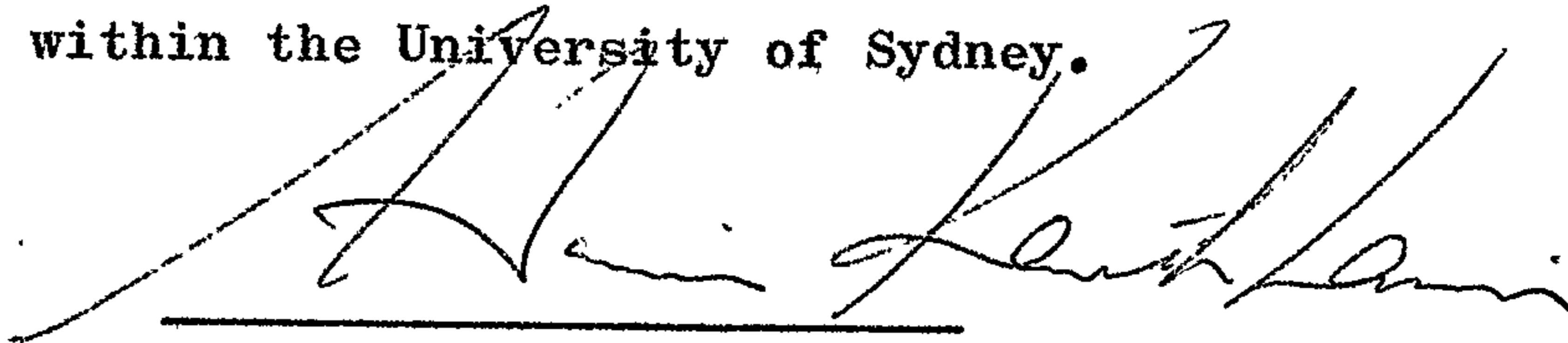


A STUDY OF GINGIVITIS

IN PREGNANCY

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A thesis embodying an original research programme
submitted by the undersigned as requirement for
admission to the degree of Master of Dental Surgery
within the University of Sydney.



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1. PREFACE

In setting out this thesis, an attempt has been made to give a detailed investigation of the gingival changes which may take place in pregnancy and with particular reference to pregnancy gingivitis or, as it is referred to in this thesis: gingivitis gravidarum. Gingivitis gravidarum is that gingivitis which occurs in the pregnant state and is attributable to, or aggravated by the gravid state. A clinical survey has been carried out and a detailed study of all available literature made. As a result of this study, comparisons are drawn between Australian born and immigrant groups.

There are many unsolved problems concerning the condition, and although this investigation does not cover any new ground in the light of world research, it is, to the best of my knowledge, the first time that a survey as comprehensive as this has been made in this country.

I am indebted to the Department of Preventive Dentistry with particular thanks to Professor N.D.Martin, Mr. S. Levine and Mr. P.D. Barnard for their advice and help in preparing this study. My sincere appreciation goes to the Superintendent of the Women's Hospital, Crown Street, Sydney, Dr. J. Murray, and the various members of the medical and nursing staff who made it possible for me to carry out my work.

2. INTRODUCTION

Pregnancy is associated with great anatomical and physiological changes of varying kinds,⁽⁵⁴⁾ and in these changes there are some which could be looked upon as pathological. The changes observed in the gingivae during pregnancy have been the object of interest for a long time. It has been a matter of discussion whether there is a specific form of gingivitis in pregnancy, or whether it occurs occasionally, or at all, among pregnant women. Three main schools of thought have prevailed in regard to its etiology. Some believe in a purely local etiology,⁽⁷⁷⁾ others in vitamin C as a primary factor,⁽⁵⁵⁾ and still others in the importance of hormonal factors.⁽¹³⁴⁾

When a woman becomes pregnant, the integrity of every organ and every function of her body is severely tested. It is common knowledge⁽⁵⁶⁾⁽¹¹¹⁾ that the growth of existing tumours may be accelerated during pregnancy and that naevi⁽¹⁾ are predisposed toward tumour formation during gestation.

There are various observations of haemangiomas on the face and neck with growth with pregnancy.⁽¹³⁴⁾⁽¹⁰⁹⁾⁽¹¹⁹⁾⁽¹¹⁸⁾ Greenhill⁽⁴⁸⁾ says that pregnancy may be regarded as either causative or predisposing to a stomatitis. Ulcerative and membranous lesions of the mouth may vary from simple herpetic lesions to recurrent aphthosis with similar lesions in the vaginal vault.

This investigation has been conducted in an attempt to expand our present knowledge of the possibility of changes in the gingival tissues of a group of pregnant women in this city. The seeming predisposition, ante-partum, of the gingival tissue to haemorrhage and the altered appearance of the gingivae frequently observed during pregnancy (when compared to the pre-pregnant and post partum condition of the gingivae) initiated this investigation.

As in any other disease, to fully understand and appreciate gingivitis in pregnancy, a thorough investigation of the associated sciences which affect the mouth, must be made. The many and varied pathological changes seen in the oral cavity can never be diagnosed with accuracy unless the normal anatomy, histology and physiology of the parts are well known. Many of the changes seen in the mouth in the gingivitis which occurs during pregnancy, cannot be correctly interpreted or related, except in the light of our knowledge of the effects of nutrition, hormones, and local etiological factors.

In view of this, I have included review sections on hormones, and nutrition, a description of the periodontium in its normal and pathological states as well as histochemical changes.

The pertinent literature concerning all aspects of gingivitis in pregnancy has been examined, and an attempt has been made to correlate the important factors regarding this subject. To this has been added the results of my own survey, to throw further light on the incidence of "Gingivitis Gravidarum". The survey has been

focused upon some of the etiological factors amongst a group of women in Sydney, which included a large number of migrants.

The material consisted of 684 pregnant girls and women, varying in age from 13 to 45 years - both married and single - from all social stratas, but with the majority coming from the lower income bracket. Of the number examined, 28 were patients attending my own surgery. The balance were public patients taken at random from The Ante Natal Clinic at the Women's Hospital, Crown Street, Sydney, during 1963. Patients at all stages of gestation were examined once to determine the existence of gingival disturbances. One hundred and forty-one patients were examined two or three times to note any changes which may have taken place during the time of gestation. Seventy-five patients were seen post natively in the Post Natal Clinic at the same hospital. The women represented a wide range of nationalities, the hospital being situated in an area which caters for a large migrant population. Oral hygiene was not particularly good in most cases, and very few patients were in the habit of receiving regular dental care.

Whilst all etiological factors will be discussed, those that were considered in this survey were: nationality, age, number of pregnancies, frequency of brushing, previous dental and medical history, stage of gestation, oral hygiene and general gingival condition. My aim was to ascertain if any of these factors were directly or indirectly connected with the appearance of gingivitis

in the gravid patient, and at the same time to find out the actual incidence of gingivitis attributable to pregnancy.

3. SURVEY OF LITERATURE

i). HISTORICAL.

In August, 1960, the expert committee on dental health met in Geneva to discuss Periodontal Disease.⁽¹²³⁾ Dr. M. Candau, the director general, in opening the session stated: "That in the past periodontal disease had largely been overshadowed by dental caries. In point of fact, the public health problems raised by periodontal disease were as great and often greater than those raised by dental caries. It was essential to ensure that the gains made in the prevention and treatment of dental caries were not lost in later life by the ravages of periodontal disease." Being one of the most widespread diseases of mankind, no country is free from it, and having a high prevalence it constitutes a major problem in modern dental practice.

Just as periodontal diseases have been described as existing in earliest records, so it has been observed from early times that pregnancy had a profound effect on the gingival tissue. Long before endocrine changes in pregnancy were known or their effect on oral tissues were suspected, observers have recorded changes taking place in the gingiva during gestation.

In 1877 A. and D. Pinard⁽⁸⁵⁾ discussed gingivitis of puerperal women, stating that it was more frequent than is commonly admitted.

The remote cause was pregnancy, but what the proximate cause was, they were not certain. They said it occurred towards the fourth month, but rarely sooner, going away a month or two post partum, especially in women who do not suckle their infants.

A solution of Iodine or Glycerole of Tannin and Potassium Chlorate was used which, while producing good effects, were far from bringing about a rapid cure.

Zentler,⁽¹²⁵⁾ in 1912, gives the case history of a patient whose general condition was very unsatisfactory during her pregnancy. The manifestations of her poor general health were very evident in her mouth. The gingival condition became quite bad, although very few deposits of calculus were present. During her next two pregnancies marked hypertrophy took place, despite careful treatment by the author. After each pregnancy the hypertrophy subsided, but at a slower rate in each succeeding one.

Ziskin and Nesse,⁽¹³⁴⁾ Fraser,⁽³⁶⁾ Kutscher,⁽⁶¹⁾ Tiilila,⁽¹¹¹⁾ Monash⁽⁷⁷⁾ and Levy,⁽⁶³⁾ in their dissertations, have traced back references to gingivitis in pregnancy to the 18th and 19th centuries showing that long before the realisation and acceptance of the germ theory, inflammation of the gums was a recognised disease of the soft tissue of the oral cavity.

Until Ziskin⁽¹³³⁾ and his co-workers attempted to solve the problem of the cause of gingivitis in pregnancy, little had

been done to define the etiology. In 1926 Monash,⁽⁷⁷⁾ reporting his findings on six cases of proliferative gingivitis of pregnancy, stated that the condition was entirely inflammatory in nature and merely represented an exaggerated degree of generalised gingivitis. He maintained that there was no gingivitis peculiar to pregnancy.

The early reports of gingivitis in pregnancy take the form of noted observations. It is only in recent years with the marked advances in dental science, that true and accurate investigation has been carried out. The very fact that the leading authorities on the subject have not reached agreement on a definite cause, makes one realise that several factors are involved in this complicated condition.

II. ANATOMY and HISTOLOGY of the GINGIVA.

The tissues which surround and support the tooth are referred to as the "periodontium." The periodontium is defined as the investing and supporting tissues of the tooth - namely the periodontal membrane, the gingiva, cementum, and alveolar bone. The periodontium is a functional organ and the different tissues do not attain their mature form until the tooth has been subject to the forces of mastication. (123)

Changes beyond the range of normal variation in the periodontium constitute periodontal disease. The term periodontal disease in its broadest sense may be defined as being all those pathological processes that involve the periodontium. They can be limited to one of the constituents of the periodontium, e.g., gingivitis, or include several or all of the tissues.

The tissues in the oral cavity which are usually affected by pregnancy, and which show changes during gestation will be described, this being the gingiva.

The gingiva is divided anatomically into five main groups.

1) Free marginal gingiva, 2) free gingival groove, 3) attached gingiva, 4) interdental papilla and 5) mucogingival junction. (102)(103)

Free marginal or unattached gingiva.

This is a roughly triangular portion of the gingiva which encircles the tooth in a collar-like fashion. Commencing at the knife edge of the free gingival margin, it extends in an apical direction to

an imaginary horizontal line from the coronal boundary of the epithelial attachment to the labial or buccal surface of the gingiva at the so called free gingival groove. Its third boundary forms the soft tissue wall of the healthy gingival crevice or sulcus.

Free gingival groove.

This groove develops on the labial or buccal surface. It is not always clearly defined, so that the division between the unattached and attached gingivae is not always discernible clinically.

Attached gingiva.

This zone extends from the free gingival groove to the mucogingival junction and the start of the alveolar or areolar mucosa. It is firm, resilient, and tightly bound down to the underlying cementum and alveolar bone. The attached gingiva is sometimes subdivided as follows: cemental gingiva - that portion of the gingiva attached to the cementum, and alveolar gingiva - that portion attached to the alveolar bone.

Interdental papilla

This zone is roughly pyramidal in shape and is the portion of the gingiva which extends into the interproximal space. If the surfaces of the teeth are in contact, the interdental papilla fills the embrasure and terminates just beneath the contact point. In the absence of proximal contact the gingiva is firmly bound down to the alveolar bone and forms a smooth rounded surface.

Mucogingival junction

In health, there is clinically, a definite line of demarcation between the attached gingiva and the alveolar mucosa. The change in colour from pink to red is very definite in a healthy periodontium. This definite colour change is an important diagnostic feature that is indicative of the health of the periodontium.

The gingival sulcus.

The normal gingival sulcus is defined as the shallow groove around the tooth, bounded on one side by the surface of the tooth, and on the other by the epithelium lining the free margin of the gingiva. It is a shallow v-shaped depression with its base at the most coronal level of the epithelial attachment of the tooth's surface. Clinically, the depth of normal gingival sulcus barely permits the entrance of a thin blunt probe. The depth of the sulcus can vary in individuals from zero to several millimetres. The depth of the sulcus is related to the health of the periodontium. The shallower the sulcus, the smaller are the chances for possible development of pathological conditions in it.

Clinical appearances of the gingiva.

Normally the gingiva is coral pink, and firmly bound down to the underlying bone. It presents a stippled appearance like that of orange peel. The stippled effect is caused by sharply outlined depressions within the epithelium. They are not present on the marginal gingiva, and since the marginal borders of the interdental

papillae are marginal gingivae, this area is smooth too. The attached gingiva is stippled as is the central portion of the interdental papillae. More than any other clinical symptom, the colour of the gingival tissue of the mucous membrane of the oral cavity is an aid in diagnosing not only periodontal disease, but also systemic aberrations. This colour of the gingiva is the combined result of the degree of thickness of keratinisation of the epithelium, the vascular supply and the presence of pigment containing cells. The shade or intensity of the normal coral pink varies considerably in different individuals. There is a correlation between the colour of the gingiva and the general cutaneous pigmentation. Thus in a fair complexioned person, with blond hair, the gingiva would be lighter than in a swarthy brunette individual. In addition to varying shades of pink, there is also physiologic pigmentation that is prominent in members of the coloured races, such as the aborigine or negro. It also occurs in others such as the Mediterranean people. This pigmentation is due to an increase in the number of melanin containing cells. In the gingiva of most individuals, with the exception of abbinos, melanin containing cells are present, regardless of whether the gingiva presents any clinically detectable pigmentation.

Keratinisation.

The gingiva is composed of two separate parts, the epithelium and the connective tissue. The gingiva surrounding the teeth is sub-

ject to forces of friction and pressure in the process of mastication. The character of this tissue shows that it is adapted to these forces. The epithelium is the stratified squamous type. It covers the free marginal gingiva, the interdental papilla and the attached gingiva. It continues as the lining of the gingival sulcus, and extends down to terminate on the tooth's surface as the epithelial attachment. According to the behaviour of the surface layer, four types of gingival epithelium can be distinguished. They are described as follows:

- 1) In fully keratinised epithelium the surface layers consist of flat, tightly packed horny scales, the transformed surface cells. Nuclei are absent.
- 2) In parakeratosis, the surface cells seem to consist of keratin but have retained pyknotic nuclei.
- 3) In incomplete parakeratosis, specific stains (e.g. Mallory's stain) show the surface layer divided into two. The deeper layer stains like keratin, but this stain is lost in the superficial layer, probably by the influence of oral fluids on the incompletely differentiated keratin of the nuclei containing cells.
- 4) Where keratinisation is lacking, the flat surface cells retain their nuclei.

The most frequent type is parakeratosis - about 50%. The next frequent type is incomplete parakeratosis - 25%. Then follows full keratinisation - 15% and non keratinisation - 10%. Presence of inflammation and/or glycogen seems progressively to interfere

with keratinisation. Clinically it is difficult to differentiate between parakeratosis and keratinisation. Below the keratinised layer there is the granular layer. However, the granular layer is only present when the keratinised layer is present. The next layer is the prickle cell layer, which is composed of several rows of polyhedral cells which connect with each other by fine spinous processes known as intercellular bridges. The basal cell layer is next and consists of a single row of dark staining cuboidal cells which are anchored to the basement membrane by short protoplasmic processes.

Connective tissue.

The connective tissue is composed of the lamina propria, the submucosa and the periosteum. The lamina propria may be divided into two portions:-

- a) a papillary layer which is immediately subjacent to the epithelium, consisting of papillary projections, which interdigitate with downward epithelial pegs,
- b) a reticular layer which is in contiguous position with the fibrous connective tissue submucosa. This in turn blends with the periosteum of the alveolar bone.

The fibrous structure of the submucosa accounts for the immovable nature of the attached gingiva. The papillary layer of the lamina propria contains the vascular supply and the innervation of the gingiva.

iii) PREGNANCY AND THE ENDOCRINE GLANDS.

The duration of the average pregnancy is about 280 days. It is divided into trimesters of approximately 3 months, each. It has been referred to as a disease of nine months duration (22) and imposes a definite strain on a woman, so that it is essential that she be kept in the best physiological condition for the whole time. The character and mental outlook of the patient is frequently changed, particularly in the early months and again in the last few weeks. (32) The pregnant woman is often hypersensitive to pain. There is often a considerable metabolic disturbance especially in the early months. Generally speaking, in the middle period of pregnancy the woman achieves her nearest approach to equilibrium. She has adapted herself to the metabolic changes of the early months and the strain of the baby's processes are not as great as in the later ones.

Pregnancy is not merely a local condition that manifests itself by an enlarging of the uterus by a maturing embryo confined within its walls. The most obvious changes that occur are, of course those taking place in the generative tract. In the past, changes taking place in pregnancy were attributed to many causes, but recent work indicates that these changes are brought about by hormones. (67) The blood increases in volume with a slight decrease in cell and haemoglobin content. (63) Certain changes in the nasal mucosa such as reddening and thickening take place.

Marked changes in the voice of a large percentage of pregnant women who sing have been noted, caused by changes in the larynx. In the digestive tract, we find very early in pregnancy, minor disorders of digestion. Constipation affects many expectant mothers due, in part, to pressure from an enlarged uterus and to a lack of tone in the abdominal muscles, caused by their distention. In the early stages about one half of pregnant women are subject to nausea and vomiting. In the urinary tract, the kidneys are under increased strain while the bladder is compressed by the enlarged uterus.

Numerous diseases and conditions have been attributed to underfunction or over function of one or more of the endocrine glands and some of these are manifested in the soft oral tissues. As these tissues are so extremely sensitive to any change in hormonal metabolism it can readily be appreciated that great changes in many of the endocrine glands in pregnancy are reflected in the gingival tissues. Moreover, because of the intense relationship between most of the endocrine glands and pregnancy, hormones exert marked and widespread influence, and have been shown to quite dramatically control the development and maintenance of the oral tissues. (62)

Hormonal upsets of ovarian origin affect females at puberty, during the menstrual cycle, and at pregnancy. Although puberty can last for some time, the gingival upset which may occur, corrects itself after this phase passes. (49)

The hormonal alteration of menstruation is of short duration and rarely causes gross change. However, gestation, occupying nine months of hormonal change, presents a very different picture. This action of hormones on the gingival tissues has been strongly stressed by Ziskin and his co-workers.

Endocrine Glands

Most of the glands of the body have ducts. The secretions which these glands manufacture are poured out through such ducts. Typical examples are the salivary and gastric glands. Another group are the endocrine or ductless glands which elaborate substances that are released directly into the circulating blood stream. These secretions usually contain hormones. A hormone⁽¹⁵⁾ is commonly defined as a chemical substance which, formed in one part of the body, is carried in the blood stream to other organs, or tissues, which it influences in a specific manner. Although the term hormone implies stimulating or enhancing action, certain hormones are now known to exert a depressing effect on certain of their target tissues. (Of historical interest is the fact that the term "hormone" was first applied by Bayliss and Starling in 1902 to secretin a substance produced in the duodenal mucosa, which has a stimulating effect on the secretions of the pancreatic juice and bile.)

Hormones are the chemical messengers of the body.⁽⁵³⁾ We

have reasons to believe that they act by influencing enzyme systems and membrane permeability. Some of the hormones are protein, others are related to the steroids and still others are relatively simple compounds.

1. Hypophysis. (15) (62) (134)

This gland may be divided into -

1. Pars tuberalis
2. Anterior lobe
3. Posterior lobe
4. Pars intermedia

The pars tuberalis and pars intermedia are of little physiological value. The only clinical alteration of importance resulting from malfunction of the posterior lobe is diabetes insipidus. This disorder is characterised by a great urinary output which may amount to 20 litres a day.

Clinical and experimental work indicates that the anterior body of the hypophysis or pituitary, controls or influences the actions of other endocrine glands. The location of the hypophysis is at the base of the brain and the fact that it develops in conjunction with the oral cavity, gave rise to the opinion among early workers that this gland was concerned with the production of phlegm. Because of this concept, the organ was originally called the pituitary gland. There is, however, a more appropriate term - hypophysis - which means "to grow under".

A consideration of this gland forms a valuable starting point in determining endocrine relationship. The activity of the hypophysis is clearly demonstrated by the striking changes which result from extensive functional deficiencies of this gland in man (e.g. Simmond's disease) and from hypophysectomy in animals. In Simmond's disease, and following hypophysectomy, widespread disorders result due to atrophy of the thyroid, gonads, and adrenal cortices. The thyroid hormone has a depressant action on the thyrotrophic function of the hypophysis since in hyperthyroidism there are to be found subnormal quantities of the thyrotrophic substances in the urine. Between the hypophysis and the adrenal cortex there is also a reciprocal relationship. It now seems certain that the anterior lobe secretes several distinct hormones. At least five have been isolated and it appears that there are further ones.

1. There are two gonadtrophic hormones: (a) F.S.H. and (b) L.H. The first stimulates maturation of the graafian follicle in the female, and spermatogenesis in the male, and the second controls the corpus luteum in the female, and the cells of Leydig in the male. In the male it is often referred to as I.C.S.H.
2. Adrenocorticotrophic hormone. This is commonly referred to as A.C.T.H. and controls the adrenal cortex.
3. Thyrotrophic hormone. (T.S.H.) is governed by the level of circulating iodine, and acts upon the thyroid gland.

4) Lactogenic hormone serves two functions: (a) to stimulate milk production and (b) to help maintain the corpus luteum during the latter months of pregnancy. This hormone is therefore called prolactin, because of its first function and the luteotrophic hormone, because of its second role.

5) Growth hormone. It is capable of producing exaggerated growth both in man and in experimental animals. During pregnancy, there is an increase in size and weight of the hypophysis. Cytologically there is an active secretion as indicated by granules, hypertrophied Golgi apparatus and abundant mitochondria.

Following hypophysectomy ⁽¹³¹⁾ there are extensive degenerative changes in the gingivae and oral mucous membranes of the female rhesus. The keratin layer is, for the most part, absent, the prickle cell layer shows marked evidence of degeneration, the cells appear irregular in shape and size and the intercellular bridges are poorly preserved.

2. The Thyroid Gland. (15) (53) (134)

The word thyroid means literally an oblong shield. It is so named because it forms a shield overlying the larynx. It is closely associated with the pituitary function and is controlled by a hormone of the anterior lobe of the hypophysis - the thyrotrophic hormone. The primary function of the thyroid gland is to govern the basic metabolic processes. It has manifold effects on diverse organs and functions. Although clinical

data does correlate the thyroid with gonadal functions, no specific hormonal connections have been verified between the two.

Thyroxin, which is the circulating form of the hormone, decreases the ovarian response of hypophysectomised rats to injections of extracts of urine of castrated women, and it appears that thyroxin inhibits the gonads directly. In any case, the action of thyroxin may be such as to produce a state of low estrogen functions.

Parenchymatous changes, increased vascularity, and epithelial hyperplasia, causing enlargement of the thyroid gland, take place during pregnancy. There is an increase in the basal metabolic rate because of increased demands on the part of the foetus. Thyroidectomy in Rhesus monkeys causes hyperkeratosis in the alveolar and areolar gingivae, epithelial intercellular oedema, increased mitosis of the basal cell layer, and deposition of mucous staining material and glycogen. Similar changes are seen in inadequately treated cretins and myxoedematous humans.

3. The Adrenal Glands (15) (53)

The adrenal cortex plays an important part in carbohydrate and mineral metabolism, capillary permeability, capacity of muscles to respond to stimulation and resistance to stress. The gonads are closely associated with the adrenal cortex.

Functional or organic alterations in the adrenal cortex

which produce abnormal changes in the sexual sphere, are referred to as adrenogenital syndrome. Since the changes vary with the type of hormone produced, the age and sex of the patient, several syndromes can be distinguished. During pregnancy, the adrenal glands are enlarged, but the diffuse hyperplasia is functional, and disappears after parturition. The change is a physiologic one.

In Addison's disease which is due to an insufficient production of the adrenocortical hormone, there is increased thickness of the surface keratin of the alveolar gingivae with parakeratosis, hyperplasia of the basal cell layer and a great increase of connective tissue. In adrenal hyperplasia, however, there is reduction of the surface keratin, pyknosis, hydropic degeneration in the stratum spinosum, and hyperplasia of the basal cell layer.

4. The Ovaries. (15) (53) (134)

The development of the sexual organs as well as the characteristics associated with femininity and masculinity are under the control of the hormones secreted by the ovaries in the female and the testes in the male. These organs, commonly referred to as gonads, serve two functions: 1) the secretion of hormones, 2) the production of the germ cells necessary for reproduction. The ovaries are two nodular bodies situated one on either side of the uterus.

At birth, the ovary contains thousands of immature graafian

follicles. Usually one of these follicles matures each month after puberty. During the process of maturation, an ovum is formed within the follicle. At ovulation, the graafian follicle ruptures and the ovum is ejected. After ovulation, the graafian follicle is seen to contain a small clot of blood. This structure is called corpus haemorrhagicum. Soon afterwards it assumes a yellow colour. It is now termed the corpus luteum. Finally, the corpus luteum is replaced by a white fibrous material and this body is called a corpus albicans.

The actual sex life of the woman extends from about the ages of 14-48 years, although there are naturally wide variations. During the child bearing age the ovaries produce two principle hormones - estrogen and progesterone. Estrogen secreted mainly by the developing follicles, is essential for the growth and development of the accessory sex organs. It is also formed by the corpus luteum. Both pituitary gonadotrophic hormones (1) follicle stimulating hormone (F.S.H.) (2) Lutinizng hormone (L.H.) and also (3) lactogenic hormone (prolactin) are involved in stimulation of estrogen secretion by these structures. In lower animals, estrogen is responsible for the appearance of the state called estrus. During the period of estrus the female animals is highly receptive to the male and there are changes in the sexual organs as well as in the secondary sexual characteristics. Although there is no distinct estrus period in the human , estrogen plays a very important role.

The naturally occurring estrogens in the human are -
E-estradiol, estrone and estriol. Estradiol has been found in the placenta and in the urine during pregnancy. Estrone is present in the urine of adult males, pregnant and non-pregnant women. Estriol is present in the urine of pregnant women, and in human placenta. Normally only relatively small amounts of estrogen are found in the urine. During pregnancy, however, there is a marked increase in the blood level and urinary excretion of estrogen. The estrogen content of pregnancy urine rises gradually, until parturition, and then decreases markedly over the next few days. In the latter months of pregnancy, the least potent form, estriol, is present in the greatest amount. At term however, it disappears from the urine, and estradiol rises suddenly in concentration. During the first eight months of pregnancy, 99% of the estrogen is excreted conjugated with glucuronic acid as the glucuronide which is an inactive detoxified form, but shortly before parturition, the amounts of unconjugated active estrogen increases markedly.⁽¹³⁴⁾ In addition, the estrogens of pregnancy serum appear to be intimately bound to the protein fraction as indicated by - 1) they do not pass through a collodion membrane, 2) they are precipitated in the protein fraction, and 3) prolonged hydrolysis is necessary to liberate them from the protein fraction. The amount of protein combined hormone ranges from 23-50% of the total estrogens. Thus

it is seen that although estrogen is present in increased amounts in the urine and serum during pregnancy, most of it is present in bound inactive form. Nevertheless, the total secretion of estrogen is so high, that sizeable quantities of free estrogen are available for body needs.

In contrast to the high levels of chorionic gonadotrophic hormone attained early in pregnancy, ⁽⁹⁰⁾ the estrogenic levels are increased very slowly after implantation. From about the sixteenth week of pregnancy there is a progressive increase in urinary estrogen until about the thirty-third week. From this time until shortly before term, the excretion curve rises more sharply. At its peak, the estrogen excretion may range as high as 25-50 mgms per 24 hours. In some instances urinary estrogen level has been observed to fall a week or two before delivery, but in other cases, a high level of urinary estrogen falls very rapidly and very little can be recovered within 4 - 7 days post partum.

Progesterone. (15) (53) (134)

Progesterone is secreted by the corpus luteum of the ovary during its period of functional activity. It appears suddenly on the day of ovulation or perhaps a day or two earlier, as indicated by the urine. Although progesterone does not appear in the urine, several metabolites of this hormone can be recovered from its source. The metabolite appearing in largest quantity

is pregnandioli.

During the first three months of pregnancy, the pregnandioli level remains approximately the same level as that found during the corpus luteum phase of the cycle, ranging between 4 - 10 mgms per 24 hours. After about 13 weeks the level increases steadily and by the 21st week may range from 10 - 40 mgms per 24 hours. By the 7th and 8th months, values as high as 60 - 100 mgms per 24 hours may be obtained. The high level continues after labour, following which there is a rapid fall to negligible amounts within 72 hours. The conversion of estrone to estriol is facilitated by the action of progesterone which also acts to prevent the catabolism of estrogen compounds. An imbalance of estrogen may occur during pregnancy as a result of progesterone deficiency.

5. The Placenta. (15) (53)

This is an organ of unquestionable importance, for, if pregnancy occurs, the placenta elaborates at least three hormones. It is a source of extra-ovarian estrogen found in pregnancy. The high value of urinary estrogen in pregnancy following oophorectomy and this high concentration of estrogen in the placenta itself, all indicate that the placenta is a source of estrogen. The placenta is also concerned with the production of progesterone. The substances supplement the ovarian estrogen and progesterone production and thus assure an adequate supply for the successful completion of pregnancy.

Another substance formed by the placenta is a gonadotrophic hormone which differs from the pituitary gonadotrophins, but resembles I.C.S.H. in its biological actions.

Chorionic Gonadotrophic Hormone. (C.G.H.)⁽⁹⁰⁾

The curve of excretion is characterised by a peak between the 20th and 50th day after the missed period, and a marked fall to a low level and a maintenance of this level up to the time of delivery. C.G.H. has been detected in the serum as early as the 12th post ovulatory day. The level rises rapidly from approximately 2 I.U. per mil. at the time of the expected menses to peak levels ranging from 400-700 I.U. per mil. between the 7th and 12th weeks of gestation. Occasionally, an unusually high concentration is maintained for a variable period of time, after which there is a sharp fall until a low level between 10 I.U. and 100 I.U. per mil. is reached at between the 22nd and 23rd weeks. This low level is maintained till about the 25th week of gestation when a slight secondary rise usually appears and is maintained until the termination of pregnancy. It then declines rapidly to relatively low levels which are maintained until a few days after parturition.

Chorionic gonadotrophic administration causes gross and microscopic degenerative changes in the gingivae and oral mucous membranes of women patients and Rhesus monkeys.⁽¹³⁴⁾ Grossly there is oedema inflammation and increased tendency to bleeding, which takes on the appearance of an acute necrotising gingivitis.

Microscopically there is reduction of surface keratin, cellular deterioration of the stratum spinosum and subepithelial inflammation.

Relaxin. (15) (28) (73)

During pregnancy and parturition in certain mammalian species including man, a phenomenon occurs, known as "pelvic relaxation". This consists of separation of the symphysis pubis with a consequent increase in the size of the pelvic cavity and decrease in its rigidity, facilitating parturition. Relaxin is a hormone concerned with the relaxation phenomenon, operating in conjunction with other factors.

Produced during pregnancy in tissues of the reproductive system, e.g. placenta, ovaries and uterus, the relative amounts formed in these tissues vary in different species. Progesterone and related adrenocortical steroids stimulate production. It is possibly protein or polypeptide in nature. The action of this hormone on the connective tissue of the symphysis pubis requires preceding sensitization of this structure by estrogen.

The specific effect of relaxin consists of an increased vascularity of the connective tissue of the symphysis followed by inhibition of water dissolutes and splitting of collagen fibres and disorganisation of the fibrous structure. There is apparently depolymerisation of the mucoprotein of the ground substance. There is however, not a great deal of information available at the present

time regarding this hormone; and some text books make no mention of its existence.

Baer,⁽⁵⁾ in a personal communication, states that in unpublished studies carried out at the National Institute of Health, on Rhesus Monkeys, it was found that relaxin had no effect clinically or histologically on any of the oral tissues.

There is a wide variation in the biological reactions of connective tissues. This can be deduced from the changes in mechanical or viscous elastic properties that are observed in injury, growth or pathological change, and especially to hormone change.

In the action of hormones on connective tissue Engel⁽³⁰⁾ says that two overlapping categories may be observed. There are hormones in which the targets appear to be chiefly the connective tissues proper. Other hormones have as their "obvious" targets certain epithelial cells or tissues. Thus estrogens not only stimulate genital tract epithelium in the female; but also sometimes the ground substance of the skin, as seen in a most striking fashion, in the skin changes in primates.

IV. CLASSIFICATION OF GINGIVAL DISTURBANCES

IN PREGNANCY.

The clinical classification of gingivitis due to pregnancy as elaborated by Ziskin and Nesse ⁽¹³⁴⁾ in 1946 is the one most frequently quoted and used. It is a modification of the classification of Zizkin, Blackberg and Stout, ⁽¹³³⁾ published in 1933. They ⁽¹³⁴⁾ state that the classification represents the progressive changes in pregnancy, and therefore more than one type may be present in the same case.

They could not find any correlation between the microscopic changes in the five clinical classifications, since these changes appeared to be essentially the same in all classes of pregnancy gingivitis.

Class 1.

This is by far the most common and is characterised by bleeding of the gums. On questioning the patient, it may be ascertained that the tendency for the gums to bleed when traumatised by the tooth brush, or food, etc. developed after the onset of pregnancy. Or, if the symptoms were present before pregnancy, it became exaggerated after conception. Haemorrhage is also easily induced with probing.

Class 2.

The next change is a slight alteration in the interdental papillae. The papilla loses its stippled appearance, becoming

glossy or glossy looking. The colour changes from pink to old rose. There is some oedema creating a mildly puffy appearance. Because of the swelling the papilla no longer maintains the sharply pointed style of a pyramid, but becomes blunted at the top. The symptoms are seen easily and if uncomplicated by local conditions may persist in an exaggerated state, throughout the term. After parturition the gums generally return to normal without treatment. It should be pointed out that the changes just described may be so slight as to be easily overlooked unless careful examination is made. On the other hand, they may be gross enough to be readily discernible.

Class 3.

In the third type of pregnancy gingivitis usually only the free gum margin is involved. It takes on the colour and appearance of a raspberry. The highly inflamed tissue bleeds easily on being probed. Its most frequent site is the anterior region although it may be found in other locations. This form is designated "Raspberry" red gums.

Class 4.

(a). The fourth type, a generalised hypertrophy of the tissues is designated hypertrophic gingivitis of pregnancy. The interdental papillae become noticeably enlarged, losing their natural contours. The colour may vary from an old rose to a deep cyanotic hue. In the presence of an irritant, the borders may turn

bright red. The amount of involvement varies. One, a few or many papillae may be involved in one or more sections of the mouth. The tumefaction of the individual papilla is gross in some and slight in others.

Class 4.

(b). The enlargement of the papillae is due in part to oedema and also to hyperplasia of the epithelium. Occasionally a proliferation of the tissue takes place. As it grows, the normal gum is pushed back and a straight blanched line of demarcation results. In some cases, the proliferative tissue which emerges from the under-surface of the papillae may grow over and cover in part the crown portion of the tooth. There is then created a favourable nidus for mouth sordes to collect and act as an irritant, with additional proliferation resulting. Other traumatic conditions such as food impactions, the presence of calculus, overhanging margins of restorations, sharp carious cavity margins, and lack of function in a restricted area further complicate the pathologic process. However, sometimes no irritants whatever are apparent.

Class 4.

(c). Occasionally several of the interdental papillae are enlarged so as to resemble tumours. As the individual papilla spreads laterally, the attachment of the gingiva becomes narrowed, giving a pedunculated appearance. But even in this distorted stage, these overgrowths must still be classed as indicative of hypertrophic

gingivitis, since they respond to treatment for this group rather than for epulis.

Class 4.

(d). In other cases the proliferation starts from the under surface of the free gum margin and spreads along the gingival crevice, covering the tooth for a few millimetres, without involving the interdental papilla.

Class 4.

(e). Another type in the same category which occurs less frequently, is the formation of a pseudopapilla. The interdental papilla may not be enlarged and the pseudopapilla may be the same colour as the rest of the gingivae. However, a definite horizontal line of separation forming a cleft, can be seen at the base of the papilla. If a flat blunt blade is placed in this line, the growth may be simply lifted from the normal tissue, and with little effort it can be removed. In all cases of hypertrophic gingivitis of pregnancy the gums bleed easily on being probed, but are seldom painful.

Class 5.

Pregnancy tumour. It is mostly confined to a single growth springing up at any point in the mouth. At the outset it appears to be an overgrown papilla. It usually is sessile or pedunculated and is cyanotic with a bright red border.

Maier and Orban (69) who carried out an investigation on 530 pregnant women divided the patients into the following:

1. No pathology with normal gingiva
2. Mild inflammation
3. Moderate inflammation
4. Severe inflammation
5. Tumour formation.

No difficulty was encountered in classifying the normal gingiva, severe inflammation and tumour formation groups. However, clinical distinction between the mild inflammation and moderate inflammation groups became more involved.

Hilming, (55) in his study of 203 pregnant women, classified the occurrence of gingivitis into 5 different degrees:

1. Gingivitis levi gradu:- Slight oedema and possibly slight cyanosis, not extended to the entire gingiva and at the most a few spots may be slightly hyperaemic or hypertrophic.
2. Gingivitis levi-medio gradu:- More oedema and more extensive, otherwise the gingivitis is of a mild character.
3. Gingivitis medio gradu:- Pronounced oedema, distinct hyperaemia or cyanosis in large parts of the gingiva, frequently excessive hyperaemia in small areas (Ziskin's "raspberry red gums") and more pronounced hypertrophy.
4. Gingivitis medio-magno gradu:- A severe gingivitis with

all symptoms pronounced and extended to the entire gingiva. If severe hyperaemia and hypertrophy are present, the condition is not necessarily found in the entire gingiva, but may involve large areas.

5. **Gingivitis magno gradu:-** A very severe gingivitis, highly developed hyperaemia or cyanosis, marked hypertrophy and extended to the entire gingiva.

As Hilming⁽⁵⁵⁾ points out, it is impossible to have accurate criteria for a pregnancy gingivitis beforehand, and, as in the literature it is stressed that a pregnancy gingivitis disappears or is ameliorated after parturition, he has divided his patients into two groups: 1) specific, and 2) non-specific, according to the above criteria. Those cases of gingivitis in the group showing improvement post partum from their last examination during pregnancy are called Specific, whilst the remainder which displayed no improvement are called Non-Specific pregnancy gingivitis.

V. PREVALENCE OF GINGIVAL DISTURBANCES

IN PREGNANCY.

The prevalence of gingival disease in pregnancy varies with every author who is prepared to give figures.⁽⁴¹⁾ This can be caused in part by a varying criteria upon which the workers base their classification.

Ziskin, Blackberg and Stout⁽¹³³⁾ report that 158 or 37.9% of the pregnant women in the group of 416 showed some form of observable gingivitis gravidarum. Of these, 111 or 70.2% had the hypertrophic type, 41.1% showed raspberry red gums, and 1.8% pregnancy tumours. These figures have unfortunately been misquoted many times by many authors, and Ziskin himself in the work with Nesse⁽¹³⁴⁾ in 1946, however in an article in 1938,⁽¹²⁷⁾ when referring to the original report, gives the incidence of gingivitis in pregnancy as 70%. Glickman⁽³⁹⁾ though, quotes Ziskin by stating hypertrophic gingivitis 0.7% and raspberry red gum 40% and combination 1.8% and does not mention pregnancy tumours. These could be all typographical errors, but it does leave the reader in a state of not knowing what are the right figures.

Hilming,⁽⁵⁵⁾ in a survey of 203 pregnant women, found that 100% of them had gingivitis in some form and 47% had a definite specific form of gingivitis gravidarum and 53% a non-specific gingivitis gravidarum. The latter referred to a gingivitis which was present during the pregnancy, but not due to it, and which

was not ameliorated by the termination of the period of gestation. As he points out, it is impossible to have accurate criteria for a pregnancy gingivitis beforehand, and it is logical to expect that a gingivitis which is causally connected with pregnancy would be ameliorated after delivery. This is the basis of his determining whether the patient had a gingivitis gravidarum, or not.

Maier and Orban,⁽⁶⁹⁾ in a study of 530 pregnant women from the 3rd to the 9th months of pregnancy; divided the patients into the following groups:

Group 1.	No pathology	236	or	44.6%
Group 2.	Mild inflammation	190	or	35.9%
Group 3.	Moderate inflammation	93	or	17.5%
Group 4.	Severe inflammation	8	or	1.5%
Group 5.	Tumour formation	3	or	0.5%

They state that it seems evident that there is no significant difference in the incidence of gingivitis in pregnant and non-pregnant individuals. In comparing the clinical picture of gingivitis in pregnancy with gingivitis in non-pregnant women or in men, we have to recognise that, in a single case, it would be almost impossible to distinguish between a gingivitis in pregnancy and a simple gingivitis. They also find that there are some clinical features which at least point to an endocrine imbalance. Such features are:

1. The localisation of the inflammation to a few gingival papillae which show a rather sharp demarcation from the attached

gingivae. The line of separation between the swollen and inflamed red papillae, and the rather pale pink and stippled attached gingiva is an important symptom.

2. The fringed edges of the inflamed marginal gingiva.
3. Hyperplasia of the papillae, tending toward tumour formation.

However, one cannot attach too much importance to this observation, as these three conditions may also be found where there is no apparent endocrine imbalance.

Looby,⁽⁶⁶⁾ in 475 primiparae observed a slight gingivitis in 40%, hypertrophic gingivitis in 10%, and pregnancy tumours in 2% of the total.

Gonzales⁽⁴⁴⁾ examined 379 pregnant women - 71.5% of these had gingivitis. He classified them thus:-

Erythematous	36.4%
Hypertrophic	13.5%
Ulcerative	2.1%
Gravidic	19.5%

Mugnier⁽⁷⁸⁾ examined 2,000 pregnant women admitted to the neonatal clinics of the combined hospitals of Paris. He states that all pregnant women present some structural change in the buccal mucosa but not all have a pregnancy gingivitis.

Schour⁽⁹⁹⁾ cites Freund, Pinard, and Schmidt who found gingivitis in 53-60% of pregnant women, while control studies in non-pregnant women of the same group showed only 15-18%.

Fraser⁽³⁶⁾ in South Africa, investigating 427 pregnant

women found 54% had some form of gingivitis, and of this number with gingivitis, 11% were very severe, and 88% were mild to bad. he did find, however, that of those patients who were seen at one particular clinic, and who were from the economic strata termed the "subeconomic" group, there were 73% of them with gingivitis.

Gridley⁽⁴⁹⁾ carried out a survey of 1,002 gravid women in Egypt. These were mostly of poor and lower middle class aged between 17 and 38 years, in their fourth to ninth month. He classified them as:-

N - normal gingivitis

G - common gingivitis without hyperplastic signs

H - hyperplastic gingivitis

T - pregnancy tumour

The latter two conditions were considered as definitely due to pregnancy. Pregnancy could not be blamed for causing gingivitis without hyperplasia, although it might be an early stage of gingivitis gravidarum or pregnancy tumour. He found:

14% had normal gingiva

60% were inflamed without hyperplasia.

23% showed definite hyperplastic signs. (Amongst this group 30 cases, or 30% showed accentuated hyperplasia.)

2.7% or 27 cases showed pregnancy tumours.

These figures for pregnancy tumours are much higher than in other

reports, but he did find that some of these occurred at the upper right lateral incisor region, where gold crowns are inserted as ornaments amongst the lower classes in Egypt.

Wilson and Skinner⁽¹²¹⁾ examined 321 pregnant women and 38 women seen one month post partum. A group of 80 non-pregnant women were also examined. These figures included edentulous patients.

The gingival conditions were classified as:-

- Class 1. Normal
- Class 2. Mild gingivitis
- Class 3. Moderate gingivitis
- Class 4. Severe gingivitis

Comparing the incidence of gingivitis in pregnant and non-pregnant control groups (Europeans only) they summarised:

	<u>Pregnant</u>	<u>Non-Pregnant</u>
No. of Cases	174	50
Class 1.	50%	76%
Class 2.	37%	16%
Class 3.	8%	8%
Class 4.	5%	nil

The difference between the figures for Class 1. cases they calculated, was significant. They found no cases of pregnancy tumours in this group.

Rohackova and Ticha⁽⁹³⁾ in Czechoslovakia, in examining 187 pregnant women, found about 80%, temporary genuine hypertrophy of

the gingival tissues. In the control group of non-pregnant women, different types of gingivitis, with symptoms less severe than those of the pregnant group, occurred only in 25%. In 1.8% of patients with pregnancy gingivitis, pregnancy tumours were observed, yet they state that they did not regress after parturition and were thus surgically removed.

Ringsdorf, Powell, Knight and Cheraskin,⁽⁹¹⁾ in Alabama, examined 366 pregnant and post partum patients. Of these, 330 or 90.2% were pregnant, and 36 were post partum. They summarised their results by stating that a definite relationship between pregnancy and gingival bleeding could not be demonstrated. In other words, pregnant and post partum patients showed the same frequency of this particular finding.

Colby and Kerr⁽¹⁶⁾ claim that pregnancy gingivitis occurs only in about 5% of pregnant women, and pregnancy tumours occur in 1-2% of patients who have pregnancy gingivitis.

Erb and Brzezinsky⁽³¹⁾ in Israel, in a survey to compare the incidence of gingivitis during pregnancy in native born Israeli and immigrant women from East Africa and Asia, gives a classification of:

1. Mild gingivitis
2. Moderate gingivitis
3. Severe gingivitis
4. Pregnancy tumours

One hundred and forty-six pregnant and 123 non-pregnant women were examined. 70.5% of the 146 pregnant women showed gingival disorders of varying degrees. 60.3% of the 53 Israeli born pregnant women were affected against 76.3% of the 93 new immigrants. They found that the difference between the pregnant and the non-pregnant group was statistically highly significant as only 37.9% of the non-pregnant group had gingivitis.

Cahn, ⁽¹⁴⁾ however, states categorically, that he does not believe that there is a specific disease entity as pregnancy gingivitis or diabetic gingivitis, as there are thousands of diabetics who never develop pyorrhoea, and a vastly greater number of women who become pregnant without ever having gingivitis.

Tiilila, ⁽¹¹¹⁾ in carrying out a study to discover cases of epulis had 453 pregnant women examined in Helsinki, and gives the following figures:

Healthy gums	312 cases	68.9%
Slight inflammation	124 cases	27.4%
Slight swelling of gingival papillae	14 cases	3.1%
Intense local hyperplasia	2 cases	0.4%
Epulis	1 case	0.2%

As the examinations were performed by a midwife who was not accustomed to evaluating the condition of the gingivae, and as attention was not directed to each gingival papilla separately, it is admitted

that many slight gingival inflammations were overlooked, and the percentage of healthy gums was thus actually much smaller.

Tammoscheit,⁽¹⁰⁷⁾ after examining 1,000 pregnant women, found normal gingiva in 450 non-specific gingivitis in 379, hyperplastic gingivitis in 159, and gingival tumours in 12. He does not agree with the term "gingivitis gravidarum" but feels that the proper classification is "gingival hyperplasia". Whilst many of the changes are of a hyperplastic nature, there are still others which are purely inflammatory, and from a study of the literature, I cannot agree with him.

As can be seen, there are conflicting thoughts as to whether the condition even exists, and amongst those who maintain its existence there is a variety of opinions as to its extent. It must be realised that amongst those who deny the existence of the condition, there are many who have not carried out their own statistical survey. Even Maier and Orban,⁽⁶⁹⁾ who claim that there is no significant difference in the prevalence of gingivitis in pregnant and non-pregnant individuals, recognise a condition and describe it, qualifying their findings by calling it an inflammatory condition of local irritative origin.

Until such times as diagnosis of gingivitis gravidarum relies less on the opinion of the examiner and more upon standardised scientific scoring the divergent results will continue.

VI. EPULIS GRAVIDARUM or PREGNANCY TUMOUR

The term epulis goes back to antiquity, and was used at that time at least, by Galen. It literally means - upon the gum. The name neither indicates the character nor the origin of the epulis. Epulides are far more frequent in women⁽¹¹¹⁾ than men and occur in women during pregnancy in particular. Conflicting views have been expressed on the epulides which develop during pregnancy and either disappear spontaneously, or at least diminish markedly after delivery. Thoma⁽¹⁰⁸⁾ does not believe that they do disappear spontaneously but to the contrary may develop to a large size if allowed to remain.

The term pregnancy tumour, adopted by Blum⁽¹¹⁾ in 1931, has been most widely used. However, it has been considered misleading, since the growth is not a tumour in the true sense of the word, nor does it indicate where the formation is growing. Others favour the term granuloma gravidarum.⁽⁸⁷⁾ Yet the most widely held opinion seems to be that this tumour-like formation appearing during pregnancy is only a local intensification of gingivitis gravidarum.⁽²⁸⁾⁽³⁹⁾⁽⁴⁹⁾⁽⁶⁹⁾⁽¹³⁴⁾⁽¹⁰⁸⁾ Gridley⁽⁴⁹⁾ suggested the term "pregulis", whereas Hilming,⁽⁵⁵⁾ on studying 203 pregnant women, says that the whole concept should be discarded due to the fact that not one case was observed in his group. Whilst other authors⁽⁶⁰⁾ consider granuloma pyogenicum and epulis gravidarum identical, the true epulis gravidarum regularly disappears after delivery, but the granuloma pyogenicum never effects a spontaneous cure. Tiilila⁽¹¹¹⁾ investigated 78 cases of epulis gravidarum and found that a high enough percentage had such features

in common as permitted their classification into a separate group called: "typical epulis gravidarum", in contradistinction to atypical epulis gravidarum. The typical epulis gravidarum is a mostly pedunculated, fairly soft tumour with an interdental attachment. It is darker in colour than the surrounding tissue and its surface is often ulcerated. Frequently they are multiple.⁽¹¹²⁾ Apart from bleeding, it may not present any symptoms. As the epulis increases in size, however, it may grow beyond the dental arch, and be traumatised by the opposing teeth, which, naturally, gives rise to pain. Pain may also be caused by food remaining under its margin,⁽³⁹⁾ or by the epulis interfering with the occlusion, in which case painful inflammatory changes may occur. In most cases it appears during the first pregnancy, disappears or decreases markedly in size post partum, and unless it is radically removed, reappears in the next pregnancy. It may grow up to a relatively large size - up to 2 cm.⁽⁹⁾ and a few incidents have been noted of an even larger tumour. Thoma⁽¹⁰⁸⁾⁽¹⁰⁹⁾ describes the occurrence of Sturge-Kalischer-Weber Syndrome with a pregnancy tumour. They occur infrequently enough to warrant reporting in various journals.⁽¹⁰⁾⁽²⁰⁾⁽³³⁾⁽⁵¹⁾⁽¹¹³⁾⁽¹²⁴⁾

Allen⁽¹⁾ describes one superimposed upon a naevus, and states that this is not an isolated case, that naevi are predisposed to tumour formation during gestation. Hirschfeld⁽⁵⁷⁾ describes a tumour with a daughter one growing from it, and a grand-daughter

growing from that. Evaluation of the incidence of it is rendered difficult by the fact that different authors refer to epulis gravidarum by different names. Monash⁽⁷⁷⁾ gives the percentage as 5% but some of the cases were gingival hypertrophies. Other authorities list their findings as follows:-

Ziskin, Blackberg and Stout	(133)	0.7%
Maier and Orban	(69)	0.5%
Hilming	(55)	0.
Gridly	(49)	2.7%
Rohackova and Ticha	(93)	1.4%
Tiilila	(111)	0.2%

The incidence figures given by different authors for epulis vary widely. As they are based on case material of varying character, and in some cases, without complete data, the results are not comparable as often the cases are too few for definite conclusions to be drawn regarding its incidence.

The site of the tumour may be predetermined by some previous irritation or infection involving the interdental papilla. Gridly⁽⁴⁹⁾ states that mouth breathing or enthusiastic tooth brushing of the anterior region of the mouth accounts for the preponderance of the lesions in that area. He also found that the upper right lateral incisor was a common site for these lesions, as in Egypt, where his survey was carried out, gold crowns are inserted on these teeth as ornaments among the lower classes.

Tiilila⁽¹¹¹⁾ found that growth seemed to be more rapid in the case of tumours appearing late, rather than early in the pregnancy. Two thirds of the cases were located in the maxilla, 54% in the anterior region, 46% on the buccal side, 20% on the lingual side and 34% simultaneously on both sides. The tumour was invariably a solitary formation but the histological picture varies in the typical epulis gravidarum, depending on the different stages of development. Different stages may occur side by side in the same tumour.

Glickman⁽³⁹⁾ is emphatic that these are not neoplasms but are inflammatory lesions of the gingiva which differ in clinical appearance from the usual inflammatory enlargement because it is the result of microscopic response to local irritating factors modified by the condition of the patient. Tiilila⁽¹¹¹⁾ supports the opinion that it is not a neoplasm proper, but a pregnancy linked vascular tumour of hormonal origin with some other possible factor involved. The therapy recommended ranges from immediate and complete surgical removal, during pregnancy, to waiting till after parturition, to see if it regresses. Glickman⁽³⁹⁾ advocates the preventive aspect as he states that it is only an exaggerated response to local irritation. He maintains that surgical treatment is contra-indicated unless it is impossible to relieve the patient's discomfort by any other means. Coolidge⁽¹⁸⁾ also stresses the importance of local irritation as the main cause. Hirschfeld⁽⁵⁶⁾ reports cases of tumours that have returned more

than once within a matter of days after removal - the new tumour being larger than the previous one. He therefore believes that they should be left, particularly as they soon decrease in bulk after parturition, and mostly disappear. Monash⁽⁷⁷⁾ does not believe in surgery unless it is urgent and interferes with the comfort or masticating ability of the patient. Blake and Trott⁽⁹⁾ believe surgery should always be avoided if possible until after parturition, when, if the lesion does not completely regress, it can be more conveniently excised without risk of recurrence. Blum⁽¹¹⁾ states that they should be removed surgically even though they disappear at times. Burket⁽¹²⁾ recommends surgical removal and cauterization if there is a haemorrhage and ulceration. Urban and Wentz,⁽⁸⁰⁾ as well as Archer⁽⁴⁾ claim that pregnancy is not a contraindication to surgical therapy. Cross⁽²¹⁾ advocates large doses of ascorbic acid in conjunction with thorough prophylaxis. Archer⁽⁴⁾ advises surgical excision with removal of any irritating causes such as ill fitting crowns or calculus, and cauterization of the base, to prevent its recurrence. He does not believe that these lesions disappear with the termination of pregnancy.

Sarkany⁽⁹⁷⁾ describes a malignant tumour which grew to the size of a grape-fruit, from a pregnancy tumour, necessitating the resection of the mandible. He feels that the transformation into a malignant neoplasm was brought about by the pregnancy changes. These changes act as stimulants for tissue proliferation in inflammation as well as in tumours. The general opinions held of the effects of pregnancy

on tumour formation, he believes, need revising to some degree, and all pregnancy tumours, because they may become malignant, should be excised as radically and as soon as possible. If after radical surgery, there is recurrence, interruption of pregnancy is indicated. This extreme opinion does not appear to be held by any other writer.

Ward and Hendrick⁽¹¹⁸⁾ do not think they are of great practical significance, unless they bleed freely, when they should be removed. They describe the case of a large birthmark which involved the lip, buccal mucous membrane and skin of the cheek. During each of several pregnancies, this haemangioma took on rapid growth which included soft nodular development and which receded after pregnancy.

Fabe⁽³³⁾ describes a tumour associated with an old naevus which, after seven months post partum still did not recede spontaneously. Amies⁽²⁾ is another who does not believe that pregnancy tumours disappear after pregnancy, but agrees that they may regress.

The pertinent literature on epulis gravidarum still fails to clarify the etiology of this condition. Many questions remain unresolved: should the epulis be removed immediately or should one wait until the termination of pregnancy? If it is to be removed immediately, should it be radical to prevent its recurrence? If it reappears with subsequent pregnancies - why? Is there always an irritant present at the site as Gridley⁽⁴⁹⁾ observed? Should

epulis gravidarum be classified as a final development of gingivitis gravidarum or as a true neoplasm as suggested by Amies ⁽²⁾ or as a pregnancy linked vascular tumour as suggested by Tiilila ⁽¹¹¹⁾?

These are some of the conflicting problems still to be resolved in this enigmatic condition.

VII. HISTOPATHOLOGY and HISTOCHEMISTRYof GINGIVITIS GRAVIDARUM.Histopathology.

The histopathology of gingivitis in pregnancy presents a problem very much like the clinical one. Although there are changes, some of which are striking, there is no single histological feature in the picture, which would make the diagnosis of pregnancy a certainty.⁽⁶⁹⁾ Ziskin, Blackberg and Stout,⁽¹³³⁾ and Ziskin and Nesse⁽¹³⁴⁾ have shown that the most apparent changes were the loss of surface keratin, which became more marked as pregnancy advanced from the fourth to the ninth months. In most cases, however, slight keratinisation reappeared in the ninth month. This has been observed by others.

Although Glickman⁽³⁹⁾ is guarded in his statement that the epithelium is, in most cases, keratinised, Ziskin, Blackberg, and Stout⁽¹³³⁾ also noticed that, in the early months, a slight hydropic change of the corneum occurred. Ziskin and Nesse⁽¹³⁴⁾ found in some cases a zone of parakeratosis varying in depth up to 4-5 cells. The former found the stratum granulosum to be generally hypoplastic, varying from a lesser change during the early stages to a more marked hypoplasia in the last months. The changes in this stratum did not take place in as orderly a fashion as in the cornified layer. Hydropic changes in the stratum granulosum was confined in the early months mostly to the superficial parts

whereas, in the last months, the entire stratum was involved. Ziskin and Nesse found that there was extensive hydropic change in the stratum spinosum with pyknotic degeneration of the nuclei towards the surface. There was an acanthosis present, as well as hyperplasia of the stratum germinativum. The peg patterns showed a tendency towards elongation and penetration deeply into the lamina propria and they frequently split. No increase in mitotic figures were seen. Pearl formation was seen in some cases. Blood vessels showed dilatation in the lamina propria. The amount of collagen and connective tissue varied in the different cases so that no definite conclusions could be drawn. There was a heavy cellular inflammatory reaction which included plasma cells, lymphocytes, leucocytes and occasional eosinophiles. In most instances the infiltration was seen, mainly near the crest of the interdental papilla, but occasionally it was distant from the areas commonly recognised as subject to irritation. There were signs of oedema in the lamina propria and epithelium.

Colby, Kerr and Robinson⁽¹⁶⁾ state that the marked vascularity of the tissues is the most characteristic feature and that the lesion resembles a granuloma pyogenicum. The excessive vascularity accounts for its bright red colour and the hyperaemic oedema for the enlargement. Glickman⁽³⁹⁾ describes both marginal and discrete enlargement as a circumscribed mass of connective tissue outlined by stratified squamous epithelium. The connective tissue consists for the most part, of numerous

diffusely arranged newly formed engorged capillaries, lined by cuboidal endothelial proliferation, with capillary formation and associated inflammation. This is a modification of the usual gingival response to chronic irritation and is responsible for the unique clinical lesion. On the basis of the microscopic findings, a gingival enlargement in pregnancy may be considered an angiogranuloma. This term avoids the neoplastic implication of such terms as fibrohaemangioma, or pregnancy tumour.

Ramfjord⁽⁸⁷⁾ says that acute lesions of gingivitis gravidarum and granuloma gravidarum are characterised by marked endothelial proliferation, ulceration and inflammation, commonly pyogenic in type. In small, early lesions the dominating endothelial component can only be recognised under high magnification. Older and more extensive lesions show marked vascularity as the vessels are quite wide with thin walls.

Maier and Orban⁽⁶⁹⁾ state that the proliferative character of epithelium, endothelium and connective tissue is probably the most characteristic feature of gingivitis in pregnancy.

Although epulis gravidarum has been discussed under a separate section, it is under Ziskin's classification, a further stage of gingivitis gravidarum.

In describing a typical epulis, Tiilila⁽¹¹¹⁾ states the histological picture varies considerably. There are divergencies from case to case, and even in the same case the picture is not

the same throughout. Dissimilar areas may occur side by side. Three stages of development are described, each having its own basic features. They are: -

1. Stage of proliferation
2. Stage of maturity
3. Stage of regression.

The division rests chiefly on the vascular characteristics at each phase, the crucial component at the proliferative stage consists of the proliferating angioblasts of developing and small capillaries. The stage of maturity is characterised by large thin-walled blood vessels. During regression, the vessels become smaller, often flattened, partly obliterated or disappear. The regressive stage is the only one in which there may be a predominately uniform picture. No proliferation state is encountered exclusively, because maturation occurs very rapidly. In most mature cases, there are already distinct regressive changes but usually areas also which show definite proliferation.

Maier and Orban⁽⁶⁹⁾ agree with Hirschfeld,⁽⁵⁷⁾ Merritt⁽⁷⁴⁾ and Monash⁽⁷⁷⁾ who consider that tumour formation is only an accentuation of the inflammatory reaction of the gingiva in pregnancy, and who maintain that the capillary proliferation which is considered a characteristic feature of pregnancy tumours would, in a given case, offer great difficulties in a differential diagnosis between a pregnancy tumour and pyogenic granuloma. Tiilila⁽¹¹¹⁾

points out that whilst these two have many features in common, they also may present features which tend to differentiate them, one from another. In epulis gravidarum there are always large lacunar vessels, whilst in a granular pyogenicum there are usually quite small vessels. In both, the vessels are thin-walled, but in granuloma pyogenicum they are generally "cuff like" without supporting stroma. The stroma in an epulis gravidarum is much richer in cells, especially angioblasts. It is pointed out that each of these tumours may, in some cases, show no clearly discernibly typical feature, and then they may be histologically highly similar.

Parma, (82) in examining the lower gums, the lower lip, and the nasal groove, of 60 normal pregnant women, found that in 54 cases capillary changes were present in the gingiva, indicative of capillary stasis or congestion. It was most marked near the margin and in the interdental papillae. In the latter the dilated vessels formed a seam of deep red colour. The capillary changes were greatest toward the end of pregnancy, but when they appeared earlier, it was possible to follow these changes as they intensified to a stage where rupture of a vessel wall and haemorrhage occurred. The microscopic changes were accompanied by the clinical appearance of swelling, hyperplasia and liability to bleeding. The clinical picture and the microscopic changes regressed simultaneously.

Histochemistry.

Whilst microscopic changes have contributed considerably towards clarifying the nature of gingival disease, more information is required than can be obtained from morphologic findings alone. The study of the basic physico-chemical changes involved in the maintenance of the normal gingival condition and the way in which these are altered in gingival disease is also necessary. This entails an investigation of metabolic activities in normal and diseased gingival tissues, utilising laboratory methods including chemical and histochemical studies, together with the study of enzyme extracts.

Gans, Engel, and Joseph⁽³⁸⁾ studied the gingivae electrometrically of 41 pregnant women and made post partum studies of 21 of them. A control group of fourteen non-pregnant women with normal gingivae was also studied. The gingival area between the lower left cuspid and first bicuspid was observed. These tests showed that in the early months of pregnancy, the density of negatively charged colloids including glycoproteins of the ground substance was decreased. The post partum potentials, with one exception, were higher than the prepartum ones, and approached the levels obtained in the controls. In the cases where severe gingival inflammation existed, the potential values were at, or below zero. The patients showing the greatest post partum rise in the dilution potentials were those who

showed no gingival involvement during pregnancy. They state that experimental studies have shown that hormones can produce changes in the colloidal properties of a variety of connective tissues, including the gingiva. Some hormones effect depolymerisation of the mucoprotein of the ground substance, or stimulate the connective tissue cells to produce watersoluble components leading to a redistribution of tissue, water and electrolytes. Proteins containing carbohydrate in their molecules are called glucoproteins or mucoproteins. It has been suggested that the former term be used for proteins containing less than 4% carbohydrate and the latter for proteins in which the carbohydrate part exceeds 4%, although this usage has not become universal.

Engel, ⁽²⁹⁾ in an article on water soluble mucoproteins of the gingivae, states that the ground substance of the connective tissue is conceived to exist largely as a highly aggregated structure which is highly plastic in a biologic sense. Its chemical and physical behaviour is thus readily modified in physiologic and pathologic states. The lability of ground substance may account in part for the response of some of these connective tissue changes in gingival tissue.

When the gingivae are inflamed, the ground substance is more lightly stained. The basement membrane about blood vessels and that adjacent to the epithelial layer is changed in inflammation. It may be swollen, or more usually attenuated or

thinned. Many of the connective tissue cells contain increased amounts of cytoplasm, glycoprotein as well as glycogen. The soluble-insoluble portions of the connective tissue ground substance may be considered as co-existing phases of a colloid. The soluble part would correspond to a water-rich, colloid-poor phase which is in equilibrium with the insoluble colloid-rich water-poor phase. Under physiologic conditions, the relative quantities of the two phases might fluctuate, but the basic composition of each phase is thought to remain constant. This type of equilibrium would permit an intake or release of water and of electrolytes within a variable range without leading to alteration in osmotic pressure of the blood or extracellular ground substance. In gingival disease there is swelling and oedema. The increased quantity of water soluble mucoprotein would correspond to an increase in a water-rich, colloid-poor faction. Similar increases in water soluble mucoproteins of the connective tissue have been demonstrated about growing organs and tumours.

In gingival inflammation associated with hormonal changes, as during pregnancy or puberty, the tissue responses, the increase in the water soluble mucoprotein might be attributed to depolymerisation of ground substance colloid, or synthesis of increased amounts of water soluble mucoprotein, or both.

Turesky, Fisher and Glickman,⁽¹¹⁴⁾ in a histochemical study of the attached gingiva in pregnancy, point out that, whilst

many agree that increased amount of water-soluble, alcohol-insoluble glycoproteins observed in gingivitis of pregnancy have been attributed to hormonal origin, and that as comparable results have been associated with gingival inflammation in non-pregnant women by Turesky, Glickman, and Litvin,⁽¹¹⁵⁾ also by Dewar⁽²⁷⁾ they felt they should study the gingivae of pregnant women without the complicating factor of inflammation. They obtained 60 gingival biopsy specimens from 30 pregnant patients aged 16-30 years, and all in their second or third trimester. They all had discernibly normal gingivae or slight to moderate marginal gingivitis. Similar control gingival biopsy specimens were obtained from 21 non-pregnant patients who had normal or slightly inflamed gingivae. The major features of the uninflamed attached gingiva in pregnancy were a change in epithelial configuration, characterised by a relative lengthening of the epithelial ridges and associated changes in the underlying connective tissue. The latter consisted of thinning of the basement membrane and a reduction in glycogen in the carbohydrate protein complexes of the ground substance. The change in epithelial configuration was accompanied by diminished surface keratinisation. There was also an increased tendency toward the occurrence of glycogen in the epithelium. An increase in glycogen has been described, associated with diminished keratinisation in the skin of pregnant women. They state that whilst in the present study, the alteration in epithelial configuration

and diminished surface keratinisation may be due to pregnancy, glycogen does tend to occur associated with such epithelial changes in the absence of pregnancy. Their findings were that the morphologic and histochemical criteria employed in this study revealed no significant difference between the pregnant and non-pregnant groups. This indicates that their findings are in conflict with the impression that pregnancy modifies the response of the gingiva to local irritation. The authors recognise that there may be significant differences in patients who develop gingival enlargements and pregnancy tumours which were not evident in these women with relatively normal gingiva. The connective tissues of the gingiva during pregnancy, when studied electrometrically, are found to have a colloidal charge density about one third lower than the normal level, and it is not restored until some months after parturition.

VIII. ETIOLOGY OF GINGIVITIS GRAVIDARUM

The cause of gingival changes in pregnancy, has long been one of great discussion, and as yet, has not been proven to the satisfaction of all. The controversy arises as to whether the gingivitis comes from within the oral cavity itself - due to purely local causes, or whether systemic factors have brought it about. The gingivitis may thus be considered as being primarily an oral disease, or may be a secondary manifestation of some other condition. The periodontium is classical as the site of disease by multiple etiological agents. Local etiological factors are those in the immediate environment of the teeth and supporting structures. Systemic causative factors refer to systemic conditions of the patient that influence the metabolism of the periodontium. Although Cahn⁽¹⁴⁾ does not believe that the condition of Gingivitis gravidarum even exists, there are many who, whilst agreeing that there is such a thing as an increase in gingivitis during pregnancy, feel that the pregnancy is only a secondary cause.

(127)(128)(129)(130)(131)(132)(133)(134)(135)(136)
 Ziskin and his co-workers, have published many articles supporting the theory of a gingivitis due to pregnancy and caused by hormonal changes. Ziskin, Blackberg and Stout⁽¹³³⁾ say that it may be accompanied by a subacute or chronic Vincent's infection. Admittedly, this can occur in any person, but no cases were observed in the group examined by me. They state

that while irritants are usually associated with gingivitis in pregnant women, this affection may develop in the absence of such irritants. They believe that irritation is a complicating factor, rather than the causative one, also that as hyperplasia is a most notable change in the epithelium because it increases with advance in pregnancy, and because it is also commonly present in certain organs of reproduction during this state. They thought it probable that pregnancy played an etiological role in the production of epithelial hyperplasia. They could not link the month of pregnancy with the inflammatory changes in the submucosa, so felt that these changes were secondary in character.

A loss of surface keratin in the oral mucous membrane is characteristic of this condition as well as changes in the vaginal epithelium, which shows hypertrophy and hyperplasia. The nasal mucosa is another epithelial surface which shows characteristic changes during pregnancy. As gestation progresses, the colour of the nasal mucosa becomes increasingly red, and by the eighth month it is very red and swollen. The changes in pregnancy, occurring in the mouth, are not specific for the oral mucosa, but occur also at other sites.

The reduced keratinisation could be caused by a change in hormonal balance resulting in a subnormal estrogen effect on the tissues. Ziskin and Blackberg⁽¹³¹⁾ have shown in castration

of female Rhesus monkeys that a lack of estrogen leads to degenerative changes in the gingival tissues and mucosa, and that castration of male Rhesus monkeys causes changes in the gingivae and mucous membranes of the mouth, resembling the effects of injections with estrogenic hormones, namely hyperkeratinisation.

Ziskin and Blackberg⁽¹³⁰⁾ in an earlier paper also showed after injecting sex hormones into monkeys, that whilst irritants often occur in gingivitis gravidarum, this affection is also present when irritants are absent. Irritation, they feel, is a complicating factor rather than a cause.

Ziskin and Nesse⁽¹³⁴⁾ claim that the amount of progesterone found in pregnancy would also tend to alter the estrogen effect, since progesterone has a sparing action on estrogens, reducing catabolism and utilisation. If the decrease in vaginal cornification during pregnancy does not occur, abortion or miscarriage may result. If, however, when progesterone is administered in an attempt to prevent miscarriage, the vaginal cornification is reduced. This could be due to its modifying estrogenic activity.

Schour⁽⁹⁹⁾ notes that there is a higher incidence of gingivitis during pregnancy and that it is characterised by a hyperaemic oedematous swelling of the gum tissue, of endocrine and vasomotor origin.

Mugnier⁽⁷⁸⁾ says all pregnant women present some structural

change in the buccal mucosa, but not all have gingivitis Gravidarum. The latter appears toward the third month, and is characterised by congestion and generalised hypertrophy. This is at a time when the vaginal epithelium has at least doubled its thickness. A generalised marginal gingivitis develops which appears to reach its maximum at about the fifth month and may also increase slightly in the eighth or ninth month, when the estrogenic curve arrives at its maximum. He states that it disappears without leaving a trace, providing there was no periodontal lesion. Others feel that it can and does cause a lasting change. As it is common belief that it probably automatically disappears after parturition, this should warn one against taking gingivitis gravidarum too lightly, from a therapeutic point of view. He further believes that gingivitis is more frequent when the child is male. Moreover, he points out that much depends on the state of health of the mouth, and that most cases seen clinically are combined with some pre-existing condition. Only when dental health is in a perfect state can there be a pure gingivitis gravidarum, which can be justifiably attributed to an imbalance of hormone excretions. In these cases even the most perfect oral hygiene, he feels, will not prevent the condition.

Lighterman and Lees ⁽⁶⁴⁾ stress the hormone and vitamin factors as the cause, and place irritation as a secondary factor, because many patients with gingivitis have no observable irritation, and others who are pregnant, with periodontal irritation, show no

signs of the condition. They include other etiological factors, such as alteration of endocrine function, increased dietary demands of the body, a complex interaction of diet and hormones, periodontal irritation, and neglected oral hygiene, all acting directly on the patient and being counteracted by the patient's resistance. The non-gravid will enjoy apparent gingival health because her body's resistance exceeds the adverse factors.

Gonzales (44) recognises a pure form of pregnancy gingivitis which occurs in women with perfect oral hygiene and healthy teeth. Bernier (7) says that it is likely that most pregnant women show some gingival changes, but that in many cases it is so slight as to go unnoticed. He also states that the belief that hormonal imbalance of pregnancy does not affect the gingivae to produce enlargements, unless pre-existing clinical inflammation is present. This, however is not completely tenable. Cases have been seen where the reaction was spontaneous and occurred in an otherwise clinically normal mouth. In isolated cases, drug, chemical or food allergy could be the cause. Frank (35) does not think that the typical gingivitis gravidarum develops until the second half of pregnancy, although even in the first few months, the gingivae may show an almost microscopic proliferation, beginning at the apex of the interdental papillae. He also supports the theory that it can occur in the cleanest of mouths, in which there is no trace of calculus or other irritation. Fraser, (36) Middleton, (75) Miller, (76)

Poletti, ⁽⁸⁶⁾ and Colby and Kerr ⁽¹⁶⁾ support the idea of a hormonal disturbance as the cause with local irritating factors contributing to it.

Maier and Orban, ⁽⁶⁸⁾ ⁽⁶⁹⁾ on the other hand, after carrying out their work on five hundred and thirty pregnant women, maintained that pregnancy cannot be considered an etiological factor of gingivitis and that the gingivitis in pregnancy cannot be considered specific. They believe that one has to consider pregnancy as a conditioning factor because gingivitis in pregnancy has certain characteristic although not exclusive symptoms. They maintain that in a healthy mouth, and with proper home care, there will be no development of gingivitis in pregnancy. If, on the other hand, a gingivitis or periodontitis is present, their course will probably be modified and aggravated by the systemic condition. If patients could be observed prior to pregnancy, during gestation and after parturition, one could notice the aggravation of prior existing periodontal conditions, during the pregnancy.

Monash ⁽⁷⁷⁾ and Merritt ⁽⁷⁴⁾ believe that there is possibly no type of gingivitis that can be said to be peculiar to, or found only in pregnancy. When, however, gingivitis is present under conditions of pregnancy, especially when accompanied by oral sepsis, it occasionally assumes an hypertrophic form.

Lite, ⁽⁶⁵⁾ in a detailed study of only twenty-two pregnant patients with marginal gingivitis, thought that there appeared to be a

connection between local irritation and hormone influence which regulates the intensity of the inflammatory reaction before parturition." Fish⁽³⁴⁾ whilst recognising the role that hormones play in epithelial softening, points out that in some people, the epithelium does not keratinise as strongly as in others. Also, the connective tissue fibres are strong and densely woven in some, and delicate, fragile and loosely woven in others. He feels that a completely adequate degree of keratinisation can be maintained in any patient by careful attention to gingival stimulation, but only where the patient has never had any serious chronic marginal gingivitis before pregnancy, or in one who has had efficient treatment. Ringsdorf⁽⁹¹⁾ and his co-workers could find no definite relationship between pregnancy and gingival bleeding. They thought that there was reason to believe that gingival bleeding was multi-causal in origin, there being more than one factor operating to determine whether this particular oral condition would arise. After examining all factors in their survey they decided that the younger pregnant patient who does not brush her teeth, who smokes, does not take vitamin supplements and gives no history of citrus intake is the type of individual who is most likely to demonstrate gingival bleeding. These items, suggesting a combination of systemic and local operating elements in the possible genesis of gingivitis gravidarum. Hirschfeld,⁽⁵⁶⁾ whilst recognising a systemic disturbance, discusses the influence of the local irritants which is evidenced by the localisation of hypertrophy. In other words, the

gingival hypertrophy is a symptom of a systemic condition which expresses itself at favoured points of local irritation, no matter how comparatively infinitesimal.

Orban, Wentz, Everett and Grant⁽⁸¹⁾ place systemic effects in a secondary or modifying role. They do not believe that any systemic factor in itself, would produce a localised inflammatory process. They also emphasize the fact that no gingivitis in pregnancy will develop if there is no gingival inflammation prior to, or during pregnancy.

Glickman⁽³⁹⁾ also agrees with those who advocate local etiology as the predominant factor, and says that in the absence of local irritation, the gingiva in pregnancy presents no noticeable clinical changes. He thinks that the accuracy of the term "pregnancy gingivitis" is subject to question because the inflammatory changes are actually initiated by local irritation. He does admit, however, that it is quite common for a patient with a slight marginal gingival change which previously attracted no particular attention, to become aware of the gingivae in the course of the pregnancy, this awareness taking place about the third or fourth month. These areas have now become enlarged or oedematous and more noticeably discoloured. A patient with a chronic marginal gingivitis and a slight amount of bleeding before pregnancy will notice an increased amount of bleeding in the course of the pregnancy. He feels that the evidence that there is an increased prevalence of

gingival disease in pregnancy requires further substantiation. The impression one has of an increased prevalence of such a condition may be due to the fact that the gingival response to local irritants which normally produce slight marginal gingivitis becomes more prominent in pregnant individuals. It is emphasized that for gingival inflammation to occur, there must be some form of local irritation. Apart from modifying the response to local irritation it does not induce inflammatory changes. The suggestion is also put forward that, because of the gravid conditions, the patient becomes more lax in her oral hygiene. This then increases the local irritating factors.

Wade, (117) Sorrin, (103) Coolidge, (18) and Blake, (9) all favour the local irritant as the cause, and Blake thinks that in the healthy, well cared for, pregnancy gingivitis is unusual. Paul, (83) after a survey of 3,310 pregnant women, and a study of 224 published papers, drew the conclusion that, normal pregnancy produces body modifications which include changes in the gingiva. However, he does not give any etiology. Pregnancy may be considered a conditioning factor, claim Orban and Wentz, (80) but they maintain that pregnancy does not cause gingivitis.

Erb and Brezezinsky (31) found in their survey, (Israeli born women as against migrants) that, where there was good oral hygiene, the incidence in gum disorders was more or less the same for both groups, but where the groups had poor oral hygiene, the migrant factors exhibited more gingivitis gravidarum.

Gorvy, ⁽⁴⁵⁾ in an unusual and possibly unacceptable dissertation, on the theory of how gingivitis gravidarum could arise, stated that organs during development arise in phase, one after the other, under the control of a set morphogenetic plan. Organs which are interrelated vary in their rates of reaction. Therefore, those organ tissues which are associated with the pregnant woman's "last link" phase in the epigenetic chain of development, must be the first to escape the plan. Her gums, related to teeth, are in turn connected with such a phase, and respond with visible growth.

Ringsdorf et alia ⁽⁹¹⁾ in bringing out the point that there is some indication that the periodontal status is related to the number of teeth, thought it might be possible to show that gingival bleeding was associated with the number of teeth present. They therefore, in their examination of pregnant women, grouped them according to whether there were 25 or more versus less than 20 teeth standing. Their results showed, however, that gingival bleeding was not significantly different in the two groups.

It has been proposed ⁽¹³⁾⁽⁵⁰⁾⁽⁵⁷⁾ that the patient hesitates to use her tooth brush in the early stages of pregnancy, because of the tendency to gag, or because of a gingival haemorrhage. This idea, and the possibility of the poor oral hygiene associated with actual vomiting in pregnancy, as a cause of gingivitis gravidarum, is however, discounted by Wilson and Skinner, ⁽¹²¹⁾

Goldman and Cohen ⁽⁴²⁾ say that although in almost all instances

the gingival changes attributed to pregnancy are only an accentuation of a previous inflammatory process, occasionally an individual may be affected, who earlier exhibited no noticeable gingival alteration, prior to the pregnancy. A gingival enlargement may be observed in an area entirely free of local environmental irritation.

Hilming's (55) observation of a clinical amelioration in the ninth month in about half of the specific cases supports Ziskin and Nesse, (134) who have shown that there is a reappearance of a slight keratinisation of the epithelium in most of their cases in this month. He also observed an amelioration that takes place in this month, and that the amelioration that takes place after delivery is often very considerable. There is much to indicate that it usually sets in as early as during the first few days of the puerperium.

Lighterman and Lees, (64) however, do not find Ziskin's theory conclusive, as they feel it is too limited in its scope to be complete.

Engel (28) feels that pregnancy can induce primary changes in the gingival tissues independently of any lowering of tissue resistance of local causes. Kutscher, (61) after summarising some of the major works on the subject, feels that the etiology of gingival disturbances noted during pregnancy remains clouded.

Bohackova and Ticha (93) established to their satisfaction that pregnancy gingivitis usually begins in the second and third month,

and sometimes it does not recede until several months after delivery, the cause being related to disturbed function of certain endocrine glands.

Wiener, Karshan and Tenenbaum, ⁽¹²⁰⁾ in working on ovarian function, showed some connection between the severity of periodontal disease and the quality of the ovarian cycle.

Perl ⁽⁸⁴⁾ feels that hormonal influences are important, and that the gingival tissues, which react to hormonal stimulation, are highly aggravated by local irritation and traumatic factors. Although his article gives a brief resume of the views of other authors, it contains very little that is new and original.

Reed, ⁽⁸⁸⁾ in an article on the mouth in pregnancy, favours a dietary cause, but his article is generally over exaggerated in his statements of possible end results, and his method of overcoming it with diet.

Ryan ⁽⁹⁶⁾ reviews Ziskin and Nesse, but gives no new facts, merely confining his writings to a precis of their work.

Jenkins ⁽⁵⁸⁾ claims that Ziskin's results were not confirmed and he cites some unpublished experiments on similar lines by Jobling which show that the question of sex hormones effecting the oral mucosa must be regarded as unsettled.

Johansen ⁽⁵⁹⁾ describes an unusual case of gingival haemorrhage which occurred spontaneously towards the end of the pregnancy.

There was no obvious cause. The bleeding originated from the interdental papilla on the lingual of the lower central incisors. The haemorrhage was difficult to control, and the author feels that the only possible cause was hormonal.

Ziserman⁽¹²⁶⁾ describes the case of a patient who had recurrent ulcers of the mouth and occasionally the vulva. The time of appearance and duration of these lesions was definitely associated with the onset and termination of her menstrual periods. During pregnancy, a complete cessation of these ulcers was noted, but immediately pregnancy was terminated, the lesions reappeared, thus indicating some hormonal action in the mouth. However, he does not elaborate to any great extent, how this takes place.

Richman and Abarbanel⁽⁸⁹⁾ state that hypertrophy and bleeding which occur during pregnancy do not appear to be due to the large amount of estrogen present, as large doses of estradiol and diethylstilboestrol administered to many women over a long period, did not produce this phenomenon. They think that the large amounts of progesterone is the exciting factor. Compare this with the idea of Ziskin and Nesse, of a sparring action of this hormone on estrogen.

In an attempt to correlate any variation in the salivary composition during pregnancy, with gingival state and caries, Speirs⁽¹⁰⁴⁾ carried out tests on 42 patients whose saliva was examined at regular intervals, from about the eighteenth week of preg-

nancy until six weeks post partum. The control group was composed of female students. However there was no consistent variation in the concentration of salivary calcium phosphates, mucin or hyaluronidase throughout pregnancy, nor was there a correlation between the presence of gingivitis and any particular salivary factor.

Horsnell and Packer, ^(57a) in a recent investigation in Adelaide, found that a group of post natal women showed a significant improvement in their gingival condition from when they were examined during their second trimester. The amelioration could not be attributed to any improvement in oral hygiene or ascorbic acid level. They did find that if the oral hygiene was adequate, the termination of pregnancy had little effect on the gingival condition.

Tiilila ⁽¹¹¹⁾ favours a hormonal etiology for epulis gravidarum for the following reasons. Firstly, in the cases described, two patients, with threatened miscarriage or spontaneous abortion had been treated with large doses of hormones, and secondly, it is indicative of hormonal influence, that epulides did not appear in greater numbers until the middle trimester of pregnancy, and that they showed an increased growth rate during the last trimester, but then regressed immediately after delivery. The study did not support the idea that the typical epulis gravidarum develops as a local exaggeration of pregnancy gingivitis since a large number of cases showed healthy gingivae in other areas and oral hy-

giene was good. Several epulides do not usually occur at the same time, but they usually do recur at the same site with each pregnancy, and this tends to indicate some local etiological factor. Some dental factor causing continuous irritation could be the cause, yet this was not observed by Tiilila.

The prolonged stress of pregnancy may cause, in some women a mild adrenal cortical insufficiency and because of local or systemic factors, an inflammatory process may develop in the gingivae. This is suggested by Bain.⁽⁶⁾ As there is little or no cortisone or hydrocortisone available in the circulation to combat the inflammation, the process goes unchecked.

Nutrition

That the nutrient demands of the growing foetus place a great drain on the reserves of the maternal organism is an established fact. The food habits of the pregnant woman are frequently modified for many reasons. The query then is posed as to what extent can these irregularities of the diet affect gingival tissues and what beneficial effect can be gained by correcting the diet.

Gompertz,⁽⁴³⁾ found in a survey of 200 pregnant patients, that general nutrition and dietary habits seem to make a difference in the mouth conditions. Women who were undernourished had more periodontal pathology.

Harris⁽⁵²⁾ found that in the north-east of Thailand, the pregnant women of the poorer classes ritually go onto a very poorly nutritious diet for the term of the pregnancy. The oral condition deteriorates badly, gingivitis becomes extremely bad, and in fact, a large proportion of them become edentulous by their late twenties. A typical diet in this area, during pregnancy, would consist of:-

Cooked glutinous rice	(78%)
Fermented fish	(16%)
Chillies	(1%)
Onions	(1%)
Greens	(¼%)
Lime Juice	
Water	

Whilst it has become increasingly apparent in recent years, that extensive pathologic changes which characterise marked vitamin deficiencies are uncommon in this country, and countries with similarly high standard of living, there are some⁽⁶³⁾ who feel that states of moderate or mild deficiency of vitamins could play the leading or at least a major role in gingivitis in pregnancy.

Levy⁽⁶³⁾ states that scurvy manifests itself in pregnant women as a result of various faddist diets, distaste for food containing vitamin C, inability to keep food in the stomach due to hyperemesis gravidarum, and diets prescribed by attending

physicians for gastro intestinal disorders.

Glickman⁽³⁹⁾ says that although acute vitamin C deficiency causes the connective tissue of the marginal gingivae of experimental animals to be altered by oedema, collagen degeneration and haemorrhage, the deficiency is not responsible for the initiation of an increase in the incidence of marginal gingivitis. The irritation of gingival inflammation in acute vitamin C deficiency requires the presence of irritation from food remnants or debris in the gingival sulcus. Levy goes on to say that it seems not unlikely that gingivitis gravidarum is a manifestation of what might be called a "localised scurvy". He does feel that whilst treatment for the condition with vitamin C and calcium should not be carried out with unlimited hope, it does seem reasonable to advise the use of vitamin therapy in such cases.

Rohackova⁽⁹³⁾ and Ticha also think that there are indications that the administration of calcium salts, and comparatively large doses of vitamins is beneficial.

Lighterman⁽⁶⁴⁾ and Lees say that pregnancy gingivitis cannot be attributed to individual hormones such as progesterone, chorionic gonadotrophin, but rather result from the complex interaction of all vitamins and hormones with periodontal irritation which may or may not be clinically present, acting as a secondary factor.

In vitamin A deficiency, certain specific pathological

changes have been observed in numerous epithelial structure, such as epithelial hyperplasia and hyperkeratinisation, but Ziskin, Rosenstein and Druker, ⁽¹³⁶⁾ showed that with large doses of vitamin A, a degenerative change in the gingivae takes place. The addition of estrogen to the administration of vitamin A resulted in overcoming the high vitamin A effect, producing hyperplasia of the gingivae with marked restoration of tissue tone.

Sud, ⁽¹⁰⁶⁾ in his work on refugees from West Punjab, found that whilst the group had a general nutritional deficiency, only 6.3% manifested specific vitamin deficiencies. A relationship between advancing months of pregnancy existed and, as the severest of the forms of gingivitis were found in malnourished pregnant women, he feels that malnutrition would be the result of pregnancy throwing a further demand on the maternal organism.

Coven ⁽¹⁹⁾ thinks that some women who, prior to pregnancy were staunch adherents to the "lamb chop and toast" school, may decide to indulge in all those foods which, previously, they refused for fear of gaining weight. The sudden reversion to soft and sticky foods may result in lack of physiologic stimulation or thorough cleansing of the teeth, with the formation of carbohydrate deposits and plaques, resulting in inflammation of the gingival area. Whilst this could be the case with some women, I do not feel that undue emphasis can be placed on this assumption. There are no figures or facts to substantiate the theory, and most women who are under medical supervision, particularly at

the larger hospitals, have their diet strictly checked to avoid an undue increase in weight.

Daro⁽²³⁾ suggests that the etiologic factors in pregnancy gingivitis are:

1. Improper diet.
2. Disease complicating pregnancy.
3. Endocrinologic imbalance during pregnancy.

He emphasizes the importance of the nutritional angle. Pointing out that the fetus is, in effect, a parasite, which takes from the mother's blood only that which it needs for its development, it necessarily follows that the gravid woman who has adequate and proper nutriment in her blood stream to supply the needs of the rapidly developing foetus will endure the strain of pregnancy with minimal upset to the body tissues. He believes that one should not assume that the patient eats sensibly just because she appears strong. One needs to realize that the majority of women in the first pregnancy, are young, and that, youth being strong, nature appears kind.

Rogers and Kean⁽⁹²⁾ say that vitamin C is sometimes advantageous in pregnancy gingivitis, but feel that oral hygiene is more important, stating that rarely will it establish itself in the presence of good oral hygiene.

Cross⁽²¹⁾ says that in gingivitis gravidarum and pregnancy tumours, the most important factors are vitamin C deficiency

hormonal alteration and trauma. His treatment is the administration of large doses of ascorbic acid daily and thorough antenatal prophylaxis.

Friedman⁽³⁷⁾ believes in adequate diet supplemented with vitamin B complex and vitamin C, in conjunction with the removal of local irritations, as well as surgery, if necessary.

Kutscher,⁽⁶¹⁾ in order to evaluate the therapeutic value of vitamin C, vitamin P, vitamin K and rutin in the treatment of pregnancy gingivitis, studied 63 cases of gingivitis in pregnancy, nearly all of which were drawn from a shelter for unwed, pregnant women. 25 patients in the first two groups successively received vitamin C (500mgm a day) for 21 days, rutin (40mgm a day) with vitamin C (300mgm a day) for 21 days and vitamin P (500 mgm a day) with vitamin C (300mgm a day) for 21 days, and vitamin K (4mgm a day) for 5 days. Each patient also received a coronal scaling every 21 days. The ten patients in group 3 received only the coronal scaling every 21 days. The eight patients in group 4 received weekly conservative subgingival periodontal treatments. The twenty patients in group 5 were untreated controls. All the patients were under observation over the same period. He states that whilst this study does not pass judgment on the value of these agents in the treatment of simple cases of gingivitis or their usefulness in preventing the initial appearance or reappearance of pregnancy gingivitis, his results gave evidence that:

1. Vitamin C, rutin, vitamin P, and vitamin K are not significantly effective in the treatment of gingivitis or gingival haemorrhage associated with pregnancy.
2. Coronal scalings arrest the progress of symptoms.
3. Weekly conservative subgingival periodontal treatments are most effective in controlling and causing regression of these gingival disturbances.

The suggestion is also put forward that vitamin C, vitamin P, and vitamin K, and rutin therapy be administered only where specifically indicated during pregnancy, and not as shot gun therapeutics in gingivitis gravidarum.

This work is corroborated by Hilming.⁽⁵⁵⁾ A daily supplement of 50mgm of vitamin C was given to 61 patients, from their first examination to parturition, and 142 patients received no such supplement to their diet. Venous blood was taken at each examination, and the content of ascorbic acid in the serum was determined. His results showed that the 50mgm of vitamin C was not capable of reducing the frequency of pregnancy gingivitis, and that this type of specific gingivitis cannot be caused by a deficiency of vitamin C, as the values of serum ascorbic acid are placed on the same level as those in the other group, which he called non-specific. He concludes that nothing indicates that the typical amelioration of part of the specific cases in the ninth month is due to a better vitamin C level, or that the char-

acteristic amelioration after delivery in all specific cases has anything to do with this. Nor has this study brought any evidence that the increased tendency toward gingival bleeding which is an important symptom in the specific pregnancy gingivitis is caused by a relatively low content of vitamin C in the blood.

A diet which is adequate at one time, may be inadequately balanced under other circumstances, with a resultant manifestation of food deficiency. That pregnancy causes a similar dietetic stress is suggested by the work of Day, ⁽²⁴⁾ upon pregnant and non-pregnant guinea pigs. This is supported by Stearns, ⁽¹⁰⁵⁾ who points out that a diet that is just satisfactory for the non-pregnant woman, may be insufficient for her during pregnancy. Studies of indigent pregnant women in Iowa showed that the diets ingested by them do not differ conspicuously, except in quantity, from their life time habits. With few exceptions, a woman whose diet is poor in pregnancy, can be considered as having had a poor diet since early childhood.

In some cases, nutritional and possible vitamin deficiencies could cause gingivitis in pregnancy, and whilst some cases may be helped by large doses of vitamin C, it does seem unlikely to me, that this is the basic cause, particularly here in Australia where there is a plentiful supply of this material, as has been substantiated by Horsnell and Packer. ^(57a) Very few, if any, expectant mothers need to be on a diet lacking in nutrition. Admittedly, there may be a tendency in some groups to

partake of more glutinous foods. This results in plaque formation with consequent irritation to the gingival tissue. That the gingiva is more susceptible to the irritation during pregnancy, could be brought about by the softening of the epithelium and a more marked reaction to this purely local cause.

Much still needs to be clarified in the pathogenesis of gingivitis gravidarum, and, after studying the published material on the possible etiology, one is forced to realise that the answer is not simply to be found, even though each author is convinced of his own solution. Whether the condition is of purely local irritative origin and modified by systemic disturbance, or whether it is of systemic origin with the local factors playing a secondary role cannot be clearly ascertained from all that is written. The effect of nutrition may also play a modifying role in this regard.

It seems highly probable that no one factor is entirely responsible for gingivitis in pregnancy. Rather is it the inter-play of several conditioning factors, each playing its own specific role.

IX. TREATMENT

Treatment for epulides has already been discussed. For the other forms of gingivitis gravidarum treatment is usually a combination of home treatment by the patient, and that carried out by the dentist in the surgery.

Fish⁽³⁴⁾ says that periodontal treatment should not be postponed on account of pregnancy - in fact pregnant women stand quite a severe gingivectomy very well - but the patient's medical advisor must be consulted. Then, once surgery has been carried out, a sincere attempt must be made to keratinise the epithelium.

Withycombe⁽¹²²⁾ believes that if no pregnancy gingivitis has occurred by the sixth month, the patient has an excellent chance of completing her gestation without any gingival disturbance. If the prognosis in the second, third and perhaps fourth month is good, the mouth can be cleaned up, brought back to normal, and maintained at this level with very little trouble.

Kutscher⁽⁶¹⁾ advocated frequent conservative subgingival periodontal treatment until such times as more effective therapeutic measures are prescribed. Whilst there has been a tendency in the past to believe that gingivitis gravidarum required little or no treatment, as it would regress and disappear after parturition, it has now been found⁽⁵⁵⁾ that a small percentage leave behind permanent damage even though there has been some improvement after delivery. One should not take it for granted that we are con-

cerned with a harmless gingivitis which automatically disappears with the birth of a child.

Every case detected during pregnancy should be treated with all the means thought necessary. ⁽¹¹⁰⁾ Glickman ⁽³⁹⁾ says that with minor modifications, the principles which generally govern the management of gingival disease are applicable during pregnancy. Prevention of gingival disease and the simplest form of treatment should be the main approach. Surgical treatment is contra-indicated, unless it is impossible to relieve the patient's discomfort by other means. This is also advocated by Blake. ⁽⁹⁾

Glickman ⁽⁴⁰⁾ divides treatment into two sections:

a) First three months. All gingival disease and local irritants should be eliminated as early in the pregnancy as is possible, before the conditioning effect of the pregnancy upon the gingival tissues become manifest. When no source of irritation can be observed, one should still look for and eliminate any potential source of future irritation. The patient should be made aware of the importance of good oral hygiene and instructed in a correct method of brushing and inter-proximal stimulation. Tooth brushing, which may not cause disturbance in the non-pregnant individual, may cause gingival bleeding as the pregnancy progresses. Commonly, the patient is using a brush which is far too stiff, and cannot adequately carry out treatment.

b) After the first three months. It is during this period that the conditioning effect of the pregnancy upon the gingival response to local irritation is manifest. Unless the changes are tumours, treatment consists of removal of all irritants, and the correction of local conditions which could produce irritation, and the institution of fastidious home treatment. Apart from the obvious local irritants, such as calculus, debris, and overhanging margins, one must not overlook functional dystrophies and food impactions. The latter could be responsible for gingival disturbances in a mouth in which there appears to be no obvious irritation.

The fact that there is marked improvement in the severity of gingival disease occurring after pregnancy does not necessarily mean a return to gingival health. It may only be in an ameliorated form.

If bleeding is bothersome and causing marked discomfort, which in turn is interfering with mastication, postponing the treatment is contra-indicated. The use of ascorbic acid and other vitamin preparations was discussed earlier under "Nutrition."

Archer⁽³⁾ points out that the fear of doing surgery on pregnant women is based on the fear of causing an abortion or even causing actual physical damage to the child. Nothing could be further from the truth. He cites Davidson, who, in an analysis of 1,000 pregnancies during which oral operations had been carried out, there was not a single case in which the operation was proved

to be the cause of any complication appearing. Hirschfeld, ⁽⁵⁶⁾ in referring to hypertrophic gingivitis of pregnancy, believed that treatment by surgery is contra-indicated except in the rare cases in which the pathological tissues cannot otherwise be successfully reduced and are causing annoyance.

Monash ⁽⁷⁷⁾ advised the elimination of all irritating factors and, if necessary, surgery, a month or two after delivery. Bressler ⁽¹³⁾ suggests preventive treatment before women become pregnant, and suggests the removal of all possible sources of irritation.

Beube ⁽⁸⁾ feels that as the condition is transient and will recede in most cases, good oral hygiene is the main treatment. Whilst stating that surgery can be employed, he thinks there is the danger of severe haemorrhage. I do not feel that this presents any real problem as I have carried out many gingivectomies and seen many done by others on pregnant women, and this has never been a complicating factor.

Wade ⁽¹¹⁷⁾ is not in favour of gingivectomy during hormonal imbalance until equilibrium is restored, as he claims only slight irritation of the marginal gingivae will cause recurrence. As the whole of pregnancy brings about an alteration in the hormonal system, I feel that if a gingivectomy is warranted, and if there is no contra-indication from the patient's state of general health, then this premise should not apply, and a gingivectomy should be carried out.

Coolidge⁽¹⁸⁾ thinks that the removal of local irritants, and improved home care by the patient until after parturition will usually result in a disappearance of the lesion. If hyperplasia persists then surgery may be employed. If there is a marked gingivitis, I do think that improved oral hygiene will help to restrain it from becoming worse, but I feel that surgery is indicated more frequently than is implied by Coolidge.⁽¹⁸⁾

Cross⁽²¹⁾ advocates daily doses of ascorbic acid (100-300mgm.) combined with thorough antenatal prophylaxis. Ziskin and Silver⁽¹³⁵⁾ point out that the improvement that takes place under local treatment does not preclude the possible systemic etiological factors, because gingivitis gravidarum responds in a similar manner to the same treatment.

Generally speaking, local conservative treatment is the popular choice. That means there will be no calculus, food impaction area due to faulty contact points, ill fitting crowns, bridges, and dentures, or broken down teeth remaining to cause any irritation. The home treatment plan is then outlined to the patient. If it can be ascertained that there is anything lacking in the diet, then steps should be taken to see that this is corrected.

There is now an increasing awareness amongst obstetricians of dental problems that might arise during pregnancy, but it is still not sufficiently widespread to make all expectant mothers

seek treatment at the beginning of pregnancy. Too frequently, dental care is neglected until the eighth or ninth month and the patient is left wondering whether to wait until the child is born or have treatment immediately. Unfortunately there still exists in the profession those who cling to the idea that no dental treatment should be carried out during pregnancy. Abortion rarely, if ever, occurs from routine dental treatment. (98) It is not a contraindication to extraction or surgery if pain is controlled, (3)(12)(107) but if the patient has a history of abortion, then procedure should be carefully considered and the obstetrician consulted if there is any doubt. It is desirable for the dentist to see the expectant mother frequently, but long appointments should be avoided. The use of local anaesthesia should be employed where possible. In the case of a highly emotional and sensitive woman, special attention may be necessary. During the first twelve weeks and the last two months, as well as during the time menstruation would normally occur, major treatment which could cause shock should be avoided.