THE AETIOLOGY, BACTERIOLOGY, PATHOLOGY, TREATMENT,

OF

PYORRHOEA ALVEOLARIS

By

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THE AETIOLOGY, BACTERIOLOGY, PATHOLOGY, TREATMENT, OF PYORRHOEA ALVEOLARIS.

The problem of dental sepsis is of importance alike to dentist and doctor, for since the work of William Hunter, the view that it is a common cause of general disease has taken a definite place in medicine. Now that the Radiogram has revealed the frequent existence of apical rarefaction due to infection in pulpless teeth, these lesions have to a great extent overshadowed the significance of pyorrhoea alveolaris as a cause of dental sepsis and by implication a cause of constitutional symptoms. Clinical evidence would suggest that all forms of dental sepsis are a potential source of danger though the fact that in many individuals gross dental sepsis may coexist with excellent health suggests that the pathology of various dental lesions is imperfectly understood.

A review of the work that has up to the present been written on the subject of pyorrhoea alveolaris shows that the present state of our knowledge is meagre and imperfect. The host of theories advanced to account for the causation of pyorrhoea and the different methods of treatment in vogue are a sufficient index of our ignorance./

ignorance. In this country many dentists are of the opinion that it is a local lesion in accordance with the views put forward with so much cogency by Mr J.G.Turner.(1) But Mr F.W.Broderick,(2) who holds equally strongly that a faulty metabolism is an essential factor in the disease, would find not a few to agree with him in a view, which in the present state of our knowledge, is somewhat speculative and lacks the simplicity of the theory of local origin. Yet this simplicity may only be apparent and even misleading. I considered it was unwise to rest content with such an aetiology without exploring thoroughly the possibility of a constitutional element. To sum up,at present it is not definitely settled whether pyorrhoea alveolaris is a local condition, or whether it is a local manifestation of some general disturbance.

To my mind the first great difficulty that we encounter in considering the subject is one of nomenclature. The dental profession as a whole seems to be uncertain what we mean exactly by pyorrhoea and the medical profession is more uncertain still. The term 'pyorrhoea' is unfortunate, meaning as it does a discharge of pus, since the majority of cases of this disease are characterised by a discharge of little or no pus. This difficulty is due directly to our very imperfect knowledge of the aetiology.

During/

During the past two years, from observance of cases at the Royal Infirmary and the Glasgow Dental Hospital, the aetiology is clear as follows:-

A. Local. Cases of marginal gingivitis caused by irritation from plaques of salivary or seruminal tartar, due to simple neglect or specific infection causing gum recession, opening up of the sub gingival space, with formation of pockets and absorption of alveolus, coexistent with destruction of periodontal membrane. With the progress of time, this condition becomes general by contiguity leading on to a destructive pyorrhoea.

B. Constitutional. Commencing primarily as a rarefaction of the alveolus with subsequent absorption of bone, not characterised ab initio by marginal gingivitis, accompanied by loosening of the teeth with little or no production of pus except in the very late stages, without plaques of tartar seruminal or salivary as the exciting cause. This constitutional type I would prefer to designate by the name of periclasia, a name suggested some years ago.

The majority of patients suffering from pyorrhoea alveolaris attending the Dental Hospital belong to the former class and it is this type of pyorrhoea which gives rise to secondary symptoms and phenomena commonly associated with dental sepsis due to absorption of toxins into the general circulation.

As regards the latter type, this is the type more often met with/

with in private practice and in Hospital. What then is the cause of this absorption of alveolus which I regard as the initial stage of the disease? Type B.

Let us consider for a moment the subject of Chronic Acidosis.

The Hydrogen ion concentration of the blood under physiological conditions is always kept constant. It is only in the terminal stages of disease that any alteration occurs and this usually precedes death. The Hydrogen ion concentration of the blood is one of the most constant things in nature and that this should be so becomes all the more surprising when one considers the number of factors which are tending to alter it. The acid base equilibrium system is a very complex one employing as it does every system in the body. In general the metabolic processes incline towards the production of acid. Acidosis does not mean necessarily an increased storage of acids or acid salts in the blood and body tissues nor an increase in the production of acid in bodily metabolism. Sellard's definition (3) of acidosis is "it is a diminution in the reserve supply of the alkaline bases in the blood and other tissues of the body, the physiochemical reaction of the blood remaining unchanged except in extreme conditions." Acidosis therefore means the impoverishment of the body in available bases following decreased capacity of the tissues to get rid/

rid of CO_2 and other acids formed in metabolism. Practically acidosis results from defective oxidation of organic acids and defective elimination of mineral acids, because of impaired Renal function, e.g. in Diabetes, nephritis, and other mixed forms. Extreme conditions of acidosis are met with clinically in diabetes, last stages of nephritis, in pregnancy, in food intoxications which are easily recognised by the physical signs but however an acidosis can be demonstrated by chemical laboratory methods to exist where there have been no acute symptoms and where it has been proved that the alkali reserve of the body has been reduced to a greater or lesser extent, termed by Sellard 'compensated acidosis'. It must therefore be evident that there must be in many cases a condition of Chronic Acidosis present without any definite signs or symptoms of the disease.

To combat a condition of acidosis the body calls on the following for neutralizing agencies:-

(a) Capacity of proteins to combine with acids and alkalis.
(b) Reserve neutralizing power/of ammonia formed in metabolism.
(c) Enormous reserve supply of bone salts, mainly Calcium.

Metabolism of Lime Salts.

Calcium is found in the body in two forms:-

(i) fixed in tissues as inorganic salts,
(ii) quantity acting as alkali reserve.

Lime salts enter the body by way of food supply but the question/

Radiogram of femora of Case 1 showing decalcification and small incomplete fracture.

question merely of ingestion and of a balanced diet is insufficient as absorption followed by utilization and fixation are necessary. Lime salts may be excreted unchanged in the faeces, absorbed into the blood stream, there utilised or not according to the metabolic requirements and excreted in the urine. Lime salts therefore form an important part of alkali reserve and the important function they perform is the gradual hardening of bones and teeth. If, however, they are required for a more vital purpose, namely to compensate an acidosis, the process of hardening of bones, teeth, etc. must stop and in fact a definite demineralization may take place with the separation of lime salts from these tissues. The metabolism of lime salts is believed to be controlled by the endocrine system. With special reference to lime salts these ductless glands may be divided into two groups:-

(1) utilizers - parathyreoid and pituitary suprarenal.

(2) secretion - gonads, ovary and testes, thyreoid.

What clinical evidence have we of this control of lime salts by endocrine apparatus?

(1) Case of osteomalacia.

A.M. Age 26.

History:- 10 years ago she felt stiffness in the points of the vertebral column, considerable pain, and was forced to take to bed: pain in legs, stiffness in both knees and ankle joints. Was in bed for one year and was then able to move about on crutches. Three months ago R. shoulder joint and wrist became painful with pain in epigastrium.

Radiogram of pelvis and vertebral column of Case 1 showing scoliosis, rostral pelvis and small fracture.

Radiogram of Humerus of Case 1 showing decalcification.

Examination marked scoliosis, pelvis compressed laterally, rostral pelvis, double genu varum and anterior curvature. Humeri have nodes on lateral aspect above deltoid insertion. Movements of both shoulder joints painful.

Radiogram shows generalised rarefaction of bones especially epiphyses, several small fractures visible in pelvis and on outer aspect of left Femur. Shoulder joints show arthritis.

Blood - slight degree of leucocytosis.

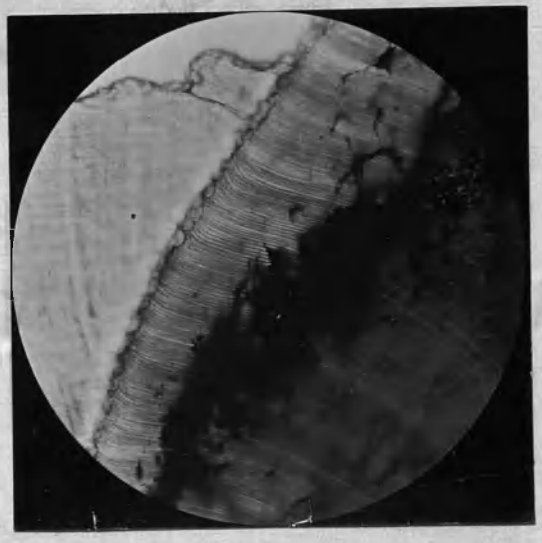
Saliva - pH = 7.2

Gums - Gums are rather pale, marked recession; no discharge of pus, no salivary or seruminal tartar.

Teeth: Well formed as regards contour. There is no Hypoplasia present. Teeth dull in colour, all teeth are present except lower incisors. Dental caries is absent save for a very small commencing approximal cavity in the upper R. central:teeth are all loose due to absorption of alveolus and she says they are gradually falling out.

Molar tooth which I obtained from this patient, when cut with a saw, the enamel was hard as in the normal but dentine was extremely soft.

Ground Section of the tooth shows following characteristics:- Well-formed enamel with marked Brown Striae of Rhetzius. Dentine shows a very large number of interglobular spaces showing that decalcification of the dentine has taken place.



Micro-photograph of case 1
Long Section stained Car-Fuchsin
showing interglobular spaces.

A.

B.

A. Micro photograph of Case 1 showing numerous dentinal inter globular spaces.

B. Micro photograph of Case 1 showing Exostosis of Cementum between roots.

At apical portion of the roots and between the roots there is evident exostostis of cementum. This I regard as an attempt on part of nature to proliferate to retain roots in position. The point particularly to be observed in this case is that periclasia is present or the early stage of pyorrhoea, Type B.

Various ductless glands have been made answerable for the onset of osteomalacia. Suprarenal by diminished secretion (Bossi), Hypersecretion of Ovary (Bell),(4) Hyposecretion of pituitary (Knowles), Hyperplasia of parathyreoids (Erdheim).

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(2) Acromegaly:-

In looking up the records in the Royal Infirmary of all cases of acromegaly for the past 20 years, I could find no reference to the mouth conditions, except:-

Miss L. Age 58.

History:- Enlargement of feet and hands commenced about age of 40 accompanied by profuse sweating, headache, increased salivation followed later by increase in thickness of the bones of the skull.

Examination:- Typical appearances of acromegaly.

Mouth - had teeth removed for pyorrhoea.

Radiogram of Case III showing calcification above and posterior to pituitary fossa.

Radiogram of Case III showing increase in thickness of femora.

Radiogram of Case III showing thickening of radius and ulna.

Lower.

Upper.

Dental Radiograms of Case III showing alveolar rarefaction.

Case III. Non Pulmonary Osteo Arthropathy - localised Acromegaly.

A.McN. Age.56.

History. Onset of symptoms Nov.1923. Knee joints affected, followed a few days afterwards by swelling and pain at wrists: joints gradually became swollen; exercise diminished pain but increases swelling.

Examination: Skin moist, muscles of arm show wasting; swelling of ankle knee and wrist joints caused by enlargement of the bones. No tenderness or redness of joints. Thickening of both tibia at lower ends, also radii.

Radiogram: subperiosteal thickening most marked in Radius and ulna; there is also rarefaction.

Cranium normal pituitary fossa calcification above and posterior to cavity;calcification in region of pineal body.

Urine: Acid. S.G.1010. No albumen.

Saliva: P_H = 6.4. Wassermann Reaction negative.

Mouth: generalised pyorrhoea present.

Radiogram shows typical alveolar rarefaction. This case, although not a true acromegaly in some respects, clinically resembles it. The bones have not grown in length because of the union of diaphysis and epiphysis having taken place and therefore could only increase in thickness.

N.B. In acromegaly there is an increase in the deposit of fixed inorganic lime due to excessive activity of Pars Ant. of pituitary. The anterior lobe of the pituitary secretes an eosinophile substance which according to Herring (1908) (5) passes/

passes into the third ventricle and thus into the cerebrospinal fluid.

Disease of the gland shows it to have a powerful influence on growth and metabolism (Cushing (6)). To cause endocrine imbalance I repeated the experiment originally carried out by Mr F.W.Broderick namely feeding rabbits on gland extract.

Rabbits were chosen for this experiment as it is probable that there is a relation between the length of the teeth and their hardness, the incisors of these animals being of continuous growth, kept at a certain length by wearing at the biting edge. If they softened they would wear more quickly. I therefore took three rabbits from the same litter one month old, fed them with the same food except that to the food of No.1 was added extract parathyreoid 1/10 gr. + extract suprarenal 1 gr; to that of No.3 was added extract ovarian 5 grs., No.2 used as control.

At the end of three months it was found that the lower incisor teeth of No.1 were 7 mms. in length, those of No.3 6 mms., those of No.2 the control 7.2 mms.

N.B. Gies found that a diet ordinarily considered to be deficient in lime salts produced no effect on the teeth of puppies in 127 days and that it was necessary to reduce the calcium content of the diet of rats to 3 mgms. per day for 70 days before signs of demineralization were found in the teeth. This shows that it is not a question of how much is provided as the effective utilization of what is provided (page 6).

Thus we see after consideration of the cases quoted, in comparison of the two diseases, that under certain conditions the body has the power of Hypercalcification or decalcification as the case may be.

Is there any evidence of the relation of endocrine derangement to acidosis?

McCarrison (7) has published some works on the suprarenal from which he draws the conclusion that in a state of acidosis produced by the feeding of animals on a diet deficient in vitamines, all the endocrine glands with the exception of the suprarenal, atrophy. He concludes that the suprarenal has an emergency action and that in a condition of chronic acidosis, this gland attempts by overacting to increase the amount of alkali assimilated by the body. Sellard says that some chronic acidoses during the period of growth might conceivably account for some of the effects of malnutrition seen in children. Again Crile (8) has shown that a state of acidosis was present with signs of exhaustion of adrenals, thyreoid, pituitary, in a number of diseases, in infections and physical exhaustion. From findings after experimental thyreoidectomy namely decreased elimination of phosphates in urine combined with diminished calcium index of blood serum.

This acidosis due to endocrine derangement, according to McCarrison/

McCarrison is brought about by:-

(1) Direct Disease of the gland or group of glands.
    (a) myxoedema,
    (b) acromegaly,
    (c) diabetes.
(2) Functionally by over stimulation.

(3) Defective feeding.

(4) Intestinal toxaemia.

(a) Case of Myxoedema.

Mrs D. Age 48.

Complained of feeling very tired, pain in all her bones, swelling of face of 7 months' duration.

Present Illness. Felt very weak on slightest exertion, face became swollen under the eyes; tongue seemed to get larger and gave her trouble in speaking. Patient shows marked malar flush, swelling of face on molar prominences, not pitting on pressure, skin bright and dry. Speech slow; extremities almost blue.

Circulatory and nervous systems - normal.

Genito-Urinary System - ceased to menstruate a year ago. When 25 years old menstruation ceased and returned after five years.

Mouth: Had teeth removed for pyorrhoea 3 years ago.

Sellard's Blood Acidosis test (9) - slight degree of acidosis present.(b) In case of localised acromegaly already quoted, slight acidosis was present.

(c) Diabetes.

To investigate the incidence of pyorrhoea or periclasia among diabetic patients I examined 50 cases including indoor and outdoor patients at the Royal Infirmary. In every case without exception where teeth were present various stages of periclasia could be found depending upon the age of the patient and the period of chronicity and severity of the constitutional disease: and in edentulous cases a history of pyorrhoea could always be obtained or that the teeth became loose and fell out. I experienced great difficulty in finding diabetic patients young enough where teeth would be present.

Let us consider some of these cases.

(1) G.McI.	Age 67.	Diabetes of 5 years' duration, upper edentulous, pyorrhoea of lower incisors.
(2) J.McG.	Age 35.	Diabetes of 2 years' duration upper edentulous, periclasia of lower incisors, gingivitis, caries marked, acetonuria +.
(3) Mrs L.	Age 57.	Diabetes of 4 months' duration; edentulous pyorrhoea 10 years ago.
(4) Mrs.B.	Age 62.	Diabetes 2 years' duration;upper edentulous pyorrhoea 10 years ago.
(5) Mrs.C.	Age 59.	Diabetes 2 years duration; edentulous pyorrhoea 5 years ago.
(6) Mrs.T.	Age 72.	Diabetes 4 years, edentulous pyorrhoea.
(7) Mrs.F.	Age 46.	Diabetes 2 years'duration, edentulous 2 years, acetonuria +.
(8) Mrs.E.S.	Age 31.	Diabetes of 2 months' duration, upper edentulous, lower periclasia and caries marked.

Lower.

Dental Radiogram of Case 9 showing alveolar rarefaction.

Dental Radiograms of Case 10 showing alveolar rarefaction in Upper Bicuspid and Upper Centrals.

(9) Mrs.G.	Age 26.	Diabetes 2 years, pyorrhoea of lowers, upper edentulous, acetonuria ++.
(10) J.S.	Age 29.	Diabetes 2 years, periclasia, acetonuria +, upper and lower teeth present.

Radiograms of cases 9 and 10 show typical alveolar rarefaction and absorption, particularly in the case of 10 caries was not present but there was marked recession around the upper first Bicuspid and the upper first Central was beginning to become loose in its attachment.

From observation of these cases I am of the opinion that where the acidosis is more severe as evidenced by the constant appearance of acetone in the urine, periclasia appears early about the age of 25 and is more rapid in its progress but where the acidosis is less severe periclasia is often present before the patients are aware that they are suffering from diabetes.

That Diabetes manifested itself in some way in relation to the teeth has been noticed by clinicians in the past and usually referred to in some vague way, e.g., Graham (10) writes "the teeth are extremely prone to caries or readily fall out."

Tuberculosis.

It has already been pointed out that acidosis would result from decreased elimination of CO_2. This condition would therefore be present among the class of sub-breathers who never quite get rid of their CO_2, e.g., phthisical patients. Accordingly I examined a number of patients at Robroyston Hospital with a view to investigating the prevalence of pyorrhoea alveolaris and its incidence as regards age of the patient and severity of the constitutional disease. In the following table I have noted the gravity of the T.B. pulmonary condition present by means of the Turban-Gerhardt classification (Imperial German Board of Health) viz. - A) B) C) Constitutional condition 1) 2) 3) Lung Condition.

Females.

Name.	Age. (Years).	Turban.	Mouth Condition.
1. Mrs.J.K.	54	C_3	Edentulous. Had pyorrhoea.
2. Mrs.F.	23	C_3	Caries ++, no pyorrhoea.
3. M.C.	39	C_3	Periclasia.
4. F.L.	33	B_2	No pyorrhoea.
5. I.H.	42	C_3	Pyorrhoea.
6. S.G.	22	C_3	Caries +, no pyorrhoea.
7. J.P.	34	C_3	Pyorrhoea generalised.

Table Contd.

	Name.	Age. (Years).	Turban.	Mouth Condition.
8.	A.K.	33	C_3	Upper edent. pyorr. lower.
9.	Mrs.C.	36	B_3	Periclasia.
10.	Mrs.A.	29	C_3	Pyorrhoea.
11.	Mrs.H.	38	B_3	Caries +,periclasia.
12.	B.G.	29	B_2 + Tabes Mes.	Pyorrhoea.
13.	J.C.	36	B_2 + Tabes Mes.	Periclasia.
14.	N.E.	25	B_2	No pyorrhoea.
15.	Mrs.P.	49	C_3	Edent. Had pyorrhoea.
16.	G.P.	19	B_1	No pyorrhoea.Hypoplasia.
17.	C.K.	20	A_1	No pyorr. Caries +.
18.	Mrs.H.	43	B_1	Pyorrhoea.
19.	Mrs.M.	30	C_3	No pyorrhoea.
20.	Mrs.B.	33	B_2	Pyorrhoea.
21.	A.McG.	19	B_2	No pyorr. Caries +.
22.	Mrs.H.	39	C_3	Upper edent.Periclasia lowers.
23.	B.U.	27	C_3	Caries +, pyorrhoea.
24.	T.G.	32	C_3	Caries, no pyorrhoea.
25.	E.G.	27	B_3	Pyorrhoea, caries ++.
26.	M.McF.	27	C_3	No pyorr. Caries ++.
27.	M.McC.	28	C_2	No pyorr. Caries ++.
28.	J.McL.	27	B_3	No pyorr. Caries ++.
29.	Mrs.M.	34.	C_3	Pyorrhoea, caries +.
30.	M.G.	22	B_2	No pyorrhoea.

Males.

	Name.	Age. (Years).	Turban.	Mouth Condition.
1.	A.B.	25	A_3	Pyorrhoea.
2.	J.McK.	45	B_3	Edent. Had pyorrhoea.
3.	R.B.	46	B_3	Edent. Had pyorrhoea.
4.	A.F.	60	B_3	Pyorrhoea lower teeth.
5.	J.C.	23	C_3	Pyorrhoea generalised.
6.	J.W.	31	C_3	No pyorrhoea.
7.	J.H.	23	C_3	Pyorrhoea generalised.
8.	F.L.	34	B_2	Pyorrhoea generalised.
9.	N.D.	20	B_3	No pyorrhoea.
10.	W.B.	29	A_3	Periclasia.
11.	E.C.	22	B_3	Pyorrhoea lowers.
12.	P.S.	38	B_3	Pyorrhoea generalised.
13.	P.B.	32	B_2	Periclasia lowers.
14.	H.P.	39	B_3 + T.B. testes.	Pyorrhoea generalised.
15.	J.T.	58	C_3	Pyorr.lowers, upper edent.
16.	J.W.	33	B_3	Generalised pyorrhoea.
17.	D.K.	20	B_3	No pyorrhoea.
18.	S.McC.	23	B_3	Periclasia uppers.
19.	J.C.	39	B_2	Pyorrhoea generalised.
20.	G.R.	25	B_3	Pyorrhoea generalised.
21.	J.T.	40	B_3	Pyorrhoea.
22.	A.M.	37	B_3	Pyorrhoea.
23.	I.J.	36	A_3	No pyorrhoea.

Table Contd.

	Name.	Age. (Years).	Turban.	Mouth Condition.
24.	A.N.	20	A_3	No pyorrhoea.
25.	P.L.	30	C_3	Periclasia.
26.	G.B.	24	B_3	Periclasia lowers.
27.	R.G.	42	A_2	No pyorrhoea.
28.	W.C.	25	B_3	No pyorrhoea.
29.	W.L.	26	B_3	Pyorrhoea.
30.	J.S.	33	B_3	Upper edent.pyorr. lowers.

Perusal of the above data shows that 76% of males and 57% of females suffered from pyorrhoea, or a total incidence of 67%, also that where the pulmonary condition is more advanced or coexistent with a tubercular condition elsewhere in the body, the onset of pyorrhoea is considerably advanced and manifests itself clinically at an earlier age. In my description of the cases I have used the term periclasia to denote those cases where no pus was present but where involvement of the peridontium was evidenced by slight gum recession and loosening of the teeth. The use of X-rays from a diagnostic point of view would have helped materially in revealing the presence of periclasia or the early stage of pyorrhoea in a considerable number of cases where pyorrhoea could not be identified clinically. It was to be observed how prevalent dental caries was among/

among these patients and how often this disease was co-existent with pyorrhoea. The soft character of the enamel and dentine of the teeth of tuberculous patients is a well-known clinical fact to every dentist, and often his despair from the conservative point of view. This extreme softness is undoubtedly due to the gradual demineralization of the teeth due to the extraction of calcium salts to compensate the acidosis caused by the faulty elimination of CO_2. The visiting dentist to the Robroyston Hospital reported in 1924, after examination of all patients admitted to the institution during the year, that 60% showed obvious pyorrhoea on admission.

My investigation shows a higher percentage and there is no doubt that by the use of X-rays a considerably higher percentage could be obtained.

Anaemia.

Upper.

Lower.

Dental radiograms of Case 1 showing typical alveolar absorption.

In my investigations I noticed, as is familiar to most clinicians, how often pyorrhoea is present among anaemic patients.

Case 1.

Miss A.N. Age 30. Secondary Anaemia.

History:- quite well up till 2 years ago complaint of nervousness, no rheumatic pains, anaemia.

Examination:- Skin has a yellowish tint, mucous membrane pale, thin and poorly nourished.

Mouth:- Periclasia present, no pus tartar, slight gingivitis.

Radiogram: shows typical alveolar rarefaction.

Blood:- fairly severe secondary anaemia present, slight polymorphonuclear leucocytosis.

Sellard's Test:- slight acidosis.

Case II.

W.McA. Age 58. Pernicious Anaemia.

History:- complaint of weakness, breathlessness, loss of weight, ankles swollen recently of 7 months' duration.

Blood:-

R.B.C.	640,000
W.B.C.	5,800
Hb.	22%
C.I.	1.5

Blood Film shows numerous poikilocytes, a few nucleated red cells, marked anisocytosis, polymorphonuclear leucocytes predominate.

Teeth:- Pyorrhoea present, gums very pale, plaques of seruminal tartar.

Leukaemia.

Dental radiogram of Case III showing marked alveolar absorption.

Case III.

J.C. Age 52. Leukaemia.

History:- One month ago feeling of extreme weakness, constipation and sickness following.

Examination: Blood:- R.B.C. 820.000
W.B.C. 6,500
Hb. 24%
C.I. 1.5

Blood Film:- Red cells vary considerably in size; several nucleated reds; white cells all of the granular type.

Mouth:- Gums pale, large plaques of seruminal tartar, no pus, periclasia present.

Radiogram shows typical absorption of alveolus.

Remarks:- I have quoted these three typical cases to show that periclasia was present. Periclasia in these cases is a result of the idiopathic condition causing the anaemia and not the cause of the anaemia. The formation of the plaques of seruminal tartar is due to the ready exudation of blood serum around the teeth.

The Hydrogen Ion Concentration of the Saliva in Pyorrhoea Alveolaris.

Dr H.E.Starr of Pennsylvania working on the Hydrogen ion concentration of mixed saliva, by the calorimetric method, in 1,300 different specimens, his subjects being divided into three classes, subbreathers, psycopathics and normals, reports that as regards the subbreathers the salivary pH was found to vary from 5.4 to 6.6 (i.e. more acid than normal); in psycopathics the pH was found to vary from 6.9 to 8.0, i.e. towards increased alkalinity, and in the normals the pH was found to vary from 6.0 to 7.0. Dr Starr (14) made simultaneous estimations of the salivary pH and the CO_2 tension of alveolar air and found that in every instance the Hydrogen ion concentration of the saliva varied directly with the CO_2 content of alveolar air, i.e. the greater the salivary pH the less the CO_2 tension. The CO_2 content of alveolar air is dependent on the CO_2 content of the venous blood which again depends on the alkali content of the blood. The CO_2 and alkali must be in the ratio of 1/20 for the blood to be in Acid-Base equilibrium; if one is increased the other must be increased in like proportion. Hence in Acidosis there is a constant drain on the alkali reserve to/

to preserve the 1/20 Ratio. In acidosis an excess of CO_2 tends to collect in the tissues similar to the condition of anoxaemia produced by high altitudes. When the Respiratory centre has become gradually used to the excess of CO_2 there is an elimination of base from the body which takes place by all excretory channels, e.g., urine, saliva.

If a Rabbit is fed on inorganic Acids so as to cause Acidosis the urine on examination is found to have an increased quantity of ammonia present and also of inorganic bases Na, K, Ca, and Mg, last two apparently derived from bones. (Wells (15)).

This will give an increased quantity of alkali in the latter secretion which should show itself in its reaction. Therefore if pyorrhoea is due to an acidosis, after consideration of the foregoing, it will be obvious that <u>the saliva of pyorrhoeic patients should be more alkaline than normal</u>.

That this is the case, using my own salivary pH as normal (pH = 6.6) I estimated the Hydrogen ion concentration of the saliva of all patients suffering from pyorrhoea using the calorimetric method of standard tubes, and found the Hydrogen ion concentration of the saliva to vary from 6.6 to 7.4.

At Robroyston I made observations on the saliva of phthisical patients and found these to be within the limits as found by Dr Starr for subbreathers.

A.N.	Salivary	pH = 7.2	Pyorrhoea.
A.McN.	"	pH = 6.8	
A.M.	"	pH = 7.2	
A.F.	"	pH = 7.4	
D.F.	"	pH = 6.3	Subbreathers.
Mrs.M.	"	pH = 6.1	
J.S.	"	pH = 6.4	
L.R.	"	pH = 6.1	
J.P.	"	pH = 6.2	

Bacteriology.

The bacteriology of Pyorrhoea is still obscure. In 1923 an excellent review of the subject was given by Professor Glynn who points out that although streptococci are the chief pathogenic organisms in periodontal infections, evidence of specificity is lacking. They do not satisfy all Koch's postulates; they can be cultivated but these streptococci do not produce the disease in animals. Kritchevsky and Sequin believe on less conclusive evidence that spirochaetes are the causal organisms. Bass and John's theory of the Entamoeba Buccalis may now be dismissed as a cause of pyorrhoea. With so little known of the aetiology and pathology of pyorrhoea it is no wonder that there is lack of unanimity.

Vaccine Therapy. Goldenburg's injection of vaccine locally into the gums based on Besredka's assertion (12) that in vaccine therapy it is necessary for the vaccine to follow the path of the infection for which treatment is given has sometimes secured temporary improvement.

Mr Arthur Bulleid (13) states that vaccines must be autogeneous but points out that as long as no causal organism is isolated vaccine therapy remains empirical.

From an examination of 50 cases I found the following organisms invariable present:- spirochaetes, fusiform bacilli, streptothrix, buccalis and streptococci on culture. From observations I agree with Professor Glynn that evidence of specificity/

specificity is lacking and am of the opinion that these organisms, ordinary commensals of the mouth, become more pathogenic once 'pocket formation' is established and give rise to sapraemia and septic intoxication.

Pathology. The latest and most extensive work on this part of the subject has been done by Dr Harold Box (11) of Toronto. He gives an elaborate classification of 13 different forms of gingivitis, a refinement both excessive and confusing. His conception appears to be a simple ulceration of the gum margin which extends into the periodontal membrane. A pocket is formed which becomes deeper. A fibrous change occurs in the periodontal membrane rarefying pericementites fibrosa while infection plays a part in the later stages. He does not believe that bacterial invasion is the primary factor. The first cause in his view is an overfunctioning of the teeth due to a deviation from their normal arrangement which so strains the periodontal membrane so as to initiate the development of rarefying pericementitis fibrosa - co called traumatic occlusion.

In my experience I consider Dr Box's theory of causation - namely traumatic occlusion, untenable in view of clinical experience. Many of the cases of pyorrhoea observed by me were in ortho occlusion.

The essential pathological lesion in pyorrhoea is a destruction of bone, gum and periodontal membrane. This leads to/

to the formation of a pocket round the tooth which becomes an ideal receptacle for bacteria and putrefactive debris. A discharge of pus is often present but is not an invariable feature. In this country the view that has received most support is that pyorrhoea commences as an inflammation of the gum margin, extending down to roots of the teeth, attacking periodontal membrane formation of a pocket lined with infected granulation tissue; concurrently with these changes there is absorption of alveolar bone. This description fits the cases I have seen which fall into my first class of aetiology, where seruminal or salivary tartar is the exciting cause.

But many cases I consider belong to the constitutional type which begin as an absorption of alveolus or periclasia, due to the absorption of lime salts to compensate a condition of chronic acidosis in the conditions in which I have indicated.

Treatment.

Neither drugs nor ionic medication can replace the careful instrumentation and mechanical cleansing. Conservative treatment may result in an arrest of the disease but there will never be a restitutio ad integrum. Extraction is the safest course which is a standard of treatment more consonant with general surgical principles. Provided that intervention comes early before the disease is too far advanced it is possible to arrest its progress and to ensure a functional mouth.

With/

With regard to the constitutional type, the prognosis is very bad; once the lesion is present it will be progressive. Local treatment is of no value and as the constitutional disease does not respond well to general treatment loss of the teeth is inevitable.

Pyorrhoea alveolaris looked at from the constitutional point of view becomes not the simple thing that Turner would have us believe, i.e., due to food stagnation and infection, but rather the result of a metabolic disturbance, thus accounting for its presence in comparatively clean mouths and its absence in the dirty,and also for the disappointments so often met with in treatment.

It has been stated by Mr Broderick that pyorrhoea and dental caries are antagonistic conditions. I agree with Mr Turner in opposition to this statement when he says "I utterly dissent." I was often struck in the course of my investigations how often pyorrhoea and caries existed pari passu.

I again agree with Mr Turner and differ from Mr Broderick regarding the part played by subgingival tartar in the aetiology of this condition; Mr Turner holds and I think quite rightly that this tartar is simply a result of the condition,while Mr Broderick believes it to be the exciting cause.

Conclusion.

On reviewing the literature on the subject of pyorrhoea alveolaris from the first publication by Fauchard in 1746 up to the present day, as regards aetiology the opinions of the various writers may be grouped as follows:-

(1) That the disease originates in some constitutional state.

(2) That the disease is caused entirely by local irritation.

(3) That the disease is due to infection of the tissues with micro-organisms.

(4) That the disease is induced by deficient exercise of the teeth, gums, and alveolar process.

I submit

(a) That my investigations have shown that the disease is due to (1) and to a combination of (2) and (3).

(b) That the cause of the constitutional type is a chronic Acidosis.

(c) That thisacidosis is produced by endocrine derangement or decreased elimination of CO_2.

(d) That this acidosis causes an absorption of alveolus due to the extraction of calcium salts for purposes of compensation.

(e) That there are two distinct types of pyorrhoea differing in their clinical appearances at the onset but in the late stages so closely resembling each other that they cannot be differentiated.

Criticism.

When demineralization is taking place in order to compensate an acidosis why is the alveolus attacked specially?

Are the other bones affected?

I have no doubt that the other bones are affected. I have had Radiograms of the femora taken in a number of cases to show decalcification, without result. Undoubtedly the degree of decalcification is too slight to show any change and it is only in the severe cases as in the osteomalacic case that X-ray evidence could be obtained.

I am of the opinion that it is masticatory stress which localises the demineralization process to the periodontium. At present this must remain a theory, but as Elliott says "Medicine owes no debt of gratitude to those who teach to her theories without proof."

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