A THESIS
ON
THE MOUTHS OF MENTAL DEFICIENTS.

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1932.
THE MATERIAL contained in this Thesis is the outcome of a close study of Mental Deficients by the Author, extending over a period of eight years, in his official capacity as Dental Surgeon to both Newcastle and Stockton Mental Hospitals.

Neither time nor expense has been spared in photographing patients, and the models obtained from them; as a photographic representation is always useful in supplementing a written description of a case.

No list of illustrations has been compiled; the photographs have been inserted in juxtaposition to the subject matter in the text which they illustrate.

Each model has been reproduced photographically at its actual size; but as some of these photographs do not show the correct contours, the actual models have been submitted for inspection.

The photograph of the model of a normal dental arch and palate is shown, for comparative purposes, on page 1.

As individual mental deficients are very often difficult to manage, many models have been lost through inability to obtain perfect impressions, most of these having been lost through struggling on the part of the patient.
A special chapter has been devoted to the clinical types of mental deficiencies; and the illustrations should be of considerable value in emphasising these types.

The description of the patients from a psychiatric point of view has been necessarily brief. No more than a short outline has been given, as a full description is outside the scope of this thesis.

My grateful thanks for the ever ready assistance so willingly accorded me at all times is due to Dr. C. A. Hogg, Inspector General of Insane for New South Wales, for permission to use the data collated by me during my period of service as the framework of this thesis; to Drs. G. Ewan and S. Minogue, Medical Superintendents of the Newcastle and Stockton Mental Hospitals respectively, who by their practical assistance and advice, were of inestimable service in classifying the clinical types; and to the Day and Night Nursing Staffs of both Newcastle and Stockton Mental Hospitals, who provided the facilities necessary for confirming the abnormal habits and sleeping postures of the patients.

I have no hesitation in stating that without the thoughtful aid of those mentioned above, it would have been impossible for me to have compiled the clinical data in this Thesis, or to have arrived at the conclusions contained herein.

B.W.C.

Newcastle, N.S.W.
August, 1932.
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Fig. 1. (Model No. 1).
B.... C...., aged 13 years.
Perfect arch.
Photograph actual size.
CHAPTER I.

INTRODUCTORY

The object of this monograph on THE MOUTHS OF MENTAL DEFICIENTS is to show that, although abnormalities occur in the palates of mental deficient, the proportion of abnormal palates in mental deficient is not greater to any extent than that of abnormalities in the maxillae of persons of normal intelligence.

The fact must be taken into account that mental deficient, as a class, carry on to adulthood many abnormal habits which, in normal children, are generally corrected at a very early age by parents, guardians and teachers. These habits, which may deform the maxilla, are continued in mental deficient owing to their lack of co-operation, without which constant correction is of no avail.

Many of the worst deformities are seen in Mental Hospitals owing to lack of correction. These deformities would also be greatly in evidence amongst normal children if it were not for the co-operation of the child.

Environment also plays an important part in connection with the abnormalities of the maxillae of patients in Mental Hospitals. Thus, the example set by certain children in a ward is often copied by other children in that ward; so that the habit of thumb sucking, for example, may be extensively practised. On the other hand, in normal home life, a child may be punished for, or
made ashamed of, sucking its thumb or fingers. The same does not apply to the children in Mental Hospitals, whose mentality often does not admit of correction.

The question of food and diet also arises in connection with the malformation of the dental arches of mental deficient; likewise as regards those of normal persons.

In institutional life, individual treatment cannot be given; the diet is arranged to suit the greatest number of patients. Should a large percentage be able to manage only minced food, or food which requires little mastication, that food must accordingly be the same for the whole number.

Constitutional causes, which affect mainly the primary type of mental deficient, also have a bearing on the abnormal development of the maxilla. The average mental deficient is ushered into the world with a very low degree of physical resistance. The physique is not of such a high standard as that of a normal individual; consequently the numerous causes of abnormal development of the maxilla are enabled to progress unimpeded.

The theory of Sir T. S. Clouston and Dr. Langdon Down, that an abnormal palate is a characteristic of mental deficiency, will be discussed fully in this monograph. The author will show that there is no connection whatsoever between the mental condition and the palatal abnormalities which exist in a certain proportion of mental
deficients; and that the shape of the dental arches and maxillae of mental deficiencies is not pathognomonic of any group, whether it be in primary or secondary amentia.

The system laid down by A. F. Tredgold in his "Mental Deficiency", 1922, published by Balliere, Tindall & Cox, has been followed by the author of this thesis in classifying patients into the primary and secondary amentia groups.

Each group has been dealt with separately where it has been possible to classify patients; but there are several border line cases which do not fall into any definite classification; these have been grouped as mental deficient only.

Patients to the number of 585 were examined by the author for maxillary abnormalities; the results have been tabulated, and the tables appear throughout the text of the thesis.
5.
CHAPTER II.

CLASSIFICATION OF MENTAL DEFICIENTS
AFTER TREDGOLD (WITH TABULAR SUMMARY)

As has been previously mentioned, the classification laid down by Tredgold has been used throughout this work, in order that a clear conception of the terms primary and secondary amentia may be conveyed.

It has been found that all mental deficiencies may be grouped into either one of two classifications namely, primary or secondary amentia. The terms "primary" and "secondary" are applied in accordance with the fundamental cause of the weakened intellect.

PRIMARY AMENTIA.

Approximately 85 per cent. of aments are the products of defective germ plasm; and for this reason the term "primary amentia" is used in describing this group. The defective germ plasm may originate through many causes; of these the commonest are ancestral alcoholism, syphilis and tuberculosis. The result of the presence of the defective germ plasm is that amentia, insanity or epilepsy may occur.

SECONDARY AMENTIA.

In this group, there are cases, approximating 15 per cent. in which there is no morbid inheritance; the growth of the brain, however, is interfered with by disease or by adverse environment, so that the patient does not develop normally.
Mental deficiencies may be further classified according to three groupings:

I: Feeble-minded.
II: Imbeciles.
III: Idiots.

I. Feeble-minded: The members of this group are able to do simple routine work, if supervised; latitude is allowed for mistakes, and a high grade of efficiency is not required. They can not use money obtained as a result of their labours to the best advantage; and they are unable to formulate any plans for improving their position in life. They can not compete on equal terms with their normal fellows.

II. Imbeciles: The members of this group require constant supervision if doing simple routine work such as scrubbing or washing. They are able to discern common physical dangers such as fire or flood; and they will not deliberately walk in front of vehicles, knowing the danger to themselves. Usually they can dress and feed themselves, but do not know the value of money; nor do they know how to spend it to any advantage.

III. Idiots: The individuals in this group are unable to avoid physical dangers, and therefore must be constantly watched. They can not dress themselves, and a number of them are unable to speak.

A practitioner is often able to classify patients into either the primary or secondary
amentia group, according to special facial or anatomical characteristics. The clinical signs of the different types are as follows:

**PRIMARY AMENITIA.**

I. Mongolism
II. Mongoloidism
(Pseudo-Mongolism)
III. Microcephaly.
IV. Simple Amentia.

I: MONGOLISM

This type of amentia is most pronounced, and little experience is required in order to distinguish it. The members of this group are so similar in appearance that it would appear as if they were brothers and sisters. They are below normal height, stunted in growth and are mostly of the imbecile group. The palpebral fissures are narrow, and slope inward in a downward direction. After the age of 5 years the tongue is enlarged, fissured and peculiarly characteristic. The hand is broad and short; the little finger is very often incurved. There is usually a broader space than normal between the big toe and the next one. Mongols are usually placid, good-natured people with a great sense of mimicry.

Of the total number of patients examined, namely 585, the Mongols represented $\frac{4.2}{2}$ per cent. or 25 patients. Tredgold states that they represent 5 per cent of adult aments.

II. MONGOLOIDISM-(Pseudo-Mongolism):

In all types of amentia there are certain border-line cases which, although they present many characteristics in common with a type, do not
possess all the typical characteristics.

Mongoloidism very closely resembles Mongolism. The members of this group usually have the oblique palpebral fissures associated with Mongolism. Their heads may be brachycephalic; their tongues, however, are rarely fissured to the same extent as are those of Mongols.

III. MICROCEPHALY.

The members belonging to this sub-group of primary amentia are characterised by having heads whose circumferences are less than normal; that is to say, less than seventeen inches. The test of measurement, however, may be waived in favour of the appearance of the head, which is usually characteristic. There is a marked recession of the anterior portion of the skull, and a marked flattening in the occipital region. This gives the appearance which is so typical; thus it is possible for a person, whose head measurement is approximately nineteen inches, to be classified as a microcephalic. The hair is usually very thick and coarse, and the stature is usually below normal. The author ascertained that the individuals in this group numbered 20 and represented 3.4 per cent. of the 585 patients.

IV. SIMPLE AMENTIA.

The members of this group have no special facial nor anatomical peculiarities; and therefore are classified as simple aments.

The four types of primary amentia described above may be further complicated by having any of the following pathological or developmental
conditions namely:

(a) Epilepsy.
(b) Paralysis.
(c) Hydrocephaly.
(d) Porencephaly
(e) Sclerosis.
(f) Deaf-Mutism.

(a). EPILEPSY: A considerable percentage of primary aments suffer from epilepsy. This disease is least frequent in the higher grades of amentia, and most common in the lowest grade.

(b). PARALYSIS is also most frequent in idiocy, and least frequent in feeble-mindedness.

(c). HYDROCEPHALY: This condition occurs amongst primary aments, but is usually associated with secondary amentia. It is characterised by an enlargement of the skull, brought about by the accumulation of cerebro-spinal fluid within the brain.

(d). PORENCEPHALY and (e) SCLEROSIS do not, in my opinion, present clinical features which are sufficiently clear to be described for the purpose of this work.

(f). DEAF-MUTISM: This type of primary amentia is quite characteristic. It represents a very minute percentage of that group.

SECONDARY AMENTIA.

CLINICAL TYPES: There is often considerable difficulty in determining some of the types of secondary amentia. Often the classification may only be suspected during life; so that it is
not possible without a postmortem examination to be assured of the correct division.

Porencephalic Amentia, Sclerotic Amentia, and Amaurotic Family Idiocy are grouped with these types, which are difficult to distinguish clinically.

The main types are as follows:

I. Hydrocephalic Amentia.
II. Syphilitic Amentia.
III. Epileptic Amentia.
IV. Cretinism.
V. Nutritional Amentia.
VI. Isolation Amentia.
VII. Oxycephalic Amentia.

I: HYDROCEPHALIC AMENTIA.

This condition depends on the accumulation of cerebro-spinal fluid within the brain ventricles. The pressure of the fluid distends the bones of the skull, so that they become widely separated. This gives the characteristic head picture, the skull being evenly distended in all directions.

The skull measurement may be slightly above the average, or it may attain to thirty inches in circumference.

II: SYPHILITIC AMENTIA.

Only a small number of patients may be classified as belonging to this group. Of the 585 persons examined, 53 representing 9 per cent were found belonging to this class.
Certain typical lesions are indicative of the condition. These may or may not be definite, so that a Wassermann test is the most authoritative method of classifying these patients. The typical lesions are:

1. Hutchinson's teeth;
2. Moon's domed molars;
3. Rhagades at the angles of the mouth;
4. Enlarged parietal bosses;
5. Depressed nose.

The majority of the patients in this group are undersized and ill-nourished; their progress in talking and walking is greatly retarded.

III. EPILEPTIC AMENIA.

The patients in this classification are, on the whole, better developed than the members of the simple amentia groups. It is often difficult to distinguish them from the patients belonging to the simple primary amentia groups. A study of the personal history of each is the only satisfactory method.

IV. CRETINISM.

Very early in life, certain distinguishing symptoms may be noticed.

The child is apathetic, and its growth is very defective when compared with normal children. The tongue is often enlarged; the skin may be yellowish in colour, and there is usually a puffiness of the face, hands and feet.
The typical appearance of a cretin when fully grown is very characteristic. The body is considerably dwarfed, the legs being short and ill-formed.

V. NUTRITIONAL AMENITY.

Tredgold quotes one case of nutritional amenity—a child born at the seventh month. The mother was delicate, and when the child was born it weighed only two and a half pounds. At the age of three and a half years, the child was no bigger than than an infant twelve months old. She was obviously very backward mentally, and died of enteritis shortly after the age of three and a half years.

VI. ISOLATION AMENITY.

The most common cause of isolation amenity is inflammation of the sensory areas as a result of infectious fevers. The centres usually affected are those of either sight or hearing, or both. Should this condition eventuate very early in life, the subjects will be shut off from educational facilities, and will never develop mentally. They are usually well grown, and exhibit no stigmata of degeneracy.

VII. OXYCEPHALIC AMENITY.

The clinical signs of this classification are manifested principally in the skull and eyes. The skull is very high, and is brought to a sharp point at the vertex. The eyes evidence exophthalmos in
a marked degree.

REFERENCES.

2 IDEM ibid., p.240.
3 IDEM ibid., p.236.
<table>
<thead>
<tr>
<th>Degree of Amentia</th>
<th>Characteristics</th>
</tr>
</thead>
</table>
| Feeblemindedness  | Members of this class
| (The mildest grade of mental defect.) | Are able to make tolerable progress at school, read, write, and do simple sums.
| | Are able to engage in routine work of a simple character with little supervision.
| | Are able to earn their own living if provided with an occupation suited to their capacity.
| | Are unable to make plans for the future.
| | Are unable to co-ordinate their conduct in such a manner as to enable them to maintain an existence independently of some outside supervision. |
| Imbecility        | Members of this class
| (A medium grade of mental defect.) | Are able to spell simple words of one syllable and count upon their fingers.
| | Are able to recognise winter from summer.
| | Are able to guard themselves against common physical dangers that threaten existence.
| | Are unable to perform work which will pay for their keep i.e., members of this class are incapable of earning their own living. |
| Idiocy            | Members of this class
| (The lowest grade of mental defect.) | Are unable to understand and avoid the common physical dangers that threaten existence.
| | Are unable to wash, and dress; and a percentage are unable to feed themselves. |
## TABLE II.

**THEDGOLD'S METHOD OF CLASSIFICATION — (TABULAR FORM).**

### PRIMARY AMNENTIA.

*(85% OF ALL AMNENTIA.)*

<table>
<thead>
<tr>
<th>ETIOLOGY</th>
<th>CLINICAL VARIETIES</th>
<th>PATHOLOGY</th>
</tr>
</thead>
<tbody>
<tr>
<td>Ancestral alcoholism, Tuberculosis, Syphilis; Morbid Heredity, as evidenced by Amaurotic, Insanity, Epilepsy etc. in ancestral history.</td>
<td>(1). Simple. (2). Microcephalic. (3). Mongolian.</td>
<td>A numerical deficiency, irregular arrangement and imperfect development of cortical neurones.</td>
</tr>
</tbody>
</table>

### SECONDARY AMNENTIA.

*(15% OF ALL AMNENTIA.)*

<table>
<thead>
<tr>
<th>ETIOLOGY</th>
<th>CLINICAL VARIETIES</th>
<th>PATHOLOGY</th>
</tr>
</thead>
<tbody>
<tr>
<td>II. MECHANICAL viz: Hemorrhage, Embolie, Trauma, Thrombosis.</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

### MODIFICATIONS DUE TO

<table>
<thead>
<tr>
<th>QUALITATIVE</th>
<th>QUANTITATIVE</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. DEFECTION</td>
<td>2. DEFECTIVE</td>
</tr>
</tbody>
</table>

### OTHER CONDITIONS

<table>
<thead>
<tr>
<th>ETIOLOGY</th>
<th>CLINICAL VARIETIES</th>
<th>PATHOLOGY</th>
</tr>
</thead>
<tbody>
<tr>
<td>Isolation or disease of special sense organs leading to defective stimuli from without</td>
<td>Isolation.</td>
<td>Localised arrest of neuronic development.</td>
</tr>
</tbody>
</table>
CHAPTER III

RESUME OF LITERATURE
WITH AUTHOR'S REMARKS.

The general consensus of opinion amongst the medical practitioners attached to Mental Hospitals, who deal with mental deficiencies as a class, is that a deformed maxilla or a high arched palate generally indicates a weakened intellect. This theory was in existence before the year 1876; as T. Claye-Shaw, Medical Superintendent of Leavesden Asylum, in that year read a paper on the subject contradicting this fallacy; the theory, however, persists to this day.

The aim of this monograph is to show that palatal deformities do exist; but that they are no indication whatsoever of a weakened state of intellect. To this end, past literature will be reviewed, dealing with the palates of mental deficiencies.

Langdon Down—quoted by Clouston—attached so much importance to the V-shaped palate in idiocy and congenital imbecility that he described it as characteristic of these conditions. Clouston states that "the condition of the palate may be a most important index of brain development, and of a liability to the developmental neuroses" whilst Ireland states: "The most common accompaniment of genetous idiocy is what has been variously called the keel-shaped or saddle-shaped, or vaulted, or lambdoid palate."

Peterson states: "The deformed palate
"is, to my mind, one of the chief anatomical "stigmata of degeneration."

There are many causes of malformed palates, which will be grouped under three separate headings in this paper namely:

1. Habit
2. Constitutional causes.
3. Faulty development of the Child.

1. HABIT: It will be shown that 58.29 per cent. of mental deficient have habits that may alter the shape of the palate from the normal, through various degrees of abnormality, to a gross deformity.

2. CONSTITUTIONAL CAUSES are those that affect the general metabolism of the patient.

3. FAULTY DEVELOPMENT OF THE CHILD may be congenital or acquired. Under this heading such important questions as food, diet and unnatural feeding of infants will be treated.

In studying the works of Langdon Down, Ireland, Clouston, Peterson, Claye–Shaw and Channing, no mention has been made of any of the above-mentioned subjects, with the exception of faulty development of the child from a congenital point of view. The gross malformation – cleft palate – is undoubtedly intra-uterine in origin; consequently it has been treated very briefly in this paper, which deals with post-natal deformities.
In the year 1876 Claye-Shaw evolved a theory disproving that a high-arched palate, so commonly found amongst mental deficientes, was indicative of a low mental calibre. He measured the palates of the inmates of Leasewden Asylum with an instrument capable of great accuracy; and his results are as follows:

"1. There is no necessary connection between a high palate and the degree of mental capacity of the individual. Some idiots have the flattest and most symmetrical palates, whilst many with strong individuality of character have highly-arched palates.

"2. There is a general relation between the shape of the palate and that of the skull as to length and breadth.

"3. A narrow pterygoid width is invariably associated with a high palate, as is also a narrow skull.

"4. The width of the first molars is almost invariably less than or equal to the inter-ptyerygoid width and is only very rarely greater.

"5. The arching of the palate has nothing to do, as regards height with premature synostosis of the skull-base.

"6. The difference in the palatal measurements of various mouths are so slight and so various that it is difficult to see of what service a palatal investigation can be in affording a clue to the mental facilities."

I have not taken accurate measurements of the pterygoid width of mental deficientes, but I
am convinced that Dr. Shaw is correct in this regard.

The pterygoid muscles, in their function of aiding, with other muscles, the protrusion and closure of the mandible must develop in direct proportion to the function they perform.

A high palate shows lack of development in the palatal region; and this development is partly dependent on the stresses and strains transmitted by the mandible, into which the pterygoid muscles are inserted. It naturally follows, therefore, that the width must be approximately the same as the palate. A wide palate will have accompanying it a wide pterygoid width and vice versa.

It is possible for microcephaly to be caused by a premature synostosis of the cranial bones; although this is not now generally accepted as the complete causation; so that, if Dr. Shaw is correct, we should find microcephalics with normal palates. The writer discovered this to be the case. Of the 20 microcephalics examined, 4 cases only were found to have abnormal palates. Of these 4 cases, 3 were very definitely thumb or finger suckers, and one was a microcephalic Mongol, whose palate was flattened and small, through circumstances enlarged upon in the section dealing with Mongolism (Chapter VI.)

Shaw states that 2 microcephalic sisters
amongst those patients he examined had the least vertical heights of any palates he had measured.

Dealing with Ireland's statement that the most common accompaniment of genetous idiocy is the keel-shaped palate, the writer found this characteristic present in only 27.7 per cent. of the 349 primary aments (genetous) examined. Grouping primary and secondary aments, it was found that the proportion was 24.8 per cent. of the 585 patients examined; thus it was not possible to agree with Ireland's statement.

As previously stated (pp. 15 & 16) in the opinion of Peterson the deformed palate is one of the chief anatomical stigmata of degeneration. No definite figures are given, but this authority quotes the following investigators:

"Talbot reported 43 per cent. of abnormal palates in 1605 inmates of institutions for the feeble-minded. Ireland makes it nearer 50 per cent. Charon, a later writer than these, found abnormal palates in 10 per cent. of apparently normal persons, in 82 per cent. of idiots and feeble-minded, in 76 per cent. of epileptics, in 80 per cent. of cases of insanity in general, in 70 per cent. of the hysterical insane, and in 35 per cent. of cases of general paralysis." 8

Clouston deals very extensively with the palate in mental deficient, and draws attention to an interesting "morphological accompaniment of many of the developmental neuroses viz: a change "in the normal shape of the hard palate." 9
Clouston examined many hundreds of palates of the same, insane and idiots, and finally decided to classify the palates into three groups namely, "Normal", "Neurotic" and "Deformed". The "Normal" palate was described by him as a horseshoe arch with a regular and wide dome. This description is the standard to-day as it was then; so that any deviation from this standard must be classified as abnormal. The "Neurotic" arch, in Clouston's description, is more of the "Gothic" type, with "the alveoli tending to run more parallel for a greater distance than the 'Typical'" (or normal.)

Clouston, in describing the deformed palate, states that it may be of any abnormal shape; that is to say, V-shaped, gothic, saddle-shaped, or having marked central bulges along the line of ossification; and various other abnormal formations. He also states: "Like all things in nature the three classes ran imperceptibly into each other with no abrupt line of demarcation."

The divisions classified as "Neurotic" and "Deformed", from the writer's point of view, are so fine, that he has found it impracticable to classify his 586 cases into these groups, and so has been content to classify them as "Normal" and "Abnormal".

Clouston classified 169 cases of mental deficient into the three descriptive groups and found that "of these, 11 per cent. were found to have "'Typical'" (normal)" palates, 28 per cent. were "'Neurotic' and no less than 61 per cent.
"Deformed" - "the average degree of abnormality in the deformed being far greater than the abnormality among the general population, that is, we found the worst palates of all amongst them."

Grouping the "Neurotic" and "Deformed" classifications under one heading, that is to say, "abnormal", Clouston found only 11 per cent. had normal palates, and 89 per cent. abnormal.

The writer's findings are quite different. He found only 24.8 per cent. of the 585 mentally deficient patients examined to have abnormal palates, and 75.2 per cent. to have normal palates.

The finding by Clouston that the worst palates were found amongst mental deficiencies is quite feasible. Mental deficiencies can not be disciplined with the same results as can normal people. Any habits which they may indulge in, tending to make palates abnormal, would be extremely difficult to check for two main reasons - firstly, the lack of co-operation on the part of the patient and secondly, the lack of individual attention and loving care in the institutions where they are confined.

The examination in prison by Clouston of 6 babies under the age of one year, and his findings that four of these infants had abnormal palates, may also be possibly attributed to artificial feeding, to the habit of thumb sucking or to many other malformeding causes. It is thus possible that mothers, who could think so little
of their infants that they should be incarcerated, would know but little of the dangers of unnatural feeding and its resultant bad habits; and thus would not correct faults that would tend to malform the palate.

The classification of palates of infants under the age of one year is a very difficult matter, owing to the alveolar bone not being completely formed. The examination of many young babies would be necessary, in order that some idea of a standard normal palate might be reached.

The author is unable to see any reason for Clouston's assumption, that the shape and size of palates have a direct relationship to the shape and size of the base of the brain.

In his summing-up, Clouston has given fifteen paragraphs to the palate and maxillary bones. He points out that there is a close relationship between the base of the brain and the palate; and that the skull in its growth and development is absolutely dominated by the brain. "Then the brain growth will in this way secondarily determine the shape of the upper maxillary bone and the palate,"

H. A. Harris, Assistant Professor of Anatomy, University College, London in an article, quotes Scammon as having shown that, from birth to the eighteenth month of age, the brain and skull grow with extreme rapidity; and that at 2 years of age, they have attained to 60 per cent, of their adult size. The author of this monograph
maintains that the size and shape of the maxillae depend more on the growth of the facial bones, which are not properly developed until adulthood; since at 2 years of age the area and shape of the palatal bones are certainly not equal to 60 per cent. of their adult size.

At the age of 7 years, the skull has attained to almost its adult size; yet considerable growth has subsequently to be made in the maxillae in order to accommodate the 12 year molars and the wisdom teeth.

Cunningham's "Anatomy" states that "the skull grows rapidly from birth until the seventh year; at that age, the orbits are almost as large as in the adult. The laminae cribrosae of the ethmoid, the body of the sphenoid, the petrous parts of the temporal bones and the foramen magnum have reached their full size. The jaws have enlarged in preparation for as well as coincidentally with the eruption of the teeth, the chief enlargement in them being in the alveolar process."

It necessarily follows that, as these bones, which help to constitute the structure of the cranium, are at 7 years of age almost fully developed, the facial bones, which continue to grow until adulthood, must influence the growth of the maxillae; as at 7 years of age the maxillae are growing in order to accommodate more permanent teeth.

If Clouston's reasoning is correct — that the
base of the skull, conforming to brain growth, determines the shape and size of the palate — there would be patients of the hydrocephalic and microcephalic types with abnormal palates. The writer ascertained that the contrary was the case. Of the group of microcephalic patients whom he examined, 20 per cent. had abnormal palates; and the abnormalities were the result in each case of malformed habits. The palates of all the cases of the hydrocephalic type were normal.

Shaeffer, Todd, Helman, Brash and many other research workers have shown that the maxilla and mandible grow with age; and that the increase in size is due to growth which normally harmonises with the growth of the facial bones.

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Shaeffer, dealing with some of the problems of genesis and development states: "The skull in man, as in all mammals, consists of two parts, the facial part carrying the teeth and developed according to the size, and the brain capsule, which develops in accord with the size of the brain. The larger the brain, the smaller the face, and the less does the face project in front of the skull; and on the contrary a small brain means a larger face, and a greater facial projection in front of the skull."

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According to Helman the height of the face increases as the individual progresses from stage to stage, until, in the course of time, old age is reached; after which there is a decrease in height taking place during senility. This process of development is accompanied by accelerations and retardations in the rate of growth.
There is an acceleration of growth during the period of late infancy, which is followed by a period of retardation during childhood. A second and most pronounced acceleration then sets in again during the period preceding puberty; to be followed by a gradual retardation in the rate of growth until senility.

1. **Growth in Height:** This growth is attained by:

   (a). Moderate increase in the dimensions of the upper and lower face.

   (b). Greater increase in the subnasal area and lower molar region; (really the alveolar process).

   (c). Development of the dentition pushing, as it were, the upper and lower face apart.

2. **Growth in width:** This growth is attained by:

   (a). Moderate increase in width in the median plane anteriorly.

   (b). Greatest increase in width in the lateral halves, especially at the gonion.

   (c). Intermediate amounts of growth posteriorly ascending from the gonion and laterally between gonion and canine.

3. **Growth in depth:** This growth is attained by:
(a). Increase in dimensions of facial structures in anterior-posterior direction. (b) Increment taking place at the posterior end of the structures concerned, palate and alveolar process of the maxillae above, and alveolar process, ramus and body of the mandible below.

Brash asserts that "the growth of the alveolar bone is of the greatest, possibly of sole importance for the general increase in height of both the maxilla and mandible" 13 and again, "the growth of the alveolar border leads therefore directly to an increase in the depth of the bodies of the mandible and maxilla." 19

There is no evidence to prove that an abnormal palate may be a reversion to a lower animal form, or to a lower type of mankind. All animals living in a natural state on natural foods, have jaws that are developed in conformity with their mode of living and the species of food that they consume. The native races of Australia, which are regarded as having a low standard of mentality have, in their natural state, well-developed palates. When they become accustomed to the civilising effects of contact with the white race, however, living on food such as is eaten by civilised peoples, they develop caries rapidly; and their offspring exhibit a type of jaw which was not in evidence in the development of the forbears.
H. E. Noble, writing concerning the teeth of the Richmond River Blacks, states: "A dentist whose practice is exclusively among whites, when he looks first in an aborigine’s mouth, will notice at once that the whole mouth and the teeth and jaws are larger in all dimensions relatively to the size of the cranium and of the whole individual."

This statement from an authority on the jaws of the Australian aborigine, together with the fact of their phylogenetic position, which shows that they are a very primitive race, also supports Clouston’s statement, that an abnormal palate is not a reversion to lower animal form, nor to a lower type of mankind.

Thumb sucking can definitely cause an abnormal palate. This statement is referred to later in the present monograph under the heading "A Description of Habits tending to Malform the Dental Arches." (Chap IX., pp.139 et seq.) Newborn idiot babies do not suck well during the first few days of their lives. To the lay mind, this is generally the first indication that the infant is subnormal. The habit of sucking, however, once it is acquired by the infant, is difficult to check. Many imbeciles and idiots suck their fingers and wrists incessantly, causing a permanent deformity to the palate; the part sucked losing its muscular tone, and becoming unduly soft.

Netz has described an interesting case of
thumb sucking, in which the mother of the child stated that it was unusual for the child not to have its thumb in its mouth. "The maxillary arch was narrowed, with the incisors partially erupted. There was a marked lack in lateral development of the tissue surrounding the incisors. The mandibular arch was shortened as a result of the loss of the first deciduous molars which were abscessed and extracted when the patient was six years of age. She was an habitual mouth breather due to thumb sucking."

The writer is unable to agree with Clouston in his statement that "in the microcephalic and Kalmuck classes of idiots, where the brain has undergone most developmental lessening, the palate is found to be highest and most deformed." The Mongols or Kalmucks as a group, have small flattened palates. The group examined by the author comprised 25 Mongols ranging in age from 19 months to 50 years. With the exception of 3 cases under the age of 3 years whose palates were normal, only 5 cases did not present the characteristic of the flattened palate. (TABLE IV., p.70.)

The group of 20 microcephalic patients presented normal palates, with the exception of 4 whose palates were abnormal, owing to malforming habits. (TABLE VI., p.80.)

The theory, that the shape of the palate is dependent on the nasal cavity, was refuted by Clouston. It is the object of the author to show that the shape of the palate, the floor of
the antra and the shape of the nasal cavity are all so interrelated that it is hardly likely that the palate may be deformed, without the antra and floor of the nose also being affected.

It will be demonstrated in this monograph that there are numerous causes of malformed palates, that may be grouped under the following headings:

1: Habit
2: Constitutional Causes, and
3: Faulty Development of the Child;

as has been previously mentioned (p.16.)

Any circumstance, the effect of which may deform the maxilla, must also deform the floor of the nose; as the floor of the nose is composed partly of the bones of the maxilla. The antra must also be deformed, as their floor is also comprised (in part) of the bones of the maxilla.

From birth, until approximately the age of 5 years, the antra are very small in size. At the age of 1 year, the fluid capacity of each antrum is approximately 5 minims. This fluid capacity increases as the child grows; so that at the age of 5 years its capacity is 15 minims. The cubic content gradually increases as growth occurs, until adulthood is reached, when the capacity is approximately 250 minims. The accelerated increase in the growth of the antra, at about 5 years of age, coincides with the eruption of the first permanent teeth on which, if they articulate properly, depends normal occlusion. Without normal occlusion we must have a deviation
from the normal in the size or shape of the maxilla, which is partly dependent, for normal growth, on normal stress and strain transmitted through the mandible. Immediately there ensues a lack of normal development in the maxilla, and hence a lack of proper development in the antra; as part of the palate comprises the floor of the antra.

24 Barton describing the deformity in the palate brought about by thumb sucking states:

"The palate is thus pressed up into a "high arch at the expense of the nasal floor, "thus lessening the lumen available for breathing "through the nasal passages with consequent "congestion and all the associated evil results."

Prognathous jaws may originate through a number of causes, and can not be taken as a reversion to an animal or lower human type. Following are three of the causes:

1: Loss of deciduous upper teeth.
2: Enlarged tonsils.
3: Disturbance of the pituitary function.

1: Prognathous lower jaw due to the loss of the deciduous upper teeth: The development of the maxilla is dependent on normal stress and strain transmitted through the mandible. Cases may occur in which the upper deciduous teeth are lost before the proper time for their natural absorption.
The maxilla in this case, will not develop properly, as the teeth, which should receive normal shocks from the mandible, are missing. Any part of the body, to be developed normally, must be constantly used. Thus the mandible will develop to a greater extent than the maxilla, as the greatest muscle traction will still be in operation on the mandible.

When the permanent teeth erupt, they will not be in normal occlusion, on account of the slight discrepancy in the sizes of the maxilla and the mandible; with the result that the mandible may be still further protruded from its normal position.

The mandible will be exercised to an excessive degree in the mastication of food; as the patient will endeavour to obtain the greatest amount of efficiency from badly occluding teeth.

2. Prognathism due to enlarged tonsils:
The enlargement of the tonsils often causes the protrusion of the mandible to ease the constant pressure from these parts when they are inflamed. The patient, in course of time, becomes accustomed to this forward position of the mandible; and should the teeth erupt, or the cusps of the teeth interlock in this incorrect position, the protrusion then becomes, to the patient, a normal instead of an artificial position.

McKeag has also shown that "Mouth breathing associated with inflammation of the tonsils, may produce a rarer but more disfiguring condition in which the lower teeth come outside the upper and
"the child has a 'bulldog' appearance. The reason for this is somewhat obscure, but it seems that the tongue and lower jaw are pulled forward by the muscles to avoid pressure on the inflamed tonsils by the posterior part of the mandible and the palatoglossus muscle, which is attached to the side of the tongue, and forms the anterior part of the fauces. The anterior relationship of the lower jaw, if maintained for some time, becomes fixed by the interlocking of the cusps of the teeth."

3. Prognathism due to disturbance of the pituitary function: Hypertrophy of the anterior portion of the pituitary body brings about, in early life, an overgrowth of the facial bones, also of the hands and feet. The mandible appears to be selected for additional growth, and becomes prognathous.

26 Tredgold quotes the case of a boy 15 years of age. "He is 5 feet 11 inches in height, weighs 12½ stone, has massive hands, a decidedly enlarged and protruding lower jaw, and a cranial circumference of 22 inches." X-ray showed a greatly enlarged sella turcica.

27 Dewey, writing concerning the diseases of the pituitary bodies states that they "have a tendency to produce overdevelopment and are especially liable to produce overdevelopment of the mandible. Not only will it produce overdevelopment of the mandible, but it will affect the development of the other bones of the face to a certain extent, but the overdevelopment of the mandible is more apparent."
Langdon Down examined the mouths of 1000 mentally deficient patients, and he arrived at the conclusion that "in by far the larger number of instances, I was able to indicate the period at which the depressed condition commenced."

Broadly speaking, Langdon Down was able to classify mentally deficient patients by their mouths; or rather, by the shape of their palates, as either primary or secondary aments. He maintains that "In children whose idiocy is accidental, arising from causes operating after uterine life, there is but slight deviation from normal condition in the state of the mouth and teeth, while it is in those whose malady is congenital, especially where arising from causes operating at a very early period of embryonic life, that the deviation of the mouth and its appendages from a normal condition is most pronounced."

The author is unable to agree with these statements, as the results of his investigations have shown that only 24.8 per cent. of all mentally deficient patients have abnormal palates, and that 75.2 per cent. have normal palates.

The further statement of Langdon Down that "In the event of the mouth being abnormal, it indicates a congenital origin; while if the mouth be well formed, and the teeth in a healthy condition, it would lead to the opinion that the calamity had occurred subsequently to embryonic life," is contradicted by the perusal of the
results of examinations made in connection with
the microcephalic type of primary ament.

Walter Channing in the year 1896, after
prolonged and careful study of the actual casts
of the mouths of 1000 idiots, found that 40.9 per
cent. were average, typical or normal. These
terms, throughout this monograph, are synonymous.

Channing has also made reference to the
discrepancies in classifying the different types
of palates, owing to personal errors, which all
investigators are liable to make. He has fol-
lowed, to some extent, the classifications of
E. S. Talbot, which are illustrative.

Talbot's classification:

1. V-shaped.
2. Partial V-shaped.
5. Partial saddle-shaped.

The actual classification of Channing:

1. V-shaped.
2. Partial V-shaped.
5. Average or U-shaped.

The writer of this monograph, however, con-
siders that, as the abnormal shapes merge into
one another imperceptibly, it is impossible
correctly to classify the palates into any
groupings other than "Normal" and "Abnormal".

For convenience of illustration, the
classifications of Channing and Clouston other
than "Average" have been grouped in the following
table under the heading of "Abnormal."

<table>
<thead>
<tr>
<th></th>
<th>NUMBER OF PATIENTS EXAMINED</th>
<th>AVERAGE: (NORMAL)</th>
<th>AVERAGE: (ABNORMAL)</th>
</tr>
</thead>
<tbody>
<tr>
<td>CHANNING</td>
<td>1,000</td>
<td>40.9%</td>
<td>59.1%</td>
</tr>
<tr>
<td>CLOUSTON</td>
<td>169</td>
<td>11.0%</td>
<td>89.0%</td>
</tr>
<tr>
<td>CHAMPION</td>
<td>585</td>
<td>75.2%</td>
<td>24.8%</td>
</tr>
</tbody>
</table>

Channing has not discussed the causes of abnormalities in palates, but from his figures he deduces the following conclusions:

"1. Two-fifths of the palates of idiots are of fairly good shape.

"2. Palates of normal individuals may be deformed."

"3. In the idiot, it is a difference in degree and not in kind.

"4. In either case it shows irregular development anatomically.

"5. Palates of average children and idiots under eight years of age probably do not in the majority of cases markedly differ.

"6. There is no form of palate peculiar to idiocy.

"7. The statement that a V-shaped or other variety of palate is a 'stigma of degeneracy' remains to be proved."

Kingsley, an American dentist of note, is quoted by Channing as having failed to find any degree of abnormality, on examination of the palates of two hundred idiots on Randall's Island,
U.S.A. "Later he went to the Earlswood
"Institution, in England, and, in company with
"Dr. Down examined the palates of the inmates.
"He found only from 5 per cent. to 10 per cent.
"deformed to any extent, and he stated that palates
"of idiots were not different from those of ordinar-
"ary individuals coming to him for treatment."

Stoddert maintains that "generally speaking,
"the degenerate palate is too high and narrow."
He quotes another writer, however, namely
E. H. Harrison "as having examined 56 patients
"at Claybury Asylum and finding that the palate
"indicative of 'Insane Heredity' is a low, broad
"palate, which is shallow or of average depth
"(11\frac{1}{2} millimetres) opposite the first bicuspid;
"while the palate indicative of 'General Degener-
"acy' (from rickets, congenital syphilis etc.)
"is characterised by an increased depth opposite
"the first bicuspid."

Sherlock is of opinion that defects of the
hard palate occur frequently amongst mental
deficients, but that there is no connection
between mental and palatal deformities. He
states "From time to time a good deal of stress
"has been laid on the supposed connection between
"mental and palatal abnormalities.

"Without committing ourselves to any state-
"ment as to their significance, we may note that
"defects of the bony palate do, in fact, occur
"fairly frequently among the feeble-minded."
The results of the examination by Sherlock of the palates of 400 male patients and 250 female patients are given below:

<table>
<thead>
<tr>
<th></th>
<th>MALE</th>
<th>FEMALE</th>
</tr>
</thead>
<tbody>
<tr>
<td>NORMAL PALATES</td>
<td>51.5%</td>
<td>54.4%</td>
</tr>
<tr>
<td>ABNORMAL PALATES</td>
<td>48.5%</td>
<td>45.6%</td>
</tr>
<tr>
<td>TOTAL</td>
<td>100.0%</td>
<td>100.0%</td>
</tr>
</tbody>
</table>

Sherlock obtained a report from Mr. C. Edward Wallace, and portion of that report is as follows:

"An experience of many years in attending to the mouths and teeth of imbecile and epileptic children shows clearly the statements that are copied from one text book to another, as to maxillary and dental deformities, are devoid of any appreciable foundation.

"A detailed examination of some thousands of these children over a long period leads me to think that as compared with the ordinary, epileptic and imbecile children have, as a class, exceedingly well developed jaws, and are above the average as regards freedom from caries."

Stoddart states: "In a normal person the arch of the hard palate is large and wide and with a moderately high vault. Generally speaking, the degenerate palate is too high and narrow."

This generalisation, without any record of statistics compiled, is not of a very definite
character. It bears out the statement of Wallis as recorded by Sherlock, already quoted on page 38.

Duncan and Millard, dealing with the defects of the body in imbeciles and idiots, state that "Irregularity of teeth, thickness of the tongue, and a very arched palate often exist and add to the vocal difficulty, as do also the open mouth, and the thin or very inert lips."

"Inability to close the jaws, to masticate perfectly, or to swallow easily without effort, is noticed in profound idiots."

Duncan and Millard are evidently of the opinion that a high arched palate is pathognomonic of mental deficiencies. In their description of their second type of idiots they state "The deformities of the mouth, palate, tongue, and lips are seen as in the profound idiots."

Describing their third type they state: "Nearly all the defects of idiocy are to be noticed in this class."

The table on page 80 shows that abnormal maxillae were present in 20 per cent. of the microcephalics examined, and that 25 per cent. of the abnormalities occurred in patients of that class who had the pernicious malformed habit of finger and/or thumb sucking.

Both Todd and Shaw have shown that it is possible for the palate in microcephaly to be very flat or normal. Todd quotes the
case of a microcephalic whose brain capacity was only 340 cc. yet the palate was normal; whilst Shaw quotes the cases of two micro-cephalic sisters whose palates were abnormally flat.

In describing the shape of the palate in congenital mental deficient, Langdon Down is of the opinion that, with very few exceptions, namely, the palates of macrocephalic idiots, the palate is narrow, with "a markedly diminished "width between the posterior bicuspids of the two "sides."

The results of the examination of the maxillae of macrocephalic patients by the author are given on pp. 77-80. In every case the maxillae were normal, and were not excessively wide; showing that excessive growth of the cranium does not cause excessive growth of the maxilla.

REFERENCES.

3. IDDEM. ibid., p.82.
10. IDEM. ibid., p.45.
11. IDEM. ibid., p.46.
12. IDEM. ibid., p.47.
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19. IDEM. ibid.
23. IDEM. ibid., p.50.
<table>
<thead>
<tr>
<th>No.</th>
<th>Author(s)</th>
<th>Title</th>
</tr>
</thead>
<tbody>
<tr>
<td>27.</td>
<td>DEWEY, MARTIN</td>
<td>&quot;Practical Orthodontia&quot; 1920. p. 128.</td>
</tr>
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<td>CHANNING, WALTER</td>
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</tr>
<tr>
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<td>ibid.</td>
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<tr>
<td>33.</td>
<td>IDEM</td>
<td>ibid.</td>
</tr>
<tr>
<td>34.</td>
<td>IDEM</td>
<td>ibid.</td>
</tr>
<tr>
<td>36.</td>
<td>IDEM</td>
<td>ibid., p. 213.</td>
</tr>
<tr>
<td>38.</td>
<td>IDEM</td>
<td>ibid., p. 118.</td>
</tr>
<tr>
<td>41.</td>
<td>IDEM</td>
<td>ibid., p. 24.</td>
</tr>
<tr>
<td>42.</td>
<td>IDEM</td>
<td>ibid., p. 13.</td>
</tr>
</tbody>
</table>
44. SHAW, T. CLAYE, Journal Mental Science, July, 1876.

CHAPTER IV.

THE INCIDENCE OF PALATAL ABNORMALITY
IN PERSONS OF NORMAL INTELLIGENCE.

An oral examination of a number of persons of ordinary intelligence was undertaken by the author with the object of determining the incidence of abnormal palatal development.

The ages of the subjects ranged from 6 years to 35 years. All patients were excluded who were wearing dentures, either partial or complete. The subjects were not taken indiscriminately, but were examined in consecutive order, as they presented themselves for professional service at the surgeries of the author. All the inmates of a boarding school for girls were also examined.

The results of these examinations were as follows:

<table>
<thead>
<tr>
<th>NUMBER OF PATIENTS</th>
<th>NORMAL PALATES</th>
<th>ABNORMAL PALATES</th>
</tr>
</thead>
<tbody>
<tr>
<td>280</td>
<td>182 or 65%</td>
<td>98 or 35%</td>
</tr>
</tbody>
</table>

The palatal deformity that is usually associated with thumb sucking and mouth breathing namely, a high constricted V-shaped type, was found to predominate. No cases of cleft palate were found amongst those with abnormal palatal defects.
CHAPTER V.

THE INCIDENCE OF PALATAL ABNORMALITY IN MENTAL DEFICIENTS.

The accompanying analysis of results obtained from the examination of 585 mentally deficient patients shows that 349 or 45.8 per cent. of that number were primary aments, and 226 or 54.2 per cent. were classified as secondary aments. Ten patients could not be definitely classified owing to lack of clinical characteristics and family history. These have been entered in the analysis as an indeterminate group.

INCIDENCE OF SYPHILIS

It is interesting to note that 55 or 9.4 per cent. of the patients examined gave positive Wassermann reactions.

Various writers differ greatly in their statements concerning the proportion of positive Wassermann reactions in connection with mental deficient. Dean examined 330 mentally deficient patients, and found a positive reaction in 51, giving a percentage of 15.4. Sherlock without the aid of a Wassermann reaction, after examining 90 patients, gave the percentage as 14.4.

Tredgold gave the percentage as 2.5, in connection with the patients examined by him. The examination in this case, however, was not aided by the Wassermann reaction; Tredgold depended entirely on clinical evidence. He states:
"Raviart and others obtained a positive reaction "in 30 per cent. of cases."

INCIDENCE OF PALATAL ABNORMALITIES.

It was found by the author that, of the 349 primary aments, representing 45.8 per cent. of the total number of mentally deficient patients examined, abnormal palates were present in 97 cases, representing 27.7 per cent.

Of the total number of mentally deficient patients examined, 226 were classified as being of the secondary type, representing a percentage of 54.2. Abnormal palates were discovered in 46 cases, representing 20.3 per cent.

Grouping the primary and secondary aments, it was found that 24.8 per cent., or 143 cases of the grand total of the cases examined (585) had abnormal palates.

The results obtained from a strict examination show that approximately 25 per cent. only of mentally deficient patients had abnormal palates.

REFERENCES.


3. THEDGOLD, A.F., "Mental Deficiency",1922.p.49
## TABLE III

**Table Showing the incidence of palatal defects and of syphilis in mental deficiency.**

<table>
<thead>
<tr>
<th>Classification</th>
<th>Number Examed</th>
<th>Normal Palates</th>
<th>Abnormal Palates</th>
<th>Positive Wassermann Reaction</th>
</tr>
</thead>
<tbody>
<tr>
<td>Primary Aments</td>
<td>349</td>
<td>252</td>
<td>97</td>
<td>3</td>
</tr>
<tr>
<td>Secondary Aments</td>
<td>226</td>
<td>130</td>
<td>46</td>
<td>52</td>
</tr>
<tr>
<td>Indeterminate</td>
<td>575</td>
<td>432</td>
<td>143</td>
<td>55</td>
</tr>
<tr>
<td></td>
<td>10</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>585</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Thus: 143 out of 575 classified mentally deficient patients had abnormal palates, i.e., 24.8 per cent.

97 out of 349 primary aments had abnormal palates, i.e., 27.7 per cent.

46 out of 226 secondary aments had abnormal palates, i.e., 20.3 per cent.

55 out of 585, the total number of patients examined, had positive Wassermann reactions, i.e., 9.4 per cent.
CHAPTER VI.

A DESCRIPTION OF CLINICAL TYPES OF MENTAL DEFICIENTS WITH SPECIAL REFERENCE TO ORAL CONDITIONS.

CLASSIFICATION OF PATIENTS

GROUP 1. (a) MONGOLS

{ (b) MONGOLOIDS.

2. MICROCEPHALIC AMENTS
3. HYDROCEPHALIC AMENTS
4. ACHONDROPLASTIC AMENTS
5. OXYCEPHALIC AMENTS
6. SYPHILITIC AMENTS
7. MENTAL DEFICIENTS WITH CLEFT PALATES.

GROUP 1 (a) MONGOLS. (TABLE IV. p.70.)

Of the 585 mentally deficient patients examined by the author, 25 or 4.2 per cent. were clinically classified as Mongols.

At the present time, the actual cause of Mongolism is obscure. It is thought to be due to endocrine disturbance. This interesting group of patients in the primary amentia classification, have a number of clinical features which distinguish them. Brousseau gives the physical characteristics as follows:

Growth: Stunted, but uniform.
Head: Brachycephalic.
Forehead: Smooth, normal — depressed temples.
Palate: High, arched, narrow.
Tongue: After 5 years of age, fissured.
Teeth: Delayed eruption.
Nose: Bridge depressed.
Eyes: Palpebral fissures, oblique, generally upwards and outwards.
Skin: Smooth in childhood, coarse and dry in adulthood.
The above characteristics describe the clinical aspect of Mongolism. The similarity of appearance in Mongolism is so striking that all individuals of this group appear to have a family resemblance to one another.

**MUCOUS MEMBRANE:** The appearance of the oral mucous membrane is most striking; it being in a condition of marked hyperaemia. The gums are considerably inflamed and swollen, and pus may be seen exuding from the gingival margins of the teeth.

Of the 25 Mongols examined, only 8 could be described as having a normal oral mucous membrane. In all the other cases, the inflammation varied from a gingivitis to a marked pyorrhoea alveolaris. The periodontal ligament seemed to have had no elasticity, being without tone, and allowing excessive tooth movement.

Hume noticed this hyperaemic condition of the mucous membrane covering the gums in a Mongol under his care. Brousseau has also noted that the teeth in Mongols "are loosely set in their sockets."

**TONGUE:** The mucous membrane covering the tongue is particularly sodden, and the fissuring
of the tongue is a noteworthy feature.

The examination of the tongues of the 25 patients in the Mongol group showed that 5 cases or 20 per cent., had normal tongues, the remainder having tongues fissured in varying degree. Of these 5 cases, however, 2 were under the age of 5 years. Brousseau notes that the tongue does not fissure early in life; so that, in all probability, the 20 per cent., featuring normal tongues would, in later life, be reduced to 12 per cent., equal to only 3 cases exhibiting normal tongues.

Both Brousseau and Tredgold quote Thomson as having stated that "The fissuring usually appears about the third or fourth year; it may be a little earlier or may be delayed until the sixth year."

The remaining 80 per cent. of patients had tongues in which the fissuring varied. Some tongues were uniformly fissured, whilst others showed lateral fissuring only.

TONGUE SUCKING: This is a habit which seems almost peculiar to Mongols; it is rarely met with in other mental deficient. It is a remarkable habit, and I feel sure that the fissured, enlarged tongue is the outcome of indulging in it. The action involved in this habit of tongue sucking, which habit might occur amongst mental deficient other than Mongols, would not have the same effect on those other mental deficient, owing to the normal tone of the tongue. When it is practised by Mongols,
however, in whom is present a permanent hyperaemic condition of the oral mucous membrane, fissuring occurs.

Of the 25 Mongols examined, it was found that 20 cases, representing 80 per cent. had this pernicious habit of tongue sucking and the inevitable fissured tongue. The remaining 4 patients who had normal tongues, were not addicted to tongue sucking. One had slight gingivitis, and 3 had normal oral mucous membranes. It will be noted that the patient aged only 19 months (N.G. figs., 2 & 6, pp. 54-55) has not been taken into account, as fissuring is not a common feature at that early age.

PALATES: In the course of investigations dealing with the abovementioned group of 25 Mongols, (whose ages, as previously stated, ranged from 19 months to 50 years), a feature common to most was noticed namely, a small, flattened palate. In those of the group who did not possess this feature, it was observed that the palates were of a high gothic type; and upon investigation, it was found that these patients practised the habit of thumb sucking.

Although Mongols, as a class, are subnormal in stature, the maxillae in particular are exceptionally small.

This discovery of a small flattened palate is in direct antagonism to the findings of
Telford Smith, Brousseau and Tredgold, but is substantiated by the statement of Langdon Down, who, while discussing a paper "Mongolian Imbecility," by G. E. Shuttleworth, said: "Mongols stood apart from other imbeciles in respect of palatal development; the bony structures of their face suffered in development, as well as other parts. This led to narrowing of their nasal passages and smallness of their jaws."

Brousseau quotes Telford Smith as follows: "The palate is always higher than normal, this condition, however, being exaggerated in appearance by the almost invariable thickening and hypertrophy of the gums all around the alveolar curve as a rule is fairly good."

Brousseau states: "The hard palate is often malformed in Mongolism. It is generally high, narrow, V-shaped. There is frequently a median ridge and the jaw is wedge shaped."

Tredgold states: "The palate is often high and narrow, the mouth open and the tongue partly protruding."

The author is of the opinion that there is a definite connection between the small, flattened palate and the enlarged, fissured tongue in Mongolism.

Practitioners are all aware of the abnormalities which form in the palate as a result of
thumb sucking namely, a high constricted palate, with ill-developed nasal airways, which give rise to mouth breathing.

The constant tongue sucking tends to lower the dome of the palate. Also, the continued passing over in the mouth, acting on mucous membrane having a lowered resistance, tends to aggravate alveolar absorption; thus preventing the proper growth of bone, and hastening its absorption.

**DENTITION:** The teeth, in Mongolism, do not erupt at the correct period in life; the eruption being invariably retarded. The permanent teeth are small in size, lacking in proper shape; and the canines do not possess character. These permanent teeth may be confused with the temporary dentition, owing to the fact that they lack the appearance of the permanent dentition in normal persons.

**Caries:** This affection is present in Mongols, but not to the extent that it appears in the mouths of other types of beings.

Broderick, in a series of articles, has given some excellent reasons why caries is not so prevalent in mouths affected by pyorrhoea alveolaris.

As previously stated, the oral mucous membrane
Fig. 2.
N.... G...., Mongol;
Aged 19 months.
Tongue normal.

Fig. 3.
J.... F...., Mongol;
Aged 8 years.
Tongue commencing to fissure;
Slight gingivitis.

Fig. 4.
I.... H...., Mongol, aged 4 yrs.
Habit of finger sucking commencing.
L... H..., Mongol, aged 4 years.
Tongue normal; arches normal.

Fig. 5.

N... G..., Mongol, aged 19 months
Tongue normal;
Positive Wasserman;
Dentition delayed.

Fig. 6.
P... R..., Mongol, aged 11 years. Fissured tongue; alveolar absorption. Palate is small and high; the patient sucks his thumb as well as his tongue. Photograph of cast actual size.
Fig. 10.

T.... B...., Mongol;
Aged 10 years.
Tongue slightly roughened;
just commencing to fissure.
No alveolar absorption.
Photograph of model actual size.

Fig. 11. (Model No. 3).
Fig. 12.
E.... N...., Mongol; Aged 16 years.
Tongue normal; arches small, but normal.
Neither gingivitis nor alveolar absorption.

Fig. 13. (Model No. 4.)
Fig. 14.
J.... K...., Mongol; Aged 17 years.
Tongue fissured; Alveolar absorption; Small, flattened palate.
Photograph or model actual size.

Fig. 15. (Model No. 5).
J. B., Mongol, aged 14 yrs.
Tongue slightly fissured;
Alveolar absorption;
Palate small.
Photograph of model actual size.

Fig. 17. (Model No. 6.)
Fig. 18.
H...M..., Mongol, aged 12 yrs.
Tongue fissured;
Alveolar absorption;
Flattened palate.
Photograph of model actual size

Fig. 19. (Model No. 7.)
K... C... aged 15 years. Mongol. Photo. of model actual size. Fissured tongue; alveolar absorption; flattened palate.
W. D., Mongol, aged 20 yrs.

Tongue fissured;
Alveolar absorption;
Palate small, wide & flattened.

Photograph actual size of cast.
Fig. 25.
C... G... Y..., Mongol;
Aged 17 years.

Fissured tongue;
Alveolar absorption;
Small flattened palate;

Photograph actual size or cast.

Fig. 26. (Model No. 10).
Fig. 27.
T.... H....., Mongol;
Aged 30 years.
Patient edentulous.
Fissured tongue.
Palate very small and shallow.

Fig. 28.
H.... N....., Mongol;
Aged 24 years.
Patient edentulous;
Fissured tongue;
very small palate.
Fig. 29.
E... H...ne, Mongol;  
Aged 24 years.  
Fissured tongue;  
Alveolar absorption;  
Palate high and small;  
Thumb sucker.  
Photograph or model actual size.

Fig. 30. (Model No. 11).
Fig. 31.
M.... G...., Mongol;
Aged 25 years.
Tongue fissured;
Edentulous;
Small, high palate;
Thumb sucker.

Fig. 32.
M... H...., Mongol, aged 7
Tongue fissured;
Edentulous;
Small, flattened palate.
Fig. 33.
M... O.M., Mongol; Aged 20 years.
Slightly fissured tongue; Alveolar absorption; Palate small and flattened.
Photograph exact size of cast.

Fig. 34. (Model No. 12).
J. . . . B. . . ., Mongol; aged 26 years.
Small, flattened palate, and fissured tongue. Photograph of model actual size.

A. . . . M. . . ., Mongol; aged 47 years.
Fissured tongue, small, flattened palate. Photograph of model actual size.
<table>
<thead>
<tr>
<th>INITIALS</th>
<th>AGE</th>
<th>HABIT</th>
<th>TONGUE</th>
<th>CONDITION OF ORAL MUCOUS MEMBRANE</th>
<th>PALAT.</th>
<th>NASSER-MANN.</th>
<th>REMARKS</th>
<th>ILLUSTRATION</th>
</tr>
</thead>
<tbody>
<tr>
<td>L.R.</td>
<td>4 yrs</td>
<td>Finger Sucking.</td>
<td>do.</td>
<td>do.</td>
<td>do.</td>
<td>Neg.</td>
<td>Teeth late in erupting</td>
<td>Figs. 4 &amp; 5</td>
</tr>
<tr>
<td>J.P.</td>
<td>8 yrs</td>
<td>Tongue Sucking.</td>
<td>Slightly</td>
<td>Slight Gingivitis.</td>
<td>do.</td>
<td>do.</td>
<td>do. do. do. do.</td>
<td>Fig. 3</td>
</tr>
<tr>
<td>T.B.</td>
<td>10 yrs</td>
<td>Thumb Sucking.</td>
<td>Normal.</td>
<td>do.</td>
<td>High.</td>
<td>do.</td>
<td>Mil.</td>
<td>Figs. 10 &amp; 11, Model No. 3</td>
</tr>
<tr>
<td>P.R.</td>
<td>11 yrs</td>
<td>Tongue &amp; Thumb Sucking.</td>
<td>Pissured.</td>
<td>Pyorrhoea Alveolaris.</td>
<td>Small.</td>
<td>High.</td>
<td>do. do. do.</td>
<td>Figs. 7, 8 &amp; 9, Model No. 2</td>
</tr>
<tr>
<td>H.M.</td>
<td>12 yrs</td>
<td>Tongue Sucking.</td>
<td>Slightly Pissured.</td>
<td>Pyorrhoea.</td>
<td>Small.</td>
<td>Flat.</td>
<td>do. do. do.</td>
<td>Figs. 18 &amp; 19, Model No. 7</td>
</tr>
<tr>
<td>J.E.</td>
<td>14 yrs</td>
<td>Thumb &amp; Tongue Sucking.</td>
<td>do.</td>
<td>do.</td>
<td>Small.</td>
<td>High.</td>
<td>do. do. do.</td>
<td>Figs. 16 &amp; 17, Model No. 6</td>
</tr>
<tr>
<td>K.C.</td>
<td>15 yrs</td>
<td>Tongue Sucking.</td>
<td>do.</td>
<td>Wide Arch.</td>
<td>do.</td>
<td>do. do.</td>
<td>do. do. do.</td>
<td>Figs. 20, 21 &amp; 22, Model No. 8</td>
</tr>
<tr>
<td>C.C.</td>
<td>17 yrs</td>
<td>Tongue Sucking.</td>
<td>Pissured.</td>
<td>Pyorrhoea.</td>
<td>Small.</td>
<td>Flat.</td>
<td>do. Alveolar absorption</td>
<td>Figs. 25 &amp; 26, Model No. 10</td>
</tr>
<tr>
<td>J.O'N.</td>
<td>20 yrs</td>
<td>Thumb &amp; Tongue Sucking.</td>
<td>do.</td>
<td>do.</td>
<td>do.</td>
<td>do. do.</td>
<td>do. do. do.</td>
<td>Figs. 33 &amp; 34, Model No. 12</td>
</tr>
<tr>
<td>L.C.</td>
<td>25 yrs</td>
<td>do. do.</td>
<td>do.</td>
<td>do.</td>
<td>Small.</td>
<td>High.</td>
<td>Edentulous</td>
<td>Fig. 31</td>
</tr>
<tr>
<td>T.H.</td>
<td>30 yrs</td>
<td>do. do.</td>
<td>do.</td>
<td>do.</td>
<td>do.</td>
<td>do. do.</td>
<td>do. do. do.</td>
<td>Fig. 27</td>
</tr>
<tr>
<td>T.H.</td>
<td>24 yrs</td>
<td>do. do.</td>
<td>do.</td>
<td>Severe Gingivitis</td>
<td>Small.</td>
<td>Flat.</td>
<td>do. Almost edentulous</td>
<td>Figs. 29 &amp; 30, Model No. 11</td>
</tr>
<tr>
<td>J.B.</td>
<td>26 yrs</td>
<td>do. do.</td>
<td>do.</td>
<td>Normal.</td>
<td>Very Small.</td>
<td>do.</td>
<td>do. do.</td>
<td>Fig. 37</td>
</tr>
<tr>
<td>A.M.</td>
<td>47 yrs</td>
<td>Tongue Sucking.</td>
<td>Pissured.</td>
<td>Pyorrhoea.</td>
<td>Small.</td>
<td>Flat.</td>
<td>do. do. do.</td>
<td>Figs. 33 &amp; 34, Model No. 15</td>
</tr>
<tr>
<td>W.D.</td>
<td>20 yrs</td>
<td>do. do.</td>
<td>do.</td>
<td>Pyorrhoea.</td>
<td>do.</td>
<td>do. do.</td>
<td>Alveolar absorption</td>
<td>Figs. 23 &amp; 24, Model No. 9</td>
</tr>
<tr>
<td>J.K.</td>
<td>17 yrs</td>
<td>Thumb &amp; Tongue Sucking.</td>
<td>do.</td>
<td>do.</td>
<td>Small.</td>
<td>do. do.</td>
<td>do. do. do.</td>
<td>Figs. 14 &amp; 15, Model No. 5</td>
</tr>
</tbody>
</table>
in Mongolism, possesses very little power of resistance to infection, being constantly in a state of inflammation.

Of the 25 patients belonging to the group under discussion, 5 were edentulous. The remaining 20 patients included only 3, who were not affected with gingivitis or pyorrhoea alveolaris.

Rumel noted the presence of pus in the swollen, hyperaemic, inflamed gums of a Mongol whom he examined. He also noted the lack of character in the teeth present, stating: "He had no permanent dentition although 18 years old."

Of the many cases of adults in the Mongol class examined by the writer, there were none in which the permanent dentition had not erupted; as previously stated, however, this dentition was greatly lacking in the appearance characteristic of the normal permanent dentition.

WASSERMANN REACTION: Only 1 case of Mongolism gave a positive Wassermann reaction, 24 cases being negative.

GROUP 1 (b) MONGOLOIDS. (TABLE V).

Very closely approximating the clinical characteristics of Mongols are those of a group of mental deficient known as Mongoloids. These patients have some of the stigmata of Mongolism in their appearance, but fall short of being true Mongols, hence the term "Mongoloids."
Amongst the 585 patients examined by the author, only 3 belonged to this class, on the border line, as it were, of Mongolism. The Mongoloids concerned had palpebral fissures which were somewhat oblique. Hands, tongue and physique were normal; but the mentality was subnormal, as in true Mongolism.

They did not practise the habit of tongue sucking. Their oral mucous membranes were normal, so that their tongues were not fissured as is the case in true Mongols. The Wassermann test in all 3 cases was negative.

**CASE 1:**
J.A., (Figs. 38 & 39).(Model No. 15.)

**Mouth:** Normal in size, with a well developed and normally domed maxilla.

**Mucous Membrane:** Of normal tone, the tongue not fissured.

**Breathing:** Natural (through the nose.)

**CASE 2:**
E.K., (Figs. 40 & 41).(Model No. 16.)

**Mouth:** Normal in size, with well-developed and normally domed maxilla.

**Mucous Membrane:** Of normal tone, the tongue not fissured.

**Breathing:** Natural (through the nose.)
<table>
<thead>
<tr>
<th>INITIALS</th>
<th>AGE</th>
<th>HABIT</th>
<th>TONGUE</th>
<th>CONDITION OF ORAL MUCOUS MEMBRANE</th>
<th>PALATE</th>
<th>WASSERMANN</th>
<th>REMARKS</th>
<th>ILLUSTRATION</th>
</tr>
</thead>
</table>
J... A..., Mongoloid, 19 years.
Normal tongue, normal mouth.
Photograph actual size of cast.
Fig. 40.
E. K. ....y, Mongoloid;
Aged 12 years.

Normal tongue;
Normal mouth.

Photograph or cast
actual size.

Fig. 41. (Model No. 16).
Fig. 42.
R.... S...., Mongoloid; Aged 21 years.
Normal tongue, abnormal palate.
Singer sucker & mouth breather.
Photograph of model actual size.

Fig. 43. (Model No. 17).
CASE 3: R.S. (figs. 42 & 43.) (Model No. 17.)
Mouth: This mouth had not the normal development of the two preceding cases, the reason being that, during the waking hours, finger sucking was practised; and during sleep, the posture was on the broad of the back, with the mouth wide open.

Mucous Membrane: Slightly congested, gingivitis being present. Tongue not fissured; palate high and not normally developed.

Breathing: Patient definitely a mouth breather.

GROUP 2. MICROCEPHALICS. TABLE VI. p. 80.

After the examination of 585 mentally deficient patients, 20 were found who could be classified into the primary amentia group as microcephalics, the percentage being 3.4 in respect of the total number examined.

The clinical aspect in microcephaly is very distinctive. A smaller head than normal is evidenced; but more diagnostic than the small head are the recession about the frontal region, and the characteristic flattening in the occipital area. This recession and flattening are
clearly illustrated in 3 of the patients namely D.A., (Figs. 57 & 58) (Model No. 24), V.L., (Figs. 46, 47 & 48) (Model No. 19) and G.S., (Figs. 44 & 45) (Model No. 18).

Tredgold states that many primary aments, having a head measurement of 19 inches, may be classified as microcephalics; provided that the recession in the frontal region, and the flattening in the occipital region are present.

Of the 20 cases examined by the author, 4 only exhibited abnormal palates. Of these abnormal cases, 3 were definitely finger or thumb suckers, and the remaining patient was a Mongol, whose palate was flattened and small.

Two cases are of especial interest:

**CASE NO. 1:** G.S., (Figs. 44 & 45) (Model No. 18)
A female aged 14 years, with a head measurement of only 14 inches; mouth normal, the palate being well domed and rounded; the patient breathing in a normal manner. A mischievous person, hopping about on one leg, keeping the ward in a state of ferment.

**CASE NO. 2:** V.L., (Figs. 46, 47 & 48.) (Model No. 19.)
A female, aged 18 years, with a head measurement of only sixteen inches. Habit present
of sucking 2 fingers, rendered sodden by the action of sucking. Mouth abnormal: jaws very constricted, palate exceptionally high and narrow. This patient also had the habit of annoying other patients, always moving about, and never still.

15 Clouston maintains that "In the Microcephalic and Kalmuck (Mongol) classes of idiots, "where the brain has undergone most developmental lessening, the palate is found to be highest and "most deformed." The writer found abnormal palates to be present in only 20 per cent. of the microcephalics examined, the remaining 80 per cent. being normal.

Two other writers namely, Todd and Shaw, have shown that microcephalic aments may have flattened palates, in contradistinction to Clouston's statements.

Todd, writing on the subject of microcephaly, describes the case of a female "whose cranial "capacity was only 340 cc., instead of the "normal 1340 cc. In spite of the fact that "her cranium is so very small, the face is "nearly of normal size and the jaws are quite as "large as one would expect in a woman of her "height."
### TABLE VI
ANALYSIS OF 20 CASES OF MICROCEPHALY.

<table>
<thead>
<tr>
<th>INITIALS</th>
<th>HEAD MEASUREMENT (IN INCHES)</th>
<th>HABIT. (MALE-FORMING)</th>
<th>WASSERMANN</th>
<th>PALATE.</th>
</tr>
</thead>
<tbody>
<tr>
<td>M.P.</td>
<td>17½</td>
<td>Nil</td>
<td>Negative</td>
<td>Normal</td>
</tr>
<tr>
<td>P.E.</td>
<td>17½</td>
<td>do.</td>
<td>do.</td>
<td>do.</td>
</tr>
<tr>
<td>N.M.</td>
<td>18½</td>
<td>do.</td>
<td>do.</td>
<td>do.</td>
</tr>
<tr>
<td>E.W.</td>
<td>18</td>
<td>do.</td>
<td>do.</td>
<td>do.</td>
</tr>
<tr>
<td>A.L.</td>
<td>18</td>
<td>do.</td>
<td>do.</td>
<td>do.</td>
</tr>
<tr>
<td>S.W.</td>
<td>18</td>
<td>do.</td>
<td>do.</td>
<td>do.</td>
</tr>
<tr>
<td>D.A.</td>
<td>18½</td>
<td>do.</td>
<td>do.</td>
<td>do.</td>
</tr>
<tr>
<td>B.R.</td>
<td>17</td>
<td>do.</td>
<td>do.</td>
<td>do.</td>
</tr>
<tr>
<td>S.C.</td>
<td>16½</td>
<td>do.</td>
<td>do.</td>
<td>do.</td>
</tr>
<tr>
<td>G.S.</td>
<td>14</td>
<td>do.</td>
<td>do.</td>
<td>do.</td>
</tr>
<tr>
<td>J.O'C.</td>
<td>18</td>
<td>do.</td>
<td>do.</td>
<td>Small, flat.</td>
</tr>
<tr>
<td>V.L.</td>
<td>16</td>
<td>Sucks 2 fingers</td>
<td>do.</td>
<td>High, constricted</td>
</tr>
<tr>
<td>S.H.</td>
<td>17½</td>
<td>Nil</td>
<td>do.</td>
<td>Normal</td>
</tr>
<tr>
<td>J.C.</td>
<td>15</td>
<td>do.</td>
<td>do.</td>
<td>do.</td>
</tr>
<tr>
<td>F.H.</td>
<td>17</td>
<td>do.</td>
<td>do.</td>
<td>do.</td>
</tr>
<tr>
<td>W.S.</td>
<td>15</td>
<td>do.</td>
<td>do.</td>
<td>do.</td>
</tr>
<tr>
<td>J.P.</td>
<td>17</td>
<td>Sucks thumb</td>
<td>do.</td>
<td>High, constricted</td>
</tr>
<tr>
<td>W.S.</td>
<td>17½</td>
<td>Nil</td>
<td>do.</td>
<td>Normal</td>
</tr>
<tr>
<td>J.C.</td>
<td>18½</td>
<td>do.</td>
<td>do.</td>
<td>do.</td>
</tr>
<tr>
<td>J.H.</td>
<td>16½</td>
<td>Sucks thumb</td>
<td>do.</td>
<td>High, constricted</td>
</tr>
</tbody>
</table>

TOTAL: 20 Microcephalics out of 585 patients, or 3.4%. 4 abnormal palates, representing 20% of the Microcephalics examined.
Fig. 44.
G.... S...., Microcephalic.
Head measurement 14\".

Palate normal in size & shape.
Photograph actual size of model.

Fig. 45. (Model No. 18).
V. L., Microcephalic. Head measurement 16".
Palate abnormal, due to finger sucking.
P....E., Microcephalic.

Head measurement 17\(\frac{1}{2}\) \".

Photograph actual size of model.
Fig. 51.
S.... W...., Microcephalic.
Head measurement 18''.

Photograph of model actual size.

Fig. 52. (Model No. 21).
Fig. 53.
B... R...., Microcephalic.
Head measurement 17".
Palate normal in size & shape.
Photograph of cast actual size

Fig. 54. (Model No. 22).
Fig. 55.
M. M...., Microcephalic.
Head circumference 18½ ".
Teeth crowded, but palate normal in size and shape.
Photograph actual size of model.

Fig. 56. (Model No. 23).
Microcephalic.
Head measurement 16\(\frac{1}{2}\) ".

Palate normal in size & shape.

Photograph of model actual size.
Fig. 59.
1..... L...., Microcephalic.
Head measurement 18".

Photograph of cast actual size.

Fig. 6C. (Model No. 25).
Fig. 61. M... P..., Microcephalic.
Head measurement 17½".
Photograph of model actual size

Fig. 62. (Model No. 26).
Fig. 63. (Model No. 27).
S... W...., Microcephalic.
Head measurement 18".
Palate normal, in size and shape.
Photograph actual size.
Fig. 64.
S... C..., aged 13 years.
Microcephalic.
Head measurement $16\frac{1}{2}$ ".

Palate normal in size & shape.
Photograph actual size of cast

Fig. 65. (Model No. 28).
Shaw, in an article dealing with the palates of idiots, describes the cases of two microcephalic sisters and found that "they give, "as a matter of fact, the lowest vertical heights "of any that I have taken, including persons of "high intelligence."  

The Wassermann reaction was negative in all the cases of microcephalic amentia examined by the writer.

**GROUP 3: HYDROCEPHALIC AMENTS.**

Three patients only could be classified as Hydrocephalic Aments out of the 585 mental deficient examined.

Tredgold states that the life of a hydrocephalic ament is usually very short; and that "there can be no doubt that a great por-
"tion of children so affected die within a "few years."

The clinical signs of hydrocephaly render diagnosis easy. The skull assumes a globular shape, being expanded evenly in all directions. The causation is obscure; but, whatever the cause, the characteristic phenomenon is an accumulation of fluid inside the skull.

Of the 3 cases examined, only 1 was a progressive hydrocephalic; the growth of the head being half an inch in general circumference.
in one year. This case, which shall be styled CASE NO. 1, was that of a female, A.J., (Figs. 66 & 67), aged 17 months; the head measurement was 27 inches. The mouth was quite normal in size and shape; the dentition was retarded, only 4 lower incisors being present. The vitality was very low; no impression was taken in order to obtain a dental cast; nor was any Wassermann reaction test considered.

CASE NO. 2: J.H., (Figs. 79, 80 & 81). A male patient, aged 3 years, with a head measurement of 21.5 inches. This patient gave a definite positive Wassermann reaction. The size and shape of the palate were quite normal. Hutchinson's incisor teeth were present, together with Moon's domed molars.

CASE NO. 3: E.G., (Figs. 68 & 69.) (Model No. 29.) A female patient aged 21 years, with a head measurement of 22.5 inches. This patient also gave a definite positive Wassermann reaction. The mouth was normal in size and shape. The dentition gave no indication of the positive Wassermann reaction.

19 Tredgold describes 3 cases of hydrocephaly for illustrative purposes; and in only one case does he indicate the oral conditions. With reference to this particular case this authority states: "The palate was high and saddle-shaped, "the teeth irregular."
Fig. 66.

A.... J...., Hydrocephalic.
Head measurement 27 " ...
Mouth normal in size & shape.
Dentition delayed.

Fig. 67.
Fig. 68.
E.... G...., Hydrocephalic.
Aged 21 years.
Head measurement 22\frac{1}{2}".
Photograph of model actual size.

Fig. 69. (Model No. 29).
Ireland states (concerning hydrocephaly): "But the palate is not vaulted as is so common "with genetous idiots and the teeth are regular "and often good."

In his illustrative cases, 3 in number, Ireland states:

First Case: "The teeth were not good, "and the palate somewhat high, like that of a "young child but not vaulted." 21

Second case: "The palate was somewhat "high." 22

Third Case: No mention of oral conditions. 23

In the 3 cases examined by the writer, the mouths were normal in size and shape.

GROUP 4: ACHONDROPLASTIC ANOMALIES.

The term "achondroplastic" is applied to those patients, whose stature is dwarfed as a result of lack of growth in the extremities. The head is normal in size. The cause is unknown.

This condition was found in only 2 cases amongst the 585 patients examined. In both these cases, illustrations of which are shown, the mouths were normal.

Case 1: M.C., (Figs.70 & 71),(Model No. 30.)

This patient was considerably dwarfed. The mouth as mentioned above, was normal in size and shape - for the age of the patient. The six year
molars were already in place, and the incisors about to erupt.

CASE 2. K.W., (Figs. 72 & 73.) (Model No. 31)
This patient was not dwarfed to the same extent as the previously mentioned case. The mouth was also normal in size and shape. The six year molars were in their correct positions, and the left upper central tooth was partially erupted. This patient had a sister — a Mongol — who was an inmate of the same institution.

Brousseau states that, in achondroplasia, "the upper jaw is broad, spreads far apart and is prognathous; the palate has a backward displacement and a tilting of its posterior end".

In the 2 cases examined by the writer, the palate was normal in all respects when compared with the normal palate of an ordinary intelligent child of the same age.

Moir has reported the case of a Chinaman whose physical condition showed that he suffered from achondroplasia.

This dwarfed Chinaman, whose height was 42½ inches, when examined orally, was found to have a normal vaulted palate. Moir states, "the head is large and globular, the occipital bones being well developed. The bridge of the nose is depressed, the lids and eyelids are not thickened; the teeth are nearly all gone; the vault of the palate is normal in appearance."
Fig. 70.
M... C... H..., Aged 8 years.
Achondroplasia.
Photograph of cast actual size.

Fig. 71. (Model No. 30).
Fig. 72.
X.... W...., aged 9 years.
Achondroplasia.
Photograph of cast actual size.

Fig. 73. (Model No. 31).
GROUP 5: OXYCEPHALIC AEMENTS.

Oxycephaly is a condition rarely found in mental deficient, 2 cases being in evidence amongst the 585 patients examined. The clinical features, as described by Tregold are as follows:

"In well marked examples the skull is abnormally high in the frontal regions, from which it rises to a more or less sharp point at the vertex; with this there is a lessened development of the supra-orbital ridges and frontal eminences. The palpebral fissures slant downwards and outwards, and with this there is marked exophthalmos".

Greig describes oxycephaly as "a congenital malformation associated with synostoses and though there may be coincident developmental defects, presents as a constant feature a characteristic deformation of the skull."

Oxycephalic patients are so rare that it is impossible to formulate any definite rule as to the shape of their palates.

CASE NO. 1: M.S. (Figs. 74, 75 & 76.) (Model No. 32.) A female, aged 19 years. Positive Wassermann reaction. Palate minute and constricted, with longitudinal fissurations; so diminutive, in fact, that the patient had extreme difficulty in formulating her words, and the enunciation was very indistinct. The anterior five teeth were definitely bicuspid, the cingula being very well developed. The upper right canine was
unrupted. The upper right twelve year and
six year molars, together with the second
bicuspids were misplaced; being in a palate-
buccal direction instead of an anterior-posterior
direction. The upper left second bicuspids was
placed buccally to the sixth year molar. There
was a remnant of a temporary tooth between the
first bicuspids and molar. To obtain space in
order to improve the speech of the patient, the
teeth posterior to the first bicuspids on each
side were extracted.

THE SAME CASE AFTER 2 YEARS. (Figs. 74 & 76.)
(Model No. 33.) This illustration shows the space
gained in the palate, and also shows the two
wisdom teeth erupting into position. The
upper right canine is still unerupted.

Though the Wassermann test was positive, no
indication of syphilis was observed in the teeth.
The six year molars were not domed (after Moon);
nor did the incisors appear as typical Hutchin-
son's teeth. The patient was a mouth breather.

CASE NO. 2. Z.M., (Figs. 77 & 78.) (Model
No. 34.) A female aged 16. Wassermann reaction
negative. The hair of this patient was wavy and
brown, the direct antithesis of that of the
previously mentioned case, which was straight,
black and coarse. The palate was high and
constricted, the rugae being well developed. The
teeth also were well developed, and of a strong
virile type. The arch was irregular; the
lateralis being posterior to the centrals, with canines erupting anteriorly to their correct positions. A considerable amount of hypertrophied tissue was present due to the chronic irritation between the canines, laterals and centrals. This patient was a mouth breather.

Following are cases reported by Greig (with reference only to their palatal areas:)

"CASE 1: C.R. 56 years. His respiration was mainly oral. The upper teeth were crowded together and irregularly spaced. The palate was high and narrow; the soft palate was cleft and the pharynx small, a combination which explained the indistinctness of his speech.

"CASE 2: M.E. 58 years. The face seemed somewhat flat and the cheek bones high, appearance perhaps dependent on want of development of the maxillary sinuses. Except for a few stumps the teeth had been lost. The palate was high arched and narrow.

"CASE 3: T.H. 45 years. The nose was fairly well developed, but deflected to the left. His upper teeth were irregular and closely packed, his palate was narrow and high and the pharynx small. Respiration mostly oral.

"CASE 4: Description of a macerated oxycephalic skull: The transverse diameter of the maxillae is much smaller than usual
Fig. 74.

Fig. 75. (Model No. 32.)

Fig. 76. (Model No. 33.)
Z.... M...., Oxycephalic.
Photograph of model actual size.
See text for description.

Fig. 77.

Fig. 78. (Model No. 34).
"and the teeth are irregularly placed and
"crowded together. The palate is very high,
"the palate ascends 19 mm. from the anterior
"alveolar margin. Its lateral walls are almost
"parallel and are 15 mm. apart between the first
"premolars. The left lateral incisor is also
"displaced lingually and is overlapped by the
"central incisor."

On comparing D. M. Greig's 4 cases with the
2 described by the author, a striking feature
presents itself namely, a high constricted
palate with irregularly placed teeth.

With the exception of the macerated skull
example, which was not investigated before death,
all the cases were mouth breathers. This, in
itself, would be sufficient to cause a high,
constricted arch, with concomitant irregularly-
placed teeth. The writer, however, is of the
opinion that the synostosis of the bones of the
cranium and face, preventing the normal develop-
ment of the palate, will cause this peculiarity;
and that they are the primary cause; the con-
dition being aggravated by the mouth breathing.

GROUP 6: SYPHILITIC AMENTS. (TABLES VII

Fifty three of the 585 mental deficient ex-
amined by the author have been classified as
belonging to the secondary amentia group of
syphilitic aments.

The following clinical signs were observed:
1. Typical Hutchinson's incisors.
2. Typical Moon's molars.
3. Enlarged frontal bosses.
4. Inequality of pupils, and diseased eyes.
5. Rhardges at the angles of the mouth.
6. Abnormalities of the palate.

1. **TYPICAL INCISORS:** The typical "Hutchinson's" incisor tooth is dwarfed, is broader at the gingival margin than at the biting edge, and is frequently notched. The notching of the incisal edge of the tooth, however is, as a rule, broken down after use; so that it can not generally be instanced as a diagnostic sign. This tooth may also be pitted; having, in a measure, the appearance of an hypoplasia. The pitting of the enamel renders Hutchinson's teeth prone to decay, especially in neglected mouths.

A proportion of the incisors in some normally intelligent persons — as also in syphilitic aments — are, to a certain extent similar in appearance to Hutchinson's incisors; yet no specific disease in these cases can be traced.

29

Black, after delineating many cases of pseudo-Hutchinson's teeth states: "There is no "special form of disease that is especially "blameable for this affliction but that any form "of disease that seriously interferes with "nutrition is liable to bring about this result, "i.e., that it is not a particular form of "disease, but that it is the condition of "malnutrition that is the cause, no matter what
"the disease which has induced that condition."

The writer has seen cases of typical Hutchinson's teeth which were certainly in no way connected with a syphilitic taint of any kind.

Typical Hutchinson's incisors were found to be present in 13 cases of the 53 in the group of syphilitic aments examined.

2: **TYPICAL MOLARS:** The typical molar, described by Moon in the year 1878, was present in 21 members of the group of syphilitic aments. The appearance of this molar tooth is as distinctive as Hutchinson's incisor. It is dome shaped: the dome, as a rule, commencing at the gingival margin. The "cusp" portion of the tooth is clearly marked from the body of the tooth, appearing to be constricted as if on a "plateau." The cusps are small, and appear to be immature; giving the tooth a "mulberry" appearance.

The writer is of the opinion that these typical six year molars are more diagnostic of the condition of the patient than the peg shaped incisors; the reason being that these molar teeth are the first permanent teeth laid down; their cusps are actually calcified at birth. This malformation, or change in the shape of the teeth indicates that a definite, pre-natal change has taken place.

Cavallaro has definitely proved that "the spirochaeta pallida is abundantly present
"in the dental follicles of the developing "syphilitic teeth"; and that "the dental 
"follicles of developing syphilitic teeth 
"have constrictions corresponding to the 
"alterations in the form of the tooth."

3: **ENLARGED FRONTAL BOSSES**: Enlarged frontal eminences were found present in 19 cases.

4: **INEQUALITY OF PUPILS, and DISEASED EYES**: On examination, 21 patients were found to be obviously suffering from abnormal eye conditions. Two were totally blind; other abnormal characteristics encountered were cataract, double pupil and inequality of pupils.

5: **RHAGADES**: Rhagades at the angles of the mouth were present in 32 cases. This affection ranged in degree from fine, hair-like lines to moist, open lineal sores.

6: **ABNORMALITIES OF THE PALATE**: Palatal abnormalities were met with in 14 cases; the normal palates numbering 39 in the total of 53 patients examined.

The habit of finger sucking accounted for the palatal abnormalities in 9 cases of the 14 patients who had abnormal maxillae.

The remaining 5 cases of abnormal palates included one patient of the oxycephalic type, whose palate was very much distorted from the
Hutchinson's teeth also arrested hydrocephalus head measurement 21\(\frac{1}{2}\)"

\(\text{Q..... F..... :}\\ \text{Hutchinson's teeth.}\\ \text{A..... C..... :}\\ \text{No oral signs.}\\ \)
Fig. 85.


Typical Hutchinson's teeth. Completely paralysed; has been bottle- and feeding cup- fed since birth.

Fig. 86. (Model No. 36).
Fig. 87.
J./. F..., Syphilitic amentia.
Hutchinson's anterior teeth;
Domed molars;
Thumb sucker.
Photograph actual size of model

Fig. 88. (Model No. 37).
Fig. 89.

Fig. 90. (Model No. 38).

Fig. 91. (Model 38).

0. . . .
G. . . .
Syphilitic amelia. Moon's domed molars; Hutchinson's anterior teeth.

Photographs actual size or models.
Faculty of Dentistry

# 9395 Box 27/59

Book: A THESIS ON
THE MOUTH OF
MENTAL DEFICIENTS

©

2 Drawings removed for wide scanning
<table>
<thead>
<tr>
<th>INITIALS</th>
<th>INGREDIENTS</th>
<th>MOLARS</th>
<th>EYES</th>
<th>PARAONTAL BOSSES</th>
<th>RHAGADES</th>
<th>PALATE</th>
<th>REMARKS</th>
</tr>
</thead>
<tbody>
<tr>
<td>J.H.</td>
<td>Centrals - 6 yr. domed molars not pitted or ridged; temporary ones normal.</td>
<td>Normal</td>
<td>Very prominent</td>
<td>Absent</td>
<td>Normal</td>
<td>Arrested hydrocephaly. See Fig. 81.</td>
<td></td>
</tr>
<tr>
<td>A.C.</td>
<td>do.</td>
<td>do.</td>
<td>do.</td>
<td>Normal</td>
<td>Perfect.</td>
<td>See Fig. 79.</td>
<td></td>
</tr>
<tr>
<td>O.F.</td>
<td>Peg shaped; no notches</td>
<td>Domed.</td>
<td>do.</td>
<td>Prominent.</td>
<td>Present</td>
<td>Abnormal.</td>
<td>A thumb sucker. See Fig. 80.</td>
</tr>
<tr>
<td>J.P.</td>
<td>do.</td>
<td>do.</td>
<td>Cataract</td>
<td>do.</td>
<td>Present</td>
<td>do.</td>
<td>See Figs. 87 &amp; 88. (Model No. 37.)</td>
</tr>
<tr>
<td>A.B.</td>
<td>do.</td>
<td>do.</td>
<td>Normal</td>
<td>Normal</td>
<td>Absent</td>
<td>Normal</td>
<td>Nil.</td>
</tr>
<tr>
<td>D.B.</td>
<td>Hutchinson's</td>
<td>Domed</td>
<td>do.</td>
<td>Normal</td>
<td>do.</td>
<td>Abnormal. See Figs. 85 &amp; 86. (Model No. 56.)</td>
<td></td>
</tr>
<tr>
<td>H.B.</td>
<td>Normal</td>
<td>Normal</td>
<td>Double Pupils</td>
<td>do.</td>
<td>Absent</td>
<td>Normal</td>
<td>Nil.</td>
</tr>
<tr>
<td>M.B.</td>
<td>do.</td>
<td>do.</td>
<td>Totally blind</td>
<td>Prominent</td>
<td>Present</td>
<td>Abnormal</td>
<td>Hypertrophied tonsils.</td>
</tr>
</tbody>
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### TABLE VII.

**ANALYSIS OF 53 CASES OF SYMPTOMATIC AMNIA.**

<table>
<thead>
<tr>
<th>INITIALS</th>
<th>INCISORS</th>
<th>MOLARS</th>
<th>EYES</th>
<th>PARTIAL BONES</th>
<th>RHAGADES</th>
<th>PALATES</th>
<th>REMARKS</th>
</tr>
</thead>
<tbody>
<tr>
<td>M.S.</td>
<td>Abnormal.</td>
<td>2 Gaps in each.</td>
<td>Normal.</td>
<td>do.</td>
<td>do.</td>
<td>Present.</td>
<td>Abnormal.</td>
</tr>
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</table>
**TABLE VIII.**

**SUMMARY OF THE RESULTS OBTAINED BY THE EXAMINATION OF 53 CASES OF SYPHILITIC AMENIA.**

<table>
<thead>
<tr>
<th>PATIENTS</th>
<th>POSITIVE WASSERMANN</th>
<th>TYPICAL HUTCHINSON'S INCISORS</th>
<th>TYPICAL MOON'S DOMED MOLARS</th>
<th>PROMINENT FRONTAL BOSSES</th>
<th>PUPILS UNEQUAL OR EYES DISEASED</th>
<th>RHAGADES</th>
<th>PALATES</th>
</tr>
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<tbody>
<tr>
<td></td>
<td>53</td>
<td>52</td>
<td>13</td>
<td>21</td>
<td>19</td>
<td>21</td>
<td>32</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>39</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>9</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>5</td>
</tr>
</tbody>
</table>
normal. Another patient, whose palate was bilaterally hypertrophied, from the area of the first permanent molar to the maxillary tuberosity, was also one of the five.

The incidence of syphilis — as indicated by the Wassermann reaction being positive — is estimated at 9.4 per cent. of the 585 patients examined.

GROUP 7: MENTAL DEFICIENTS WITH CLEFT PALATES. The incidence of cleft palate is treated in the next chapter (VIII.)

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<tr>
<th>No.</th>
<th>Author</th>
<th>Title and Details</th>
</tr>
</thead>
<tbody>
<tr>
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<tr>
<td>19.</td>
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<td>ibid., p.140.</td>
</tr>
<tr>
<td>21.</td>
<td>IDEM</td>
<td>ibid., p.141.</td>
</tr>
<tr>
<td>22.</td>
<td>IDEM</td>
<td>ibid., p.142.</td>
</tr>
<tr>
<td>25.</td>
<td>MOIR, GORDON</td>
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<tr>
<td>27.</td>
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</tr>
<tr>
<td>29.</td>
<td>BLACK, G.</td>
<td>&quot;Dental Cosmos&quot;, Nov.1908, p.1161.</td>
</tr>
<tr>
<td>30.</td>
<td>CAVALARRO, J.</td>
<td></td>
</tr>
</tbody>
</table>
CHAPTER VII
CLEFT PALATE IN MENTAL DEFICIENTS

Cleft palate - as is the case with a high constricted palate - can not be taken as an indication of mental deficiency.

This condition is not frequently met with in mentally deficient patients; the proportion of cases is the same as in normally intelligent persons.

Only 5 cases, representing 0.8 per cent., were found amongst 585 patients examined.

1 Langdon Down found the condition present in 0.5 per cent., and Ireland in approximately 1 per cent. of the mental deficients examined by them, whilst Peterson found only 2 or 3 cases amongst 450 patients whom he examined.

4 Brophy determines the incidence of cleft palate amongst the normal adult white population of the United States of America as 0.38 per cent.

The Wassermann reaction showed negative results in each of the author's 5 cleft palate cases.

Two cases are illustrated in the present work:

1. That of O.W. represents a bilateral cleft palate with a double cleft lip. (Figs. 92 & 93.) (Model No. 39.)
2. That of O.M. represents a case of simple lateral cleft palate, with cleft lip. (Figs. 94 & 95), (Model No. 49.)

There were no cases of cleft in the lower lip.

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Fig. 92.

0..... W..... Cleft Palate.
Bilateral cleft of double hare lip.
Photograph of model actual size.

Fig. 93. Model No: 39).
Fig. 94.
0..... X.....
Lateral cleft with hare lip.

Photograph of model actual size.

Fig. 95. (Model No. 40).
RESULTS OBTAINED FROM AN INVESTIGATION OF HABITS TENDING TO MALFORM THE DENTAL ARCHES OF MENTAL DEFICIENTS. (TABLE IX. p.127.)

An investigation was undertaken by the author of any habits which might tend to malform the palates of a series of 585 patients.

The most common malforming habits conducive to abnormalities were found to be as follows:

1. Mouth breathing.
2. Lip sucking or biting.
3. Finger, Thumb and Wrist sucking and biting.
4. Tongue sucking.
5. Postural.

1. **MOUTH BREATTHING**: It was found that, of the number of patients examined, 33.0 per cent. did not breathe through the nose during sleep.

2. **LIP SUCKING or BITING**: Less than 1 per cent. (.51) practised this habit.

3. **FINGER, THUMB or WRIST SUCKING or BITING**: 11.79 per cent. practised one or other of these habits.

4. **TONGUE SUCKING**: This habit, which appears to be peculiar to Mongols, was indulged in by 4.1 per cent. of the patients examined.

5. **POSTURAL HABITS**: It was found
This patient has the habit of biting her wrist. When the opportunity occurs, she chews grit and grass. The anterior teeth protrude, while the posterior teeth are very much worn down, due to the grit chewing. The models show the typical curve of Spee, rarely found in modern mouths. The muscles of mastication have developed the arches to the normal, counteracting the malformation in the palate, which would be the result of the wrist biting.
that, during sleeping periods, the patients constantly occupied certain positions which might tend to malform the palate and dental arches. Certain otherwise inexplicable deformities may be explained in this manner, and very definite abnormalities may be caused; especially when the face is pillowed upon a portion of the upper extremity. The percentage of patients examined evidencing this habit was 8.89 per cent.

It has been shown that, of the total number of mental deficient examined, 24.8 per cent. had abnormal palates; whilst 58.29 per cent. had habits which might tend to cause abnormalities. (See Table IX.) This fact is quite comprehensible by reason of close association with this type of patient.

Many patients have habits, which they practise throughout their waking hours, tending to counteract any malforming habits practised during sleep.

In demonstration of this fact, the case of a patient addicted to wrist biting, for example may be submitted. The results of this habit may be nullified if the patient should chew grit, or masticate vigorously. Figures Nos. 96 & 97, Model No. 41, illustrate a case in point. Here, the palate was very well developed, and the cusps of the teeth well worn, showing marks of constant hard work. The muscles of mastication had developed the dental arches to such an extent that, although there was a considerable overbite on the part of the incisors, the palate was normal and the approximating occlusal surfaces of the teeth
showed the typical Curve of Spee.

Many other examples might be adduced showing that an abnormal habit may be counteracted by some other habit, either normal or abnormal. Thus, although the proportion of patients with abnormal habits is much higher than the proportion of patients having normal palates as evidenced by the clinical examination, these results are not contradictory.
### Table IX

**Analysis of Results of the Examination of 585 Mental Deficients with Reference to the Habits Tending to Malform Palates and Dental Arches.**

<table>
<thead>
<tr>
<th>Habit</th>
<th>Number of Patients</th>
<th>Percentage to Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Mouth Breathing (whilst asleep)</td>
<td>193</td>
<td>33.00%</td>
</tr>
<tr>
<td>2. Lip sucking or biting</td>
<td>3</td>
<td>.51</td>
</tr>
<tr>
<td>3. Finger, thumb &amp; wrist sucking or biting</td>
<td>69</td>
<td>11.79</td>
</tr>
<tr>
<td>4. Tongue sucking</td>
<td>24</td>
<td>4.10</td>
</tr>
<tr>
<td>5. Sleeping in mal-forming postures</td>
<td>52</td>
<td>8.89</td>
</tr>
<tr>
<td>(N.I.)</td>
<td>341</td>
<td>58.29</td>
</tr>
<tr>
<td>Total</td>
<td>585</td>
<td>100.00%</td>
</tr>
</tbody>
</table>

58.29 per cent. of the patients practised habits tending to alter the shape of the palate and/or the dental arches.
A DESCRIPTION OF HABITS TENDING TO MALFORM THE DENTAL ARCHES.

During infancy and early childhood, whilst the young child is growing at a very fast rate, the osseous structures are hardening so that they may function properly in later life. It is at this critical period that malforming habits may commence, and result in lasting damage if not corrected.

There are many habits which, if acquired, cause malformations. The following are the most common:

1: Mouth breathing.
2: Negative lip pressure.
3: Thumb and finger sucking.
4: Lip sucking and lip biting.
5: Tongue sucking.
6: Wrist biting.
7: Faulty mastication.
8: Excessive salivation, or faulty control of saliva.
9: Sleeping in incorrect postures.

1: MOUTH BREATHING: Mouth breathing may commence as a habit following a cold, and may be continued unconsciously by the person concerned. The presence of adenoids or hypertrophied tonsils, or both predisposes to mouth breathing, by causing constriction of the nasopharynx. A deflected septum is also a likely cause.
Models, actual size, of a typical mouth breather and thumb sucker, showing constricted and high arch with overlapping incisors.

Fig. 98. (Model No. 42.)

Fig. 99. (Model No. 42.)
Typical model of the mouth of a mouth breather and thumb sucker. The upper incisors are protruded and do not bite normally with the lower incisors. The palate is high and constricted.
On inspiration with the mouth open, as in mouth breathing, the tongue lies passive in the floor of the mouth, with its tip resting against, but exerting no pressure on, the lingual surfaces of the mandibular incisors. The air is drawn directly into the mouth; it is not filtered nor warmed, as is the case when inhaled through the nose. It does not flow over the floor of the nose, nor does it exert any pressure which would tend to expand the palate. The mandible is kept in position mainly by atmospheric pressure; and a mouth breather has a "pinched" appearance about the chin, owing to loss of muscle tone and development. On account of the loss of air pressure, the muscles depressing the mandible retard its normal development, since their weight continually exerts a force on its anterior portion.

**INSPIRATION WITH THE MOUTH CLOSED, OR NORMAL BREATHING THROUGH THE NOSE:** During normal breathing, several pressures are created; all of which tend to expand the dental arches. This gives them a well rounded appearance, with teeth lying in their true anatomical positions.

If one take a deep breath with the mouth closed, it will be noticed that the tongue does not lie passively in the floor of the mouth, as it does in mouth breathing. It exerts a constant outward pressure on the teeth and alveolar arches. The tongue exerts pressure unconsciously
Fig. 7. Diagrammatic section of "gothic" palate (I) compared with normal palate (II). The articulation of the lower with the upper tooth by means of lateral inclined planes is also illustrated.

Fig. 101.
(See p. 133.)
against the whole of the hard palate, and tends to mould it into a rounded, domed form.

The air, passing through the nose - where it is filtered and warmed - passes into the naso-pharynx, thence into the lungs. As it passes the posterior margin of the soft palate, the tongue causes a back pressure; which also tends to widen the tongue posteriorly, and to expand the palate generally.

The continuous normal use of the nose always widens the nares; allowing, during exertion, a greater passage for the air, and making the mucous surfaces less prone to infection. This widening of the air passages, creating a wider nose, makes the floor of the nose wider; with the result that the palate becomes more domed and flattened.

With the widening of the nose and palate, more space is gained for the antra, which become normal, healthy and less prone to infection.

Pickerill gives an illustration, reproduced, (Fig. 101) which shows that there is no difference in actual measurement from the lingual gingival line "A" to the mid-line raphe "B", in the bicuspid area, between the gothic or V-shaped palate, and the well-rounded normal palate. This proves that the V-shaped palate does not originate from lack of tissue, but more from lack of expansion in the maxilla.
McKeag has shown that "the commonest type of deformity resulting from mouth breathing shows a narrow, high upper arch, protruding upper incisors and a receding chin, and is often followed by serious periodontal disease of the lower incisors, and later of the upper ones also. The normal negative pressure in the mouth being lost, the palate develops in an upward instead of a horizontal direction and consequently narrowing or lack of development of the nasal passages occur."

A confirmed mouth breather is rarely found without enlarged tonsils and adenoids; but cases of enlarged tonsils and adenoids are found in which there is no resultant mouth breathing.

Where mouth breathing is associated with enlarged tonsils and adenoids, the appearance of the patient is characteristic. There is a "pinched" aspect in the nose and chin; many a child has a stupid look, and is backward physically as well as mentally; until the physique is built up again after surgical treatment for the naso-pharyngeal growths.

2. NEGATIVE LIP PRESSURE: The lips are the bulwarks against which the teeth are forced in normal breathing. During mouth breathing, the lips do not exert any pressure on the teeth. The lips are drawn away slightly as air flows around the teeth, drying the saliva and depositing dust in the oral cavity. The mouth being
J. W.,

The model shows greater abnormality in the position of the upper left lateral tooth than is shown in the photograph. This patient has the habit of placing his thumbnail under the tooth and plicking it forward.

Fig. 102.

Fig. 103. (Model No. 44).
Illustrating marked overlap of the maxillary anterior teeth due to the habit of finger biting.
R.... C.... This patient has the habit of continually biting his bent forefinger. The photograph, (actual size), of the models shows the maxillary incisors protruding, and a retruded lower jaw.

Fig. 107. (Model No. 46).

Fig. 106.
The constant sucking of one finger has protruded the incisor teeth and rotated the right central.

Fig. 108.

Fig. 109. (Model No. 47).
open, the upper lip loses its tone through loss of muscular use. The upper lip, in course of time, thins out and curls upward, with consequent foreshortening; any pressure which it might have previously exerted being now removed.

As the maxillary arch narrows, the teeth protrude as a matter of course; thus we find that the lower lip thickens, and tends to force the teeth further away from their true positions. Whilst this is going on, the thickened lower lip exerts a greater force than usually on the lower teeth, forcing them backwards; and, as there is no compensating pressure within the mouth from the passive tongue, we find that the lower arch becomes considerably flattened in contradistinction to the upper arch; becoming narrow, and higher than normal.

3. **THUMB AND FINGER SUCKING:** The damaging results of this habit may be seen very often in the victim's fingers or thumbs. These members are often thinned out and underdeveloped; a condition that is patent to the least discerning eye. It will be readily realised that much more damage is done to the soft yielding palate and surrounding tissues.

The thumb is normally flexible and hard; if constantly sucked, it tends to pull forward the premaxillary bones, altering the normal curvature in the region of the incisor teeth; the incisor teeth protrude, causing a lack of development in the superior lip.
Fig. 110. (Model No. 48).

Fig. 111. (Model No. 49). Photographs of casts, actual size, showing abnormalities caused by thumb sucking.
The thumb is often bent whilst it is being sucked by the infant. This brings the ball of the thumb in close contact with the palate, which is an additional cause originating a high, narrow arch, instead of a broad, flattened dome.

When the habit of sucking several fingers is indulged in, the constant pressure creates a wider type of protrusion of the incisor teeth, than is apparent in connection with the habit of thumb sucking. This is due to the greater area of tissue utilised for pressure and sucking.

This distorted palate may be illustrated by means of casts of the mouth of a child; however, the child's lowered vitality, susceptibility to colds, oral infections and ultimately its patently disfigured mouth themselves prove the damage that has been done. The constant muscle pull and suction in the one direction cause a high, constricted palate, with all its resulting evils. The palate is prevented from developing normally, with the result that the anterior teeth become irregular, and forced out of place; and it is possible, by studying the shape of the mouth and irregularly placed teeth, to decide definitely which finger or thumb is being sucked.

The first irregularity noticed is the pursing up of the tissue in the region just behind the anterior palatine canal. Then the arch narrows; and finally the anterior teeth become irregular. A central or lateral tooth may often be twisted out of the normal and depressed; so that its
Fig. 112. (Model No. 50).

This model shows the anterior 4 teeth protruding in practically a horizontal manner. This abnormality is due to the habit of wrist biting.

Photograph actual size of model. (since deceased).

Fig. 113.

Showing sores on lower lip due to the habit of lip biting. No abnormality is yet noticeable in his dental arches.
Illustrating an abnormal profile. Protruding upper anterior teeth due to the habit of lower lip biting. Normal breathing.
biting edge is often three sixteenths of an inch above the level of its neighbours.

The constant pressure often forces back the anterior lower teeth, making a very flat arch, so that there is a considerable space between the opposing incisors. They are then unable to carry out properly their function of incising food, this function eventually being lost; and as growth depends upon efficient functioning, lack of growth results in the anterior portion of the jaws. When the permanent incisors erupt, there is not sufficient bone development for their normal placement; they then become irregularly spaced, one or more of the teeth sometimes being forced to occupy such a position that they may never erupt.

4. LIP SUCKING and LIP BITING: Often, through the loss of height between the opposing jaws through the incorrect occlusion of the six year molars—brought about by many causes, chiefly the early loss of the deciduous molars—a large overbite may result. In this condition the lower lip is often irritated by the biting edges of the upper incisors; with the result that the habit of biting or sucking the lip may eventuate.

Very little sucking or biting is required in order to render the lower lip irritable; the lip eventually thickens, often to double its normal size. The thickened lip tends to force backward the lower incisors, and to protrude the upper incisors. Thus the vicious cycle
is again set in motion; the upper lip shortens, loses tone and exerts no pressure on the upper incisors.

McKeag has shown that mouth breathing, thumb sucking, lip biting and, to a lesser degree, tongue biting, are responsible for the majority of cases of preventable dental deformities.

These malforming habits become quite mechanical in action, and show, as a result, characteristic deformities. The results of the action set up by continued mouth breathing have already been indicated. "Thumb sucking produces a splaying forward of the upper front teeth, and gives the unfortunate child a vacant appearance, which is a perpetual and dreadful handicap in life. "Lip biting is very common, and may be associated with tongue biting; the latter by itself usually causes a gap between the upper and lower front teeth, but the child may habitually keep the tongue between the back teeth of the upper and lower jaws on one or other side, with a resultant displacement of the teeth on that side."

5. ** Tongue Sucking:** Although tongue sucking is not very prevalent amongst normal children, it is a common habit amongst the mentally deficient class, especially the Mongol type. Thomson noticed this peculiar habit, and the author has observed it often in the Mongols under his dental care. Why this type of patient has this habit, he has been unable to ascertain;
unless it be that the Mongols are wonderful mimics.

The habit consists of sucking the tongue whilst turning its flexible anterior portion over in the mouth, and forcing the inferior surface against the upper incisors. This action tends to flatten the palate.

6. **WRIST BITING**: This habit is also peculiar to mental deficient, owing probably to the lack of correction in institutions, and the lack of co-operation on the part of the child. A model illustrating this condition is submitted (model No. 16); a photograph of the case in point was taken for this work. (L.N., figs. 96 & 97.)

As deformity was discovered in the fingers and thumbs of patients addicted to the habits of finger and thumb sucking, so it was observed that the wrist, in the habit of wrist biting, was thinned and scarred from the constant irritation.

7. **FAULTY MASTICATION**: Should the teeth on one side of the mouth be persistently evaded in mastication, owing to soreness, lack of development or loss of the teeth, it is found that diminished function means maldevelopment.

In the area where mastication is evaded, there is less development than elsewhere. It is possible for one half of the maxilla to be well developed, and the other half to be poorly
developed. Bone development is absolutely depend-
ent upon muscle pull and the normal stress trans-
mitted. The well developed jaws of the uncivilised
natives, who live on natural foods, with their
normally domed, symmetrically proportioned arches,
should be compared with the jaws of the highly
civilised races of to-day. The results of the com-
parison would show the difference to be appalling.

8. **EXCESSIVE SALIVATION or FAULTY CONTROL
OF SALIVA:** This habit is mostly confined to
mentally deficient persons. The thickened,
sore lips associated with the constant "drooling"
of saliva play their part in malformed the
dental arches. The lips being sodden and
thickened, and exerting no restraining pressure on
the teeth-(as normally they should, conforming
them into a broad arch)- allow the teeth to drift;
so that the resultant arches are shallow, with the
teeth in both jaws protruding and spaced.

9. **SLEEPING IN INCORRECT POSTURES:** It has
been known that thumb sucking and mouth breathing
are common causes of deformed dental arches.
Only recently, however, has the habit of sleeping
in incorrect postures been known to contribute
some of the hitherto inexplicable deformities
in the palates of mental deficient.

Much research work has been carried out by
Harvey Stallard, H. A. Pullen, M.B. Markus, M.B.
Jordon, and A.W. Crosby on the subject of incorrect
sleeping postures.

Stallard maintains that "20 per cent."
"of the children appearing at the pre-school-
age clinics have malocclusion that may be
attributed in part to such habits. Of the
children having malocclusion before the age
of 6 years 33 per cent., I have found, have
malocclusions resulting from pillowing upon a
part of the upper extremity. The maxilla is
more often affected by the unilateral contrac-
tion, the right side suffering more frequently
and more intensely than the left."

Stallard also refers to many of the magnificent-
ly shaped and developed skulls found amongst
children who suffer from malocclusion, due to
incorrect pillow habits. This, he states, is due
to the habit of never sleeping on the back so that
the cranium is never compressed.

Many of the cases presenting bilateral
saddle-shaped arches are directly due to the
habit of pressing the cheek against the shoulder
whilst asleep. — "The various saddle-shaped
arches correspond in their configuration to the
shape of the body, around which they have been
partly formed."

A. H. Parmelee has shown that cases of
variation from the normal occur in the heads of
infants, owing to the intrauterine posture.
The deformity is probably due to the extreme
flexion of the head, which has become partially
rotated; so that the shoulder, in this position,
is constantly pressed against the head in the
region of the jaw.
Striking cases of temporary deformity are often seen, in which the face has an asymmetrical appearance. There is a noticeable depression under the angle of the jaw, and the jaws on one side may not approximate. This, however, does not interfere with the natural nursing of the child; so that, in the course of approximately 10 weeks, the jaws are normal.

The marked asymmetry, which may become normal, is due to the disappearance of the temporary facial paralysis; together with the exercising of the jaws and surrounding parts in the natural feeding of the child. The increased blood supply to the parts concerned, and the energy used in obtaining its food supply, cause the child to develop naturally.

Both Pullen and Markus have confirmed the statements of Stallard, that abnormalities in the shape of the dental arches may be brought about by incorrect postural habits.

Crosby sums up his findings in the following manner:

"1. Habits may be the primary cause of malocclusions.
"2. Habits very often are accessory factors in producing malocclusions.
"3. Claim is not made that postural or other habits are the etiologic factors behind all malocclusions but there are a sufficient variety of cases showing harmful postures
"to realise that they are an important part in diagnosis."

Jordon also confirms the work of Stallard; and in explaining the manner in which the habits commence states: "It has been known for many years, that if a child lies continually on his back, he will flatten the back of his head; or, if he is not turned continually in infancy, the cranium will be unevenly contoured. Physicians always caution young mothers to turn their babies frequently. However, to prevent children from having colic, they are often put on their stomachs and taught to lie more or less continually on their faces. Sometimes they "pillow first on one side, then on the other."

In normal, healthy sleep, the posture changes are frequent. Johnson has shown that the position is changed as often as every half hour throughout the period of sleep. This change of position is an involuntary one, and is brought about by natural causes.

Should the habit of sleeping in one position be acquired when a child is very young, it is found that the head may be flattened in the area that is constantly pressed against the pillow. The sleeping postures of 585 mentally deficient patients under the care of the author were investigated by him; and 8.8 per cent. of the patients were found to have postures assisting to explain their malformed dental arches.
E... D... This abnormal sleeping position, in which the hands are clasped under the chin, has caused the lower jaw to be retracted, and the upper right central to be forced forward.
J. W... Faulty sleeping position, combined with mouth breathing, has caused a retruded lower jaw resulting in malocclusion.
Abnormal sleeping position has forced the posterior maxillary teeth in a lingual direction. Although the patient has a positive Wasserman, I believe that the notching of the anterior maxillary teeth is due to his incorrect bite. Most of the stress of mastication and occlusion being on his anterior teeth.
L.... B.... Abnormal maxillary arch due to bad sleeping position and mouth breathing.

The posterior teeth are occluding lingually to their normal position.

Fig. 123.

Fig. 124. (Model No. 55).
The most common sleeping postures tending to malform the dental arches are as follows:

1: That in which the patient sleeps partly on the side, with the hands resting one within the other; the palm of one hand cupping the chin, with the fingers of the other hand pressing upon the cheek; forcing the teeth lingually, and causing a "cross-bite", a form of malocclusion. (E.D., Figs. 117 & 118, Model No. 17.) In the subject of this photograph, the upper left central is being forced forward by the pressure of the thumb.

2: That in which pressure is brought to bear on the angle of the mandible, so that the mouth is forced partly open. Thus one nostril is flattened, lessening the area of inhalation; and mouth breathing is the result. (J.W., Figs. 119 & 120, Model No. 18.) In the case illustrated in this photograph, pressure is applied to the chin by the hand, so that the mandible is forced backward, causing a large overbite.

3: That in which the patient sleeps with the side of the face resting on the dorsa of the hands in such a manner that the mandible is forced forward, thus causing malocclusion. (B.C., Figs. 121 & 122, Model No. 19.)

4: That in which the hands are placed under the cheek, giving rise to constant pressure by the face, thus causing "cross-bite". This type of malocclusion, in which the maxillary posterior teeth occlude lingually to their normal positions,
C.... P.... Abnormal sleeping position, where the palm of the hand, constantly pressing in a forward direction, causes a slight prognathism. The pressure of the fingers, resting on her arm, has made the posterior teeth of the maxilla to bite lingually to the lower teeth.
Fig. 127.

6... L... V. Faulty sleeping position, with the chin resting in the hollow made by elbow joint. The lower jaw is retruded. Photograph or model is actual size.

Fig. 128. (Model No. 57).
A saddle shaped arch due to incorrect sleeping position; the patient alternately sleeping on either side, with the thumb of hand pressing lingually to lower teeth.
This paralysed patient is never happy unless her right wrist is placed in the position shown... The pressure transmitted to the lower jaw, together with the habit of mouth breathing, has caused the lower jaw to be not properly developed, with the result that it is retruded from its normal position.
naturally decreases the palatal area. (L.B., Figs. 123 & 124, Model No. 20.)

5: That in which the hands are in such a position that the constant pressure prevents the normal, anatomical occlusion of the teeth, and also prevents the proper development of the maxilla. This incorrect posture results in lasting damage to the dental arches. A case in point is shown in Figs. 125 & 126, (C.P.) (Model No. 21.)

6: That in which the patient sleeps with the chin resting in the bend of the elbow, either retruding the mandible, or preventing its proper growth. (G.L., Figs. 127 & 128, Model No. 22.)

7: That in which the hand is doubled up, so that the thumb presses heavily in the region of the bicuspids and molars, whilst the patient is sleeping on the hand. These circumstances are conducive of mouth breathing; as in this position it is very difficult to keep the mouth closed. This, in itself, is sufficient to constrict the dental arches; but the pressure, transmitted through the thumb, pressing heavily causes the bicuspids and molars of the maxilla to form a "cross-bite", instead of a normal overlap. (V.P., Figs. 129, 130 & 131, Models Nos. 23 & 24.)

It must be remembered that a child sleeps, on the average, ten hours a day; and should any one of these incorrect sleeping postures become a habit, it may result in permanent damage to the dental arches.
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CHAPTER X.

CONSTITUTIONAL CAUSATION OF
MALFORMATION OF THE DENTAL ARCHES.

General, or constitutional causes of malformed dental arches, include those causes that affect the general metabolism of the body to such an extent, that they interfere with the development of the teeth, and of their supporting structures.

The diseases of childhood that are accompanied by a high temperature have a deleterious effect upon the epithelial structures. Of these diseases scarlet fever, measles and chicken pox are remarkable for their damaging results. These ailments often prevent the enamel organ from developing normally; so that the tooth may be entirely lacking in contour; or the enamel may be so hypoplastic or pitted that caries become rampant; leading ultimately to the loss of the tooth or teeth, with the consequent development of malocclusion.

When malocclusion eventuates, there are many factors which prevent the normal development of the maxilla and the mandible, some of which are as follows:

1. Loss of certain of the teeth, allowing other teeth to drift into abnormal positions.
2. Loss of certain of the teeth, preventing the normal eruption of other teeth; so that they
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may never erupt, and impaction may result.

3. Loss of normal stress and strain transmitted through the lower jaw, so that the parts concerned do not develop to their normal extent.

4. Loss of function, causing lack of development due to loss of muscle traction.

5. Loss of teeth causing abnormal development of one half of the maxilla, should the disease select teeth on one side only of the dental arch.

The disease may cause only an hypoplasia of the enamel, malforming the contour of the teeth. This prevents normal mesio-distal contact, which allows the teeth to move slightly from their true anatomical positions. Should this be the case, the false opposing cusp relationship may so lock the jaws during mastication, that only a hinge-like masticatory action may be brought about. This action, being unnatural, will prevent the normal jaw development; giving rise to the numerous pernicious consequences due to the loss of masticatory efficiency.

Syphilis usually causes a loss of tooth contour; so that normal mesio-distal contact is lost, resulting in malocclusion and dental arch deformity.

Rickets, a disease characterised by faulty tooth and bone formation, generally brings about abnormalities in the dental arches.

The hypoplastic condition of the teeth so frequently seen in rickets predisposes them to
caries and loss of normal contact. Both conditions engender malocclusion, with a resulting abnormal condition of the maxilla and the mandible. The formation of the bone structure in the maxilla and the mandible is lacking in density; the laminae durae are ill-defined, and the trabeculae of the bone are irregularly arranged and poorly calcified.

This abnormal condition of the supporting structures of the teeth allows them to drift from their true anatomical positions, causing malocclusion. The lack of density in the maxilla and the mandible allows muscle traction to have full play: The teeth are not in their correct alignment for withstanding the force of the muscle traction; the result of the muscular force therefore, is a distortion of the bones concerned. The muscle traction tends to render more obtuse the angle of the mandible, resulting in an elongated and "straightened" lower jaw.

1 Mellanby has shown that, in rickets, the jaw bones become narrow and distorted; whilst 2 Dewey has demonstrated that there is a tendency to form thick ridges lingually to the molar areas.

In rickets, the angle of the mandible is "straightened", by reason of the action of the muscles of mastication. The result is that malocclusion develops, of a type in which the molar teeth are in occlusion, whilst the incisor teeth do not articulate; a condition known as "Mordex Apertus."
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CHAPTER XI.

FAULTY DEVELOPMENT OF THE CHILD RESULTING IN MALFORMATION OF THE DENTAL ARCHES.

Faulty development of the child may be either
1. Congenital, or
2. Acquired.

1. CONGENITAL FAULTY DEVELOPMENT.

As a result of imperfect cell union, as in primary amentia, the child may be born into the world with a physique and/or mentality, which is not capable of withstanding the influences around him. The result is that he does not develop properly, and is physically subnormal.

This child will naturally develop many physical ailments, to which a normal child may be immune. The subnormal child will show retarded development in every respect; and consequently there will be lack of normal maxillary and mandibular growth.

Should the cell union be perfect, and the mother undernourished and overworked, it would be expected that the offspring would be physically or mentally imperfect in some respect. If the environment be suitable for the proper growth of the child, the child will eventually become physically normal; if unsuitable, one would expect the physical condition to remain imperfect; and that no improvement in the general bodily
metabolism could take place.

During the development of the dental and oral tissues, disturbance in their formation may occur. Such disturbance chiefly takes one or other of the following forms:

I. Cleft Palate.

II. Disturbed Normal Functioning of the Ductless Glands.

III. Abnormal attachment of the Frenum Labii.

IV. Supernumerary Teeth.

V. Missing Teeth.

The most serious disturbance of fetal development must arise during the first three months of pregnancy, when various parts of the developing head fail to unite, or are improperly developed.

I. The most common deformity occurs in the upper jaw, and consists of a cleft palate, which may or may not be accompanied by a cleft lip. In some cases a wide opening may be established, connecting the nasal and oral cavities; or the deformity may be very slight, so that a minute opening only may result.

Cleft palate is caused by the failure of the several component parts of the palatal processes to unite. The structures adjacent to the cleft are unable to develop in a normal manner, and thus become retracted from the cleft. Cleft palate may be caused by (a), lack of development of the parts concerned or (b), mechanical interference
between the developing parts so that they do not unite.

(a). **LACK OF DEVELOPMENT**: Brophy has shown that cleft palate has occurred in three generations of a single family; and that there is an inherited susceptibility to this condition. **Bunting** has shown that malnutrition may be one of the predisposing causes. He quotes the case of lion cubs born in captivity having cleft palate. "These deformities were traced to a "deficient diet of the mothers" and when the "deficiency was corrected the cubs were born with "normal lips and palates."

Brophy and Keith maintain that there is no lack of development nor deficiency of tissue at birth. **Keith**, in a personal communication to **Brophy** states: "I agree with you that in the "majority of cases of complete cleft of the pal-"ate, there is no deficiency of tissue at birth "nor for some time after birth. I also agree that "the cleft, however wide, is not due to a "deficiency of tissue in the several elements "which form the palate, but is entirely due to "the fact that when the various embryonal parts "or elements are developed and come together in "the second month of development, the process of "union, which should occur then by a means similar "to union by first intention, is delayed and "does not take place, hence the several palatal "elements, being inco-ordinated by union, tend "to separate as growth occurs, the cleft increasing "during each month of growth. The exact cause of "the separation of the parts and the enlargement
"of the cleft is probably due to several factors, "tongue pressure, muscle traction, and also the in- "dependent process of growth in each individual "part."

(b). MECHANICAL INTERFERENCE: In many cases of cleft palate, bands of fibrous tissue have been found separating the two halves of the maxilla, preventing the normal union of the two approximating surfaces.

In the developing fetus, the mandible and tongue are developed earlier than the maxillae. In certain positions of the fetus, it is possible that pressure is brought to bear on the mandible; so that the tongue may be forced between the two halves of the developing maxillae, with the result that they may never unite.

Brophy has shown that "in young infants "who have cleft palates, I have always found that "by slightly forcing the chin upward, the pressure "of the lower jaw against the segments of the upper "jaw will spring the cleft bones farther apart." This shows that a cleft, small in early fetal life, becomes wider as age progresses, owing to the constant springing apart of the edges of the cleft brought about by mandibular action; especially is this so when the muscles of mastication are utilised.

II. DISTURBED NORMAL FUNCTION OF THE DUCTLESS GLANDS

Any disturbance, which takes place in the normal functioning of the ductless glands, interferes with the normal growth of the body.
Diseases of the pituitary bodies cause an overgrowth of the facial bones, as well as overgrowth in the hands and feet. When the disturbance takes place in adult life, the disease is termed Acromegaly; and should the overgrowth in the facial bones be very excessive, it is termed Leontiasis Ossea. Disturbed thyroid function causes a lack of development of the body, as instanced in Cretinism. Naturally, there is a lack of normal palatal development.

III. ABNORMAL ATTACHMENT OF THE PREMOLAR LABII.

This condition may be congenital, and the resulting malocclusion is very characteristic. The central incisors are spaced, resulting in an improper mesio-distal contact of the teeth, which causes incorrect occlusion, and hence a maxilla which is not normal in size and shape. The abnormal attachment of the frenum, being too low down on the gingival border of the gum, forces the central teeth apart as they erupt; and they are kept in that position by the fibrous attachment.

IV. SUPERNUMERARY TEETH.

Supernumerary teeth may cause an abnormally developed maxilla and/or mandible. They are usually found in the incisor region. If they are placed in the normal line of contour of the teeth, they increase the width of the arch, causing the maxilla or mandible to become abnormal in size and shape.

Bunting has shown that "supernumerary teeth often appear in several members of the one family, "and may be traced through several generations."
V. MISSING TEETH.

Through the absence of teeth, the normal mesiodistal contact is lost, allowing the teeth present to drift from their true positions. This often causes asymmetry in the maxillae, should there be teeth missing on one side only. Should the corresponding tooth or teeth be missing from both sides of the maxilla, disharmony in the arches is not noticed; but a generalised smallness is the result.

2. ACQUIRED FAULTY DEVELOPMENT.

One of the greatest factors in producing malformation of the maxilla and mandible is the effect brought about by

I. Incorrect Food; and
II. Incorrect Diet.

This result may ensue from the effect of incorrect food and diet on the teeth particularly, and on the constitution as a whole.

I. INCORRECT FOOD.

In order that the osseous structures of the body be properly formed, food that is correctly balanced in quality and quantity must be eaten. Food that lacks calcifying materials will not build osseous structures fit to withstand the everyday needs of life. The jaws will not be developed properly, if food be selected that does not require the proper usage of those parts; as development only arises as the result of use. Teeth that are used constantly to masticate efficiently are not so prone to caries, as teeth used to masticate over-soft foods.
In the present day, the custom is becoming prevalent of selecting only foods that are tender, and of passing over foods requiring mastication. The author is convinced that the selection of soft foodstuffs is responsible for the preponderance of artificial dentures over natural teeth in parents over the age of 35 years. On account of the artificial dentures, foods are selected that may be masticated easily. These soft foods are supplied also to the rising generation, in whom the necessity of vigorous mastication does not arise; so that there is a consequent lack of development of the jaws.

Pickerill has shown that, if the mandible is not developed normally, neither can the maxilla be developed normally.

He states: "The lower teeth articulate with the upper teeth by means of inclined planes from within outwards, and that any outward movement of the lower teeth is bound to be followed by a corresponding movement in the upper teeth, and if this does not take place neither can the upper teeth move outwards. Comparatively few muscles are attached to the upper jaw; it is not moveable, and its stimulus to growth and development must come largely from the impact of the lower teeth against the upper in the act of mastication."

Soft, starchy foods are prone to cause caries
readily, owing to the adhesion of food particles to the teeth. These food particles ferment rapidly, causing in the tooth enamel the initial lesion so essential to caries.

In incipient caries, the teeth often become sore; thus the child is prevented from masticating efficiently, the result being, in time, a lack of development on the side evaded in chewing.

If caries should attack a tooth to such an extent that it be found necessary to have the tooth removed surgically, maldevelopment of the maxilla and mandible ensues, owing to the loss of function.

II. INCORRECT DIET.

The subject of diet, in relation to the development of the child is of such transcendent importance, that the author has considered it advisable to assign to a separate chapter his observations on incorrect diet, and the part it plays in faulty child-development.

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CHAPTER XII.

INCORRECT DIET: ITS PART IN MALFORMING THE DENTAL ARCHES.

The fact may not be questioned, that incorrect diet and improper feeding play an important part in the maldevelopment of the human jaws; whether they belong to mental deficient, or to normal children.

During the last decade, research workers such as Truby King, May Mellanby, Howe, and many others have demonstrated that, although a child may be fed on a diet whose calorific value is perfect, it may fail to progress in a satisfactory manner physically. The reason for this is that the diet is lacking in certain chemical substances, which have been termed "Vitamins."

These Vitamins, for want of better terms to distinguish them, have been styled Vitamins A., B., C., and D.

**Vitamin A.** is found chiefly in butter, cod liver oil, egg yolk, spinach, carrots, peas and green leafed vegetables.

**Vitamin B.** is found in such foods as egg yolk, cereals, spinach, cabbage, onions and tomatoes. Milk contains very little vitamin B., and when added to white bread, to make the dish termed "bread and milk", has very little vitamin value.
Vitamin C. is absolutely essential to life, and is found in green vegetables and fruit; and the greatest content is found in the juices of oranges, lemons, tomatoes and cabbages.

Vitamin D. is the chief calcifying vitamin. A diet having a low vitamin D. content, creates osseous structures that become imperfectly developed. Vitamin D. is chiefly found in egg yolk, fish fats, cod liver oil, milk, cheese and animal fats, but never in lard and bacon.

Mellanby, experimenting on puppies, selected 3 from the same litter, and labelled them a., b., and c. a. and c. were the animals to be experimented upon with a special diet, whilst puppy b. was to be fed on a normal diet.

Puppy a was fed on a diet rich in vitamins, and it was found that the jaw bones and teeth were strong, healthy and perfectly developed; the laminae durae being dense and even in thickness, and the trabeculae of the bone being well defined and systematically arranged.

Puppy c was fed on a diet scientifically arranged, but lacking in fat soluble vitamins. In this case, remarkable differences in the bone structure and the development of the jaws were noticed. The jaw bone was thick, but less compact and curved; the laminae durae were ill-defined and the trabeculae of the bone were
irregularly arranged and poorly calcified. The jaws were narrow, and the teeth crowded as a matter of course.

_Puppy b_, which was the control, had a normal jaw, with teeth regularly spaced.

Following are several of the results of the Mellanby experiments:

"The character of the teeth and jaws, as regards both their general development and microscopio structure, can be greatly influenced by diet."

"The development of perfect teeth can be ensured by an adequate supply of vitamin D."

"If the diet of a puppy is changed from one poor in fat soluble vitamins to one rich in these substances, there is an immediate response shown by improved calcification. When the process is reversed the response is delayed for a comparatively long period."

"Since teeth as well as bones are largely built up of calcium and phosphorus, it is essential especially in the developing animal that the diet should contain a certain supply of these substances."

If a diet, rich in vitamin D, but fairly low in calcium and phosphorus, be assimilated, the bone structure will be good. If, however, vitamin D be absent, even a diet rich in calcium and phosphorus will not accomplish the same good result.
Howe also conducted a similar range of experiments with guinea pigs and monkeys; and his findings are similar to those of Mellonby. Two normal young monkeys were used for one experiment and their weight, size and general development were tabulated. One monkey was kept on its usual diet rich in vitamins, and it grew naturally with perfect skull and dental arch development. To quote Howe's own words, "The other monkey was placed on a diet good in all respects except that it contained no vitamin C. For 23 months this animal received only occasional small quantities of orange juice. At the end of the time it died suddenly in the cage. Now both these monkeys were on a normal diet long enough to develop the crania normally for quite a while after birth,"

The difference in the size of the skulls, facial development and dental arches after death was astonishing. The monkey fed on a deficiency diet demonstrated the fact that the skull had practically stopped growing, from the time that the deficient diet had been administered. The dental arches were narrow, and were not developed sufficiently to permit the normal eruption of the teeth. Thus the teeth were crowded, irregular and very curious.

The findings of Mellonby and Howe are confirmed in dental practice. Very often, great improvement has been observed in the general physique and dental arch development of children,
when the diet has been properly regulated, and added to by substances rich in vitamins.

Cod liver oil alone has worked wonders; but the recently improved, more palatable combinations containing vitamins A and D from activated ergosterol seem to give better results.

REFERENCES.

2. IDEM, ibid., chap. 17., pp. 303 et seq.
CHAPTER XIII.

THE UNNATURAL FEEDING OF INFANTS.

One of the fundamental causes of malformed maxillae is the unnatural feeding of infants.

1 Dreyfus, discussing bottle feeding, writes: "In analysing the cause of certain classic deformations of the dental arch, as thumb sucking, I have been able to establish the great fault to be found in the use of the present day teats."

The natural milk supplied by the mother of the infant is the ideal food; yet many mothers either do not seem to be able adequately to feed their children, or will not persist in this natural method.

2 Howe states: "There can be no question but that human milk is the ideal food for infants. It is more than food in the sense of containing the raw materials for tissue building and fuel for energy. It is a biological fluid containing glandular extracts and other unidentified substances which act in such a manner that it is called a protective fluid. Perfectly balanced in its proportions, it is adapted to all the needs of the developing infant."

Bottle feeding has to be resorted to when breast feeding is unavailable; and the use of the
bottle as an artificial substitute for the natural breast is prone to cause malformation of the jaws of the infant.

There are two main factors in bottle feeding which tend to deform the maxilla:

1. Artificial food; and
2. Bottle feeding.

1. ARTIFICIAL FOOD: Much research work has been carried out by Truby King to show that "patent" foods, which are chiefly used in artificial feeding, are not as efficient as humanised cow's milk. Babies flourish, as a rule, for a time only on patent foods. They may become fat, but have not the firm flesh of a naturally fed baby, nor of a baby fed on humanised cow's milk. They are more prone to diarrhoea or to other gastric disturbances; as they do not seem to have the resistance necessary for warding off illness, such as is apparent in the naturally fed child.

Feeding infants on patent substitutes for natural milk prevents them from developing as satisfactorily as the naturally fed babies. The bone formation is softer than it should be; so that when stress is applied by the teat of the bottle to the maxilla, the bones, which have not the hardness to resist the pressure, become malformed.
Model of patient aged seven years. Illustrates the lasting damage done to arches through the loss of all deciduous teeth, together with the superior 6-year molars. The upper arch is narrow and not developed. The profile has been spoilt, and mastication is impossible. The centrals are being forced out of position.
Fig. 156. Twins. The one on the left was breast-fed; the one on the right was bottle-fed on condensed milk. (New York Health News)

Fig. 136.

From "Nutrition of Mother and Child" - C. Ulysses Moore. (See p. 187.)
Fig. 137.

From "Operative Dentistry for Children"—
M. E. Jordan. (See p.p. 187-8.)
Moore has shown that the death rate of infants, according to tables compiled by the Metropolitan Life Insurance Company was ten times greater in bottle fed babies than in naturally fed babies. He also quotes Jacobi as having stated that 85 per cent. of the deaths during the first year of life occur in bottle fed infants.

Once teeth begin to erupt, the high carbohydrate content of patent foods causes rapid caries through fermentation. This is shown in the number of cases of well formed and perfect two year molars, which have erupted after the feeding bottle has been dispensed with, the remaining deciduous teeth being very carious. The early loss of deciduous teeth may bring about abnormality in the shape of the developing jaws, which will be fully treated in a subsequent chapter (XIV).

2. BOTTLE FEEDING: Bottle fed babies are prone to have malformed jaws. At the most critical period in a child's life, when tissues are soft and may be moulded, a hard substitute for the firm, yet yielding breast may have to be used, which causes a malformation of the maxilla.

In order that an infant may obtain milk, it must use an appreciable amount of energy. The child creates a vacuum in the buccal cavity, and the vacuum so created chiefly causes the flow of milk. In order to create this vacuum,
the lips must seal the area surrounding the
nipple, so as to prevent the ingress of air.
The lips perform a certain amount of exercise
in the act of sealing the area around the nipple,
so that they become strengthened. Thus by
the time that the teeth erupt, a definite elastic
band of tissue is formed, which prevents the
protrusion of the incisors.

The muscles of mastication are developed
normally by exercise, thus developing naturally
the mandible and the maxilla, so that there is
sufficient space for the teeth to erupt.

When bottle feeding is resorted to, no
work is performed by the child. The food is
obtained by pressing, with the tongue, the
teat of the bottle against the palate. The
action thus set up must, in time, cause a high
palate. Dreyfus has shown that "The pressure
which a child has to exercise to compress the
"teat against the palate in order to extract
"milk, may reach 500 to 600 grams (1 to 1-1/3 lbs.)
"This shows that the palate of the infant must
"oppose a resistance at each suck or rather 'loll',
"in other words, the palate of the infant bears a
"regular pressure which may amount to 500 or 600
"grams. The effort of the action of the tongue
"from front to back would be to project the
"anterior part of the maxilla giving it a 'V'
"form and to raise the median part of the palate.
"This would favour a protrusion of the incisors
"and lead to an ogival or gothic palate."
This eventually leads to a deflected nasal septum and crowded turbinate bones; leaving the nasal passage in a fit state for infection, due to the inflamed mucous membrane. The reduction in the air space in the nasal cavities engenders in the child the habit of mouth breathing, with all its attendant dangers.

The creation of the vacuum in the baby's mouth in normal nursing, compels the atmospheric pressure within the nasal cavities to cause the palate to descend, thus enlarging the palate.

The effect of lowering the base of the nasal cavities would be the lowering of the nasal septum, which would also be kept straight. This would lead to larger airways and better developed antra less prone to infection; resulting in a much healthier child.

A bottle fed baby, not having been trained to exert muscular force in obtaining its food, does not know how to masticate; and considerable trouble is experienced in teaching it how to masticate its food. The food is swallowed in lumps, in the same way as the patent food has been allowed to trickle down its throat. The dental arches are not properly developed; the teeth are crowded, and eventually become carious; since there are not any natural cleaning spaces such as Nature supplies in the normally developed mouth.

Howe states: "So great is the prevalence
of maxillary deformities with their attendant malocclusions that we seem to have accepted them as a sort of necessary evil and to have become so absorbed in remedial measures that we have neglected to study fundamental causes."

It has been shown that bottle fed babies have, on the average, deformed maxillae. This deformity may be corrected, if a special diet is commenced before the third year. The diet must be rich in vitamin A and D; it must consist of foods selected in order to necessitate mastication. Ample sunlight and fresh air, with correcting deep breathing and facial muscle exercises are also necessary.

A striking case of a deformed maxilla due to bottle feeding is illustrated by Dr. C. Ulysses Moore in his book "Nutrition of Mother and Child", here reproduced. (Fig. 157). It will be noticed that the jaws of the breast fed baby were almost 40 per cent. broader than those of the bottle fed twin sister.

Jordon reprints a photograph from "The New York Health News", reproduced on p.182 (fig.136) of twins one of whom had been breast fed, and the other bottle fed on condensed milk, rickets subsequently developing, Whilst Jordon included this illustration to demonstrate the deleterious effect of artificial feeding on the general physique, one feature of especial interest to a dental surgeon presents itself, namely, the difference in the facial contours. It will be noticed that the breast fed twin (left) has apparently well-formed
jaws, whilst the artificially (bottle) fed child has a physiognomy apparently indicating the thickened, spongy jaws generally associated with rickets.

REFERENCES


3. KING, SIR F. TRUBY, "Feeding and Care of Baby". Macmillan, 1925.


A large proportion of parents take no interest in the deciduous teeth of infants, as they consider that, as the teeth will be eventually exfoliated, the permanent dentition will suffice for the needs of the child.

Since the advent of the six year molars is a normal physiological action, usually without untoward symptoms such as feverishness or diarrhea, these teeth are often considered as being deciduous. No especial care is taken of them; so that they may become carious, and perhaps beyond remedial action.

The early loss of deciduous teeth through caries brings about a loss of masticatory power; which, in its turn, prevents the maxilla from becoming normally developed. The lack of development brings about a crowded dentition, which may cause the impaction or non-eruption of one or more of the permanent teeth. It may also allow the permanent six year molars to drift mesially, causing malocclusion, with its many attendant abnormal developments of the maxilla and the mandible.

Caries of a deciduous tooth may cause soreness, thus forcing the child to masticate on one side of the mouth only. As development is dependent
upon muscular activity, one side of the maxilla is developed at the expense of the other side. This habit may be practised even after the permanent dentition has erupted; the result being an abnormal maxillary development on one side.

The loss of the first permanent molars, very common in modern child life, has an important bearing on the profile. In crowded mouths, in order to make room for the erupting teeth, it has often been the practice to extract the first permanent teeth; with the result that there is a lack of development in the area of the molars. The face loses its normal profile, being diminished in a vertical direction, and terminating in a prominent pointed chin. The lower incisors have a tendency to be forced lingually. This assists to accentuate the prominent chin, as the lips naturally fall back with the incisors.

If the carious deciduous teeth reach the suppuration stage, much damage may be done to the child's physique. The toxins may be absorbed either directly through the blood and/or lymph stream, or by being mixed with the food. In any case, the child becomes sick and lethargic, and does not progress normally.

A photograph of a model has been taken for this work (Figs. Nos. 134 & 135) showing the loss of height between the opposing jaws, and the consequent loss of masticatory power in a child 7 years of age. This child was delicate and
undernourished. The permanent upper molars, together with all deciduous teeth had been extracted. The illustration shows that the 2 permanent upper incisors are being forced labially. There is lack of development in the whole maxillary arch, due to the loss of the teeth.

1. McKeag has shown that "Even now, there are hundreds and thousands of parents who firmly believe that the loss of the milk teeth is unimportant, because they will be followed by a second set. The proper growth of the jaws is, to a very great extent, dependent on the presence of the teeth. Where a temporary molar is lost on one side before its time, the permanent molar in its growth drifts into the space provided, and the permanent bicuspid, which should occupy the position of the deciduous molar is crowded out of line. The bone of the jaw develops less than on the side where the deciduous teeth have been retained until the permanent teeth under them are ready to erupt."

REFERENCE

PALATAL MALFORMATION OCCASIONED BY SEPTIC CONDITIONS OF THE ANTRA

It has already been shown that hypertrophied lymphoid tissue plays a considerable part in the malformation of the dental arches, bringing about (1) the constricted "gothic" type of maxilla or (2) prognathous lower jaws.

Sepsis within the antra may cause an entirely different type of malformation. It is well known that, under certain conditions, inflammation may progress to the stage of bone formation. The presence of the Staphylococcus Aureus group of micro-organisms often stimulates this growth. As this is the most common group of pyogenic micrococci found in the antra, it is quite possible that the results obtained from the examination of 120 cases of abnormal palates by Dr. A. Yates is correct.

To quote Dr. Yates' words - "In 127 cases the deformity was equal on the two sides. In the 3 unilateral cases, examination proved that there was a chronic maxillary sinusitis on the side which showed the deformity and this was confirmed by washing out a quantity of pus from the antrum.

"Of the 127 cases in which the deformity was bilateral, 6 showed deformity of the superior maxilla only and in these there was maxillary sinusitis, 121 showed deformity of all facial
"bones in which the maxilla and mandible shared."

REFERENCE.

CHAPTER XVI.

SUMMARY AND CONCLUSION.

The former portion of the Thesis — (Chapters I — VII) — has been devoted, inter alia, to the classification of Mental Deficients into groups and sub-groups based on the methods of Tredgold (Chapter II.), and to a description of the clinical characteristics of the different types, with special reference to oral conditions, and particularly the incidence of palatal abnormalities. (Chapter VI.).

A resume of a quantity of literature on the subject of Mental Deficiency was made, especial emphasis being laid upon the opinions of the various authorities concerning the theory, that the palate is one of the stigmata of mental degeneracy. (Chapter III.).

The author's findings as a result of his examination of 565 mentally deficient patients and of 280 persons of normal intelligence under his dental care were also given, together with his conclusions thereon. (Chapters IV., V., VII., and VIII.). These findings are detailed in Tables III — IX., but may with advantage be recapitulated here:

(a). The incidence of palatal abnormality, in persons of normal intelligence totalling 280, was 35. per cent.
(b). The incidence of palatal abnormality in the respective groups and sub-groups of primary and secondary amentia were as follows:

1. In the total number of mental deficient (585) - 24.8 per cent.
2. In primary aments (349) - 27.7 per cent.
3. In secondary aments (226) - 20.35 per cent.
4. In Mongols (25) - 12 per cent.
5. In Mongoloids (5) - 33.3 per cent.
6. In microcephalic aments (20) - 20 per cent.
7. In hydrocephalic aments (3) - nil.
8. In achondroplastic aments (2) - nil.
9. In oxycephalic aments (2) - 100 per cent.
10. In syphilitic aments (53) - 26 per cent.

(c). The incidence of the various types of primary and secondary amentia in the total number of mentally deficient patients was as follows:

1. Mongolism - 4.2 per cent.
2. Mongoloidism - .51 per cent.
3. Microcephaly - 3.4 per cent.
4. Hydrocephaly - .51 per cent.
5. Achondroplasia - .54 per cent.
6. Oxycephaly - .34 per cent.
7. Syphilitic amentia - 9.05 per cent.

Cleft palate was found in 5 patients, the percentage being .8 per cent.
Of the 585 mentally deficient patients examined, 58.29 per cent. practised habits tending to alter the shape of the palate and/or the dental arches. Positive Wassermann reactions were evidenced by 9.4 per cent.

In the latter portion of the thesis, (Chapters VIII. - XV.), the author dealt with the probable causes of palatal deformity, as follows:

1. Habits, especially those practised by mental deficient. These were thoroughly investigated, and the results showed that, although the percentage of patients, practising habits which could malform the palates and dental arches, was higher than the percentage of patients having abnormal palates and dental arches; yet many of the results of malformed habits were nullified by vigorous mastication and other habits practised during waking hours. (Chapters VIII. and IX.).

2. Constitutional causes; for example, diseases of childhood, such as rickets, measles, scarlet fever, et cetera. (Chapter X.).

3. Faulty development of the child, either (a) congenital, due to imperfect cell union; or disturbance in the formation of dental and oral tissues during fetal development; or disturbed normal function of the ductless glands (Chapter XI.); and (b) acquired, due to the use of incorrect food, resulting in caries caused by a deficiency of vitamin D, as indicated by the experiments of Mellanby and Howe; (Chapter XII.).
4. Unnatural feeding of infants: under this heading, the deleterious effects of bottle feeding were contrasted with the beneficial results accruing from utilising Nature's provision for the nourishment of the child. (Chapter XIII.).

5. Early loss of deciduous teeth, due to the presence of caries, through neglect; causing a loss of masticatory power and consequent maldevelopment of the maxilla. (Chapter XIV.).

6. Septic condition of the antra, as a result of which inflammation may progress to the stage of bone formation. (Chapter XV.).

If, as has been maintained by the majority of authorities on the subject, a deformed palate is one of the stigmata of degeneracy, it would have been anticipated that the bulk, if not all, of the author's mentally deficient patients would have evidenced this especial characteristic. It will be seen, however, from the preceding summary (b, l., p.195.) that barely one quarter (24.8 per cent.) of the mental deficiencies examined by the author had abnormal palates. It will also be seen from the summary (a, p.194), that the proportion of abnormal palates, (35 per cent.), in the total number of patients of normal intelligence examined, was much greater than the proportion in the mentally deficient patients. It must be stated, however, that the abnormality, when present in mental deficiencies, is generally of a more pronounced form than the abnormality when present
in persons of normal intelligence.

The author has come to the conclusion, therefore, that the shape of the palate and of the dental arches of mental deficient is not pathognomonic of mental degeneracy; in other words, there is no relation between the mental condition and palatal abnormality in mental deficiency.

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