<table>
<thead>
<tr>
<th>Distance between fixed points on first premolar or first deciduous molar</th>
<th>Distance between fixed points on 1st permanent molar</th>
</tr>
</thead>
<tbody>
<tr>
<td>Without Adenoids</td>
<td>26.31 mm</td>
</tr>
<tr>
<td>With Adenoids</td>
<td>25.19 mm</td>
</tr>
<tr>
<td>Difference</td>
<td>-1.12 mm</td>
</tr>
</tbody>
</table>

In case of hand-fed children he found:

<table>
<thead>
<tr>
<th>Distance between fixed points on first premolar or first deciduous molar</th>
<th>Distance between fixed points on 1st permanent molar</th>
</tr>
</thead>
<tbody>
<tr>
<td>Without adenoids</td>
<td>25.61 mm</td>
</tr>
<tr>
<td>With adenoids</td>
<td>24.90 mm</td>
</tr>
<tr>
<td>Difference</td>
<td>-0.71 mm</td>
</tr>
</tbody>
</table>
The presence of adenoids, therefore, is evidently prejudicial to a proper development of the jaws, although the difference is more pronounced in children who were breast-fed.

These experiments of Colyer, constitute another argument in favour of the breast-feeding of infants.

The influence of heredity on irregularities in the position of the teeth has been referred to (page 11).

3. Causes lying outside the teeth as factors concerned in tooth-decay.

(A) THE ORAL SECRETIONS

It readily occurs to one that the secretions of the mouth will have some influence on the teeth. In fact, we have seen, that Wedl (1870) classifies dental caries under Anomalies of the Secretions. He considered caries due to fermentation of abnormal secretions of oral membranes and salivary glands, in consequence of which, acids develop and these attack the teeth.

Suesserot in Dental Cosmos. 1870. P 129. writes under "Use and Abuse of the Saliva". He considers first the chemical composition of the teeth, and argues therefore that acids are prejudicial to them.

Saliva he says must be kept alkaline to neutralise the acid changes in fermentation. Excessive stimulation of salivary glands through acid decomposition, will lead to fatigue of the salivary glands.
A saliva of feeble alkalinity; or even neutral, may then actually carry acid products and decomposition into crevices out of reach, and thus be an agent in direct antagonism to that which nature intended it to be.

Mr. Sewill (Prevention of Dent. Caries p 27. 1888) ascribes to vitiation of the oral secretions an important influence in the production of caries.

Black (Dent. Cos. 1895, p353 etc.) says "The active cause of caries is a thing apart from the teeth themselves acting upon them from without, and from a consideration of the facts thus far developed, the logical inference is, that the cause of the difference in the liability of individuals to caries of the teeth is something in the constitution operating through the oral fluids, and acting upon the active cause of caries, hindering, or intensifying its effects.

And in (D.Cos. 1899 p.627) he also says, "Observations already made, render it certain that caries has its beginning only when the conditions of the oral secretions are such that micro-organisms causing caries form gelatinous plaques, by which they are glued to the surface of the teeth."

Miller (D.Cos. 1904, p981 etc.) sums up the results of his observations on the relation of the saliva to dental caries, thus:-

1. Quantity of Saliva. Relative dryness is apt to be associated with caries.
2. Viscidity of Saliva. Viscidity favours development of bacteria, but its importance in regard to caries is much exaggerated. One may have much caries with very thin saliva, and little with viscid saliva.

Lohman's view, that caries is due to the action of the mucin of the saliva, stands at variance with some of the simplest facts of dental pathology and bacteriology.

3. Reaction of saliva. Alkalinity favours development of bacteria, but this must be overcome before acids can do harm. During the day the acidity is neutralised by the saliva, but at night, and in deep cavities, it will have only a slight effect. Taking all things into consideration, we are forced to the conclusion that the chances for the production of caries are about as favourable in a mouth with alkaline as in one with acid saliva.

4. Intensity of fermentation in mixture of food stuff with saliva:

It was found that the addition of grape sugar or glycogen did not in the least influence the intensity of the fermentation, or the amount of acid formed. We may conclude that the intensity and rapidity of fermentation of food stuffs retained in the mouth is very great, and practically independent of small amounts of carbo-hydrates in the saliva. (This carbo-hydrate in saliva is referred to later).
5. Antacid action of the Calcium Salts in Saliva. This may be estimated at zero. The Salts in Saliva amount to 0.17%, with say 10% of this as Calcium Salts. Ten times this amount to bread and Saliva did not have the slightest effect upon the course of fermentation, or upon the amount of acid formed.

6. Action of fermentable Carbo-hydrates in solution in Saliva as a cause of caries. There is none in sufficient amount to materially influence the progress of caries.

7. The antiseptic action of the Saliva. Neither the oral fluids as a whole, nor their separate constituents - mucus, potassium, sulphocyanate, etc., have the power to prohibit, or even perceptibly retard, the growth of bacteria.

8. Saliva of Immunes develops, in the presence of carbo-hydrates, in and out of the mouth, on an average, a little less acid than that of highly susceptible persons. The difference is not constant and not sufficiently marked to account for the marked difference in susceptibility.

Kirk (Dent. Cos. 1911, p341) says: "It is in the direction of salivary composition that the true solution of the etiology of dental decay is to be sought out, and, we believe, found. Clean teeth do not decay is a catchy formula, but also misleading even if true in the ideal sense. The phrase is obstructive in
that it tends to concentrate attention upon an erroneous conception as to the causes of tooth decay."

Kirk's view will be referred to later. (p. 62)

Since Miller's work, the most important researches on the oral secretions with reference to dental caries are, so far as I know, those of Pickerill. He points out in his book (page 127) that the salivary secretion is purely reflex in origin, and that its quality varies according to the nature of the stimulation of the sensory nerves of the tongue, and, to a lesser extent, those of the buccal mucous membrane. He further says that the study of salivary secretion has been, until comparatively recently, almost entirely confined to stimulation of the Efferent nerves concerned, and that, therefore, the stimulus is not a natural one, and that it stimulates all the fibres in the nerve at one and the same time, and there is reason to believe, that different sets of these fibres have a specific effect upon salivary secretion.

In order to collect saliva which had been secreted in response to natural stimuli, — that is, by placing substances in the mouth and masticating them, he inserted a small canula into Stenon's duct, and by means of this canula, the parotid saliva was led outside the cavity of the mouth, and collected in a suitable vessel. By means of another arrangement, made after taking a plaster cast of the floor of the mouth, he
was able to collect the mixed saliva from the sublingual and submaxillary salivary glands into another vessel. He points out that the mere estimation of the percentage composition of any given sample of saliva is not enough; it is the ratio of the amount of constituent considered per c.c. to the rate per minute at which this constituent is secreted, that is all important. That is to say, a flow of 1 c.c. per minute of saliva containing 5% of a substance is not so important as a flow of saliva containing 2% of this substance but secreted at a rate of 5 c.c. per minute. The total amount or value of any salivary constituent in the mouth per minute is termed the "index" for that constituent. This may be, for example, the "alkalinity index", the "phosphatic index" the "ptyalin index" etc.

The amount of alkalinity was estimated against $\frac{N}{50}$ H$_2$SO$_4$. The indicator used was Methyl orange, as this is not influenced by the presence of CO$_2$, whereas Litmus is, and Litmus might also give an amphoteric reaction with acid phosphates. Phenolphthalein is also affected by CO$_2$.

The following is one of Pickerill's tables showing results of his estimations of the alkalinity of the saliva following stimulation by the substances named in first column.
<table>
<thead>
<tr>
<th>Substance masticated or used as Stimulant</th>
<th>PAROTID Amount in c.c per min</th>
<th>PAROTID Alkalinity per c.c</th>
<th>SUBLINGUAL and SUBMAXILLARY Amount in c.c per min</th>
<th>SUBLINGUAL and SUBMAXILLARY Alkalinity per c.c</th>
<th>TOTAL from ALL GLANDS Amount in c.c per min</th>
<th>TOTAL from ALL GLANDS Alkalinity per c.c</th>
</tr>
</thead>
<tbody>
<tr>
<td>Normal Resting Saliva i.e. collected 2 hours after a light mid-day meal of same character to show increase or decrease. (Duration of experiment, fifty days).</td>
<td>0.15</td>
<td>1.0</td>
<td>0.65</td>
<td>0.76</td>
<td>1.60</td>
<td>1.75</td>
</tr>
<tr>
<td>Bread &amp; Butter (soft)</td>
<td>0.50</td>
<td>1.0</td>
<td>5.00</td>
<td>1.40</td>
<td>11.00</td>
<td>1.40</td>
</tr>
<tr>
<td>Orange</td>
<td>0.30</td>
<td>1.0</td>
<td>1.35</td>
<td>0.83</td>
<td>3.30</td>
<td>0.83</td>
</tr>
<tr>
<td>Biscuit (soft)</td>
<td>0.50</td>
<td>1.2</td>
<td>3.20</td>
<td>1.00</td>
<td>7.40</td>
<td>1.00</td>
</tr>
<tr>
<td>Apple</td>
<td>0.40</td>
<td>0.5</td>
<td>1.20</td>
<td>1.00</td>
<td>3.20</td>
<td>0.80</td>
</tr>
<tr>
<td>Cake</td>
<td>0.80</td>
<td>0.6</td>
<td>3.30</td>
<td>0.90</td>
<td>8.20</td>
<td>0.80</td>
</tr>
<tr>
<td>Chocolate</td>
<td>0.10</td>
<td>1.0</td>
<td>0.80</td>
<td>1.00</td>
<td>1.80</td>
<td>1.00</td>
</tr>
<tr>
<td>Brown Bread</td>
<td>0.55</td>
<td>0.8</td>
<td>1.80</td>
<td>0.75</td>
<td>4.70</td>
<td>0.75</td>
</tr>
<tr>
<td>Grapes (very ripe)</td>
<td>0.45</td>
<td>2.0</td>
<td>2.00</td>
<td>1.20</td>
<td>4.90</td>
<td>1.20</td>
</tr>
<tr>
<td>Celery</td>
<td>0.10</td>
<td>1.5</td>
<td>2.00</td>
<td>0.80</td>
<td>4.20</td>
<td>0.80</td>
</tr>
<tr>
<td>Carrot (boiled)</td>
<td>0.25</td>
<td>1.0</td>
<td>2.00</td>
<td>1.57</td>
<td>4.50</td>
<td>1.57</td>
</tr>
<tr>
<td>Carrot (raw)</td>
<td>1.00</td>
<td>1.1</td>
<td>5.50</td>
<td>1.02</td>
<td>13.00</td>
<td>1.02</td>
</tr>
<tr>
<td>Figs</td>
<td>0.10</td>
<td>2.0</td>
<td>3.30</td>
<td>1.03</td>
<td>6.80</td>
<td>1.03</td>
</tr>
<tr>
<td>Dates</td>
<td>0.30</td>
<td>1.1</td>
<td>1.80</td>
<td>0.70</td>
<td>4.20</td>
<td>0.70</td>
</tr>
<tr>
<td>Meat (mutton)</td>
<td>0.10</td>
<td>1.0</td>
<td>1.20</td>
<td>0.83</td>
<td>2.60</td>
<td>0.83</td>
</tr>
<tr>
<td>Radish</td>
<td>0.25</td>
<td>1.0</td>
<td>2.00</td>
<td>1.57</td>
<td>4.50</td>
<td>1.57</td>
</tr>
<tr>
<td>Stewed Apple</td>
<td>0.10</td>
<td>1.1</td>
<td>5.50</td>
<td>1.02</td>
<td>13.00</td>
<td>1.02</td>
</tr>
<tr>
<td>Lemon</td>
<td>0.10</td>
<td>1.1</td>
<td>5.50</td>
<td>1.02</td>
<td>13.00</td>
<td>1.02</td>
</tr>
</tbody>
</table>
### Total Alkalinity per minute, or Alkaline Index.

<table>
<thead>
<tr>
<th>Alkalinity per c.c</th>
<th>Amount in c.c per min</th>
<th>Alkalinity per c.c</th>
<th>Alkaline Index</th>
</tr>
</thead>
<tbody>
<tr>
<td>1.05</td>
<td>1.73</td>
<td>1.65</td>
<td>1.05</td>
</tr>
<tr>
<td>0.80</td>
<td>1.28</td>
<td>1.50</td>
<td>1.00</td>
</tr>
<tr>
<td>1.36</td>
<td>15.00</td>
<td>1.90</td>
<td>1.70</td>
</tr>
<tr>
<td>1.03</td>
<td>2.90</td>
<td>1.70</td>
<td>1.00</td>
</tr>
<tr>
<td>1.40</td>
<td>14.38</td>
<td>2.30</td>
<td>1.50</td>
</tr>
<tr>
<td>0.86</td>
<td>2.84</td>
<td>1.70</td>
<td>1.00</td>
</tr>
<tr>
<td>1.02</td>
<td>7.60</td>
<td>2.30</td>
<td>1.20</td>
</tr>
<tr>
<td>0.87</td>
<td>2.60</td>
<td>1.80</td>
<td>1.00</td>
</tr>
<tr>
<td>0.84</td>
<td>6.90</td>
<td>1.80</td>
<td>1.20</td>
</tr>
<tr>
<td>1.00</td>
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THE AMOUNT OF SALIVA

From this table it is seen that the amount of saliva per minute varies very much, both from the parotid, and from the sublingual and submaxillary glands. Also the amount of parotid saliva is much smaller than that from the other glands. It is noticed also that "the most sapid substances produce the greatest flow, and of these sapid substances the ones which are MOST ACID are the most powerful."

THE ALKALINITY OF THE SALIVA

The alkalinity of the saliva shows similar variations, and here again, the MOST ACID substances produce the greatest alkalinity, sweet substances, e.g., cake, bananas, very ripe grapes, being markedly inferior in this respect.

It is evident from the column under "Saliva obtained fifteen minutes after stimulation" that the effects of these food-stuffs is continued for some time after mastication. The effects of bread and butter, bread (white and brown), cake, and biscuit, in producing such a small amount of saliva, and in addition, a saliva of markedly inferior alkalinity, is most important, seeing that these articles form such a large part of modern dietaries, and are susceptible to undergo lactic acid fermentation in the mouth.

By impregnating biscuits with common salt (NaCl), and quinine sulphate, and also acid tartrate of potassium the amount and the alkalinity was found to be increased compared to biscuit alone. Other tables are given to
show that alkaline substances have a depressant action, or at all events, have scarcely any stimulating action, on salivary secretion.

Further, organic acids, such as citric acid, tartaric acid, are much better salivary stimulants than such inorganic acids as sulphuric or hydrochloric acid.

Attention is also called (p 155) to the fact that increased mental excitement and diminished muscular exercise – two concomitant conditions of higher civilisation – both tend in the direction of decreased salivary secretion, and therefore, less effective cleansing of the mouth after meals.

**EFFECTS OF ACID STIMULI ON OTHER CONSTITUENTS OF THE SALIVA.**

Most of Pickerill’s work must be of interest from the physiologists’ point of view, but for present purposes it is perhaps enough to state that the same degree of attention as was devoted to the study of the amount and alkalinity of the saliva, was also devoted to the study of such important constituents as the ptyalin, potassium sulphocyanate and phosphates. He argues that all these constituents are of value and importance in protecting the teeth against dental caries. As in the case of the amount and alkalinity experiments it was found that food with no distinct flavour, or containing alkalies, had a depressing effect on the salivary glands, so in the estimations of the constituents just named – ptyalin, potassium sulphocyanates and phosphates, it was found that the same kinds of food had a depressing, or scarcely stimulating effect, on their secretion, whereas...
articles containing vegetable or fruit acids, had a markedly stimulating effect on their secretion.

MUCIN IN RELATION TO DENTAL CARIES.

Miller (Dental Cosmos. 1905, p 1293) in a paper entitled "New Theories concerning Decay of the Teeth" discusses Lohman's Mucin Theory of caries. Lohman maintained that mucin is converted into mucic acid, and that this acid attacks the enamel. He assumed that some acid taken into the mouth, e.g., acetic, citric, tartaric, etc., liberates this acid. After a series of experiments, Miller writes (p 1299), "In a résumé of all these experiments, it must be affirmed that the hypothesis that the sole cause of decay of the teeth is to be found in the precipitation of mucin from the saliva by means of chance weak acids brought into the mouth with the food is theoretically, logically, and experimentally without foundation."

Pickerill quotes Cook as having shown that mucin can only be converted into mucic acid under aseptic conditions. Mucin by itself in the presence of mouth-organisms undergoes an alkaline decomposition, and is therefore to be regarded as a protective agent against the attacks of acids. There is evidently a close relation between viscid orropy saliva and caries - the viscosity depending on amount of mucin present - but Miller found that there may be no decay with an
intensely viscid saliva, and much decay with thin saliva. Pickerill states that "the association observed clinically between ropy saliva and acute caries in children may be due to one, or a combination, of all of three causes:

1. It may be a special characteristic of chronic pain with a "nervous" or mental effect;
2. It may be due to the original cause of the caries - i.e., a too liberal consumption of free sugar.
3. It may result from a deficient alkalinity of the saliva.

Kirk's Theory of:

THE PRESENCE OF A DISSOLVED CARBOHYDRATE in the Saliva as a cause of Dental Caries

Kirk's statement that "it is in the direction of salivary composition that the true solution of the etiology of dental decay is to be sought out" has already been quoted.

In the British Dental Journal of December 16th, 1913 he discusses his theory of caries susceptibility in some detail. He says he made the suggestion to Miller in 1902 that susceptibility to caries might possibly be conditioned upon the presence in the saliva of a dissolved carbohydrate, the product of metabolism, and not wholly upon the debris of alimentary carbohydrates in the mouth. After some experiments Miller rejected this suggestion. Kirk, however, is satisfied that the saliva, particularly of those susceptible to caries, does contain
a carbohydrate substance capable of reducing Fehling's solution and capable of fermentation. He says the activities of this dissolved carbohydrate, when it exists, correspond more closely to those of glucose than they do to glycogen, and that this metabolic carbohydrate in the saliva is a sugar and not glycogen seems to be strongly indicated he says, by other investigations he has made. He regards the carbohydrate content of the saliva, the contained metabolic carbohydrate derived from the blood - as being the pabulum which is split up into lactic acid by the bacterial ferments in the mouths of caries susceptibles, and it is this metabolic carbohydrate in such cases, other conditions being favourable to the localisation of the process, that is the factor which determines susceptibility to caries of the teeth.

Kirk next refers to the experiment of Claude Bernard in which glycosuria followed puncture of the floor of the fourth ventricle, and also to later experiments by Cushing and others, in which it has been demonstrated that the posterior lobe of the pituitary body has a controlling relationship upon carbohydrate metabolism.

Kirk appears to assume that there is an increased carbohydrate tolerance consequent to some impairment of function of the posterior lobe of the pituitary body, and as a result, an increase of sugar content of the blood, that this sugar appears in the saliva by dialysis from the blood through the salivary gland.
I am not able to advance any criticisms in refutation of Kirk's theory, I can only state that, considering the prevalence of dental caries, it is difficult to believe that hypophyseal irritation can be so common a disorder as to be an important factor in the etiology of Dental Caries.

Pickerill examined the saliva for glucose in a number of healthy and sick persons, including cases of diabetes severe and mild, nephritis, acute and chronic Grave's Disease, typhoid fever, phthisis, gastro-enteritis, anemia, neurasthenia, aneurism, locomotor-ataxy, and syphilis, but on no occasion did he find a sign of glucose.

**Self-cleansing power of the mouth susceptibility to Caries.**

It is of interest to note that Breese (British Dent. Journal, June 16th 1913) in his examination of over 1000 school-children, found great differences in the self-cleansing power of the mouths. Where the self-cleansing was best the teeth were most immune. The children were all living under the same conditions; therefore, he concludes some mouths have teeth much less susceptible to lodgment of food on them than others have. This factor seems more important than the possible presence of a dissolved carbo-hydrate in the saliva in regard to the liability to dental caries.

**Internal Secretions and Dental Caries.**

Where the causes are so obscure it is almost natural to expect that the internal secretions would be assumed to exert an important influence on the teeth. We have seen how both the normal activity on the part of the pituitary gland. Others have considered thyroid activity at fault. In a paper in the British Medical Journal (June 27th, 1914) Pickerill produces results of experimental investigations regarding internal secretions and dental caries. He concludes very cautiously thus: "There is some reason to think that deficiencies in the secretion of the thyroid, and perhaps the pituitary and thymus glands, are concerned in the lowering of the resistance of the tissues to dental caries. It is necessary, however, to be careful in generalizing. Because thyroid insufficiency may be a causative factor in certain patients, it does not follow (to say that it is always a cause of caries. There are many other factors, physiological and pathological which must always be taken into consideration."
J. S. Wallace has written several books on this aspect of the subject. He seems to have little doubt that he has discovered the cause of dental caries, and that cause is to be found in the nature of the food stuffs consumed by modern peoples. In his Prevention of Dental Caries he says: "The cause of the prevalence of Dental Caries is that the natural food stuffs are to a large extent deprived of their accompanying fibrous parts, and prepared and consumed in a manner which renders them liable to lodge and undergo acid fermentation in the mouth, while from the same cause and the induced conditions, the micro-organisms of the mouth lodge and multiply, and augment the rapidity and intensity of the acid fermentation."

Wallace points out also that the coarser parts of the food excite a more copious flow of saliva in addition to requiring a greater amount of mastication. They also have a detergent action on the teeth, so that adherent masses or plaques of bacteria, are frequently removed, or rather, are not allowed to form on the teeth. If teeth could be kept clean in this respect then caries would be prevented. Wallace's views may be summed up by saying that the more fibrous parts of food as supplied by nature are intended to play the part of a physiological tooth-brush. He does not overlook, however, the importance of acid substances in inducing a copious flow of saliva, for in his Supplementary Essay, 1906, p 33, after deprecating the highly refined character of the flour in bread-making he says: "So also the modern preparation of food tends to deprive it of the natural fresh vegetable acids which would
otherwise almost invariably accompany vegetable food. Thus it correspondingly happens that the saliva is not so fully stimulated as it should be.

BREAD IN RELATION TO DENTAL CARIES

This is the title of a paper by T. G. Read, reported in Proceedings Royal Med. Soc. (Odont. section) Vol. VI, PtIII, p 11. He points out that the natural ferments of the wheat originate in the columnar cells which separate the kernel from the germ. Under suitable conditions as regards temperature and moisture, these ferments begin to act, one ferment acting on the cellulose and one on the starch. The ferments of wheat, if not destroyed previously, act during malting, bread-making, and its equivalents, mastication and digestion. As a result, sugar is formed. When the unimpaired ferments are present in the flour, the sugar will not be found in the flour, but it will be found in the bread.

The millers, Read goes on to say, destroy the vitality of the germ by heat. (The germ may also be removed by "bolting"). Flour containing germs cannot be kept a long time without becoming acid, but it makes bread that does not become stale as quickly as most bread does now.

All freshly made flours are free from acid, but all breads are slightly acid and become more so when stale. Stale natural acid of bread does no harm to the teeth. It is the forming of nascent lactic acid that does harm, but once formed, it soon loses
the stronger decomposing action of its nascent state. When bread, made from flour containing the unimpaired ferments is eaten, it has to remain in the mouth for hours for any acid to be formed. Therefore it does no harm while passing through the mouth. When bread, however, made from flour in which the natural ferments are destroyed is eaten, lactic acid is rapidly formed, by micro-organisms in the mouth, and this disintegrates the tooth tissue without any bread resting on the tooth. Harm so done cannot be reduced by eating detergent or cleansing food or by washing or brushing the teeth after meals. Lodgment of food around the teeth is more a sign than a cause of caries. Conversion from starch to lactic acid is only a matter of a few seconds in the mouth.

Read refers to experiments showing effects of chewing the two types of bread on the teeth. Teeth are placed with their crowns embedded in freshly chewed bread, some in bread from flour containing ferments, and some in bread from flour in which the ferments were destroyed, and kept in warm room. The teeth are removed from the chewed bread, cleaned, and replaced in freshly chewed breads, about as frequently as the mouth is filled and emptied during eating. The teeth embedded in the bread made from flour with impaired ferments are corroded, but the teeth embedded in the bread made from flour containing the natural ferments remain unaltered.
Stone milled flour contains the natural ferments, whereas in Roller milled flour the ferments are either destroyed or got rid of.

The same writer quotes the findings of chemists showing an increase in the acidity of 40% during mastication of bread made from Roller milled flour. Apparently there was scarcely any increase in acidity of the bread masticated when it had been made from stone milled flour.

The foregoing is a summary of Read's paper. It ought to be added that the views advanced did not meet with a ready acceptance on the part of the audience of dentists.

**Sweetmeats**

With regard to the question of sweetmeats and dental caries it may be added that there is reason to believe that indulgence in such sweetmeats favours the production of tooth-decay. The question was discussed in the British Medical Journal, Vol. II., 1911. Some well-known dental practitioners took part in the discussion, but it cannot be said that the evidence produced in favour of view that sweetmeats is an important cause of caries was at all convincing.

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CONCLUSIONS.

In comparison with ancient races, so far as this comparison is possible by means of the study of the skulls of those races, modern civilised races show a great increase in the incidence of dental caries.

Civilisation, both among ancient and modern races, is associated with an increased incidence of dental caries.

No one-idea theory is sufficient to account for the prevalence of dental caries.

The Chemico-parasitic theory of Miller is the one most generally accepted though it has been described as being based chiefly on test tube experiments.

Marked immunity or susceptibility to dental caries is not explained by the Chemico-parasitic theory.

Kirk's theory, that a dissolved carbohydrate is in the saliva and that this accounts for the marked susceptibility of some persons to dental caries is not sufficiently corroborated.

Hopewell-Smith's theory (p 54) of a specific micro-organism acting under torn edges of the enamel cuticle may be an interesting speculation, but it does not rest on experimental evidence.

Defects in the structure of the teeth favour the caries producing cause or causes.

Breast-feeding of infants has a favourable influence on the teeth in rendering them less liable to caries, though this influence is only slight.
A good flow of saliva possessing as high a degree of alkalinity as possible is of great importance as a natural self-cleansing agent in the mouth.

Pickerill's researches leave no room for doubt, that food stuffs should possess a distinct flavour, and that the acid flavour of vegetable or fruit acids is the best, as this produces a saliva not only greatest in quantity, but possessing the largest amount of its natural protective constituents.

Bread should preferably possess a fair degree of coarseness, as this also stimulates the self-cleansing factors in the mouth.

There is some evidence that bread made from stone milled flour produces less lactic acid in the mouth than does bread made from roller mill flour. (The lactic acid in its nascent state in the mouth attacks the teeth.)
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