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.

[Signature]
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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.
Pregnancy is associated with great anatomical and physiological changes of varying kinds, 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, others in vitamin C as a primary factor, and still others in the importance of hormonal factors.

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

There are various observations of haemangiomas on the face and neck with grew with pregnancy. Greenhill 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, vary-
ing 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 nataly in the Post Natal Clinic
at the same hospital. The women represented a wide range of nation-
alities, 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 con-
dition. 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 Tammin 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, 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. (125)

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.

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 peristeum. 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 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. 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. The blood increases in volume with a slight decrease in cell and haemoglobin content. 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 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.
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 gonadotrophic 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. Adrenocorticotropic 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 thyro trophic 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 in-
jections 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 epithel-
ial hyperplasia, causing enlargement of the thyroid gland, take 
place during pregnancy. There is an increase in the basal meta-
bolic rate because of increased demands on the part of the foetus. 
Thyroidectomy in Rhesus monkeys causes hyperkeratosis in the alveo-
lar and areolar gingivae, epithelial intercellular oedema, in-
creased 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) (132)

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) Lutinizing 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 -
B-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 grad-
ually, 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 glu-
coronide 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, 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 mgs 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.**

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 pregnandiol.

During the first three months of pregnancy, the pregnandiol 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) (55)

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, 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.)

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. 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) (75)

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 dissolves 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 Stou (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:

<table>
<thead>
<tr>
<th>Group 1.</th>
<th>No pathology</th>
<th>236</th>
<th>or</th>
<th>44.6%</th>
</tr>
</thead>
<tbody>
<tr>
<td>Group 2.</td>
<td>Mild inflammation</td>
<td>190</td>
<td>or</td>
<td>35.9%</td>
</tr>
<tr>
<td>Group 3.</td>
<td>Moderate inflammation</td>
<td>93</td>
<td>or</td>
<td>17.5%</td>
</tr>
<tr>
<td>Group 4.</td>
<td>Severe inflammation</td>
<td>8</td>
<td>or</td>
<td>1.5%</td>
</tr>
<tr>
<td>Group 5.</td>
<td>Tumour formation</td>
<td>3</td>
<td>or</td>
<td>0.5%</td>
</tr>
</tbody>
</table>

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 primaparae 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:

<table>
<thead>
<tr>
<th></th>
<th>Pregnant</th>
<th>Non-Pregnant</th>
</tr>
</thead>
<tbody>
<tr>
<td>No. of Cases</td>
<td>174</td>
<td>50</td>
</tr>
<tr>
<td>Class 1.</td>
<td>50%</td>
<td>76%</td>
</tr>
<tr>
<td>Class 2.</td>
<td>37%</td>
<td>16%</td>
</tr>
<tr>
<td>Class 3.</td>
<td>8%</td>
<td>8%</td>
</tr>
<tr>
<td>Class 4.</td>
<td>5%</td>
<td>nil</td>
</tr>
</tbody>
</table>

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 (95) 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, 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 pyorrhea, and a vastly greater number of women who become pregnant without ever having gingivitis.

Tiilila 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: 125 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 per-
centage 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 exis-
tence there is a variety of opinions as to its extent. It must
be realised that amongst those who deny the existence of the con-
dition, there are many who have not carried out their own statis-
tical 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 condi-
tion of local irritative origin.

Until such times as diagnosis of gingivitis gravidarum
relies less on the opinion of the examiner and more upon standard-
ised 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\(^{103}\) 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\(^{(28,39,49,59,134,108)}\) pregnancy is only a local intensification of gingivitis gravidarum. 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 attach-
ment. 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 occlu-
sion, 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. Thomas (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 gra-
vidarum 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:

<table>
<thead>
<tr>
<th>Author</th>
<th>Incidence</th>
</tr>
</thead>
<tbody>
<tr>
<td>Ziskin, Blackberg and Stout ((133))</td>
<td>0.7%</td>
</tr>
<tr>
<td>Maier and Orban ((69))</td>
<td>0.5%</td>
</tr>
<tr>
<td>Hilming ((55))</td>
<td>0.</td>
</tr>
<tr>
<td>Gridly ((49))</td>
<td>2.7%</td>
</tr>
<tr>
<td>Rohackova and Ticha ((93))</td>
<td>1.4%</td>
</tr>
<tr>
<td>Tiilila ((111))</td>
<td>0.2%</td>
</tr>
</tbody>
</table>

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 com-
parable 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. Burkett\(^{(12)}\) recommends surgical removal and cauterity if there is a haemorrhage and ulceration. Orban 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 cauterisation 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 indi-
cated. This extreme opinion does not appear to be held by any
other writer.

Ward and Hendrick(118) do not think they are of great practi-
cal significance, unless they bleed freely, when they should be
removed. They describe the case of a large birthmark which in-
volved the lip, buccal mucous membrane and skin of the cheek. Dur-
ing 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 spon-
taneously. 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 reoccurrence? 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 HISTOCHEMISTRY

of 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. Ziskin, Blackberg and Stout, and Ziskin and Nesse 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 is guarded in his statement that the epithelium is, in most cases, keratinised, Ziskin, Blackberg, and Stout also noticed that, in the early months, a slight hydropic change of the corneum occurred. Ziskin and Nesse 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 state that the marked vascularility 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 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 angiogramuloma. 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 angioblastas 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 eculus 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 eculus 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 cusp 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 no 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 Calm\textsuperscript{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.

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\textsuperscript{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 estrogentic 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 (31) 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, (154) 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. Hatscher, (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 (95) 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, in working on ovarian function, showed some connection between the severity of periodontal disease and the quality of the ovarian cycle.

Perl 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, 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 revues Ziskin and Nesse, but gives no new facts, merely confining his writings to a precis of their work.

Jenkins 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 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. 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 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 (4%)
- 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.

Robackova\(^{(93)}\) 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 Bees 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 Drucker, (156) 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.

Darce\textsuperscript{(25)} 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 nourishment in her bloodstream to supply the needs of the rapidly developing fetus 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\textsuperscript{(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\textsuperscript{(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 (500mg a day) for 21 days, rutin (40mg a day) with vitamin C (500mg a day) for 21 days and vitamin P (500 mgm a day) with vitamin C (500mg a day) for 21 days, and vitamin K (4mg 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 shotgun 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 nothing 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.

Hutscher (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. Glickman 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.

Glickman 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\(^{(5)}\) 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 reoccurrence. 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\(^{(13)}\) 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.\(^{(13)}\)

Cross\(^{(21)}\) advocates daily doses of ascorbic acid (100-300mg.) combined with thorough antenatal prophylaxis. Zickin 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 of 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. It is not a contraindication to extraction or surgery if pain is controlled, 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.
4. CLINICAL SURVEY OF THE GINGIVAL CONDITION OF ANTE NATAL AND POST NATAL WOMEN IN SYDNEY.

1. Introduction

This investigation was based on the examination of 684 pregnant women and 75 post natal patients, most of whom are of modest means. Case records were available for all patients. This survey was carried out to determine whether pregnant women exhibited a condition which can be termed Gingivitis Gravidarum, as well as to examine their periodontal state. It is impossible to have accurate criteria for gingivitis gravidarum; the ideal control being the patients themselves before pregnancy, and then observing any changes which take place during the nine months.

In assessing whether these patients had indeed, a gingivitis due to, or aggravated by, the pregnancy, it was necessary to rely on the patient's information as to whether there was bleeding, swelling or other changes now noticeable, which were not present before pregnancy commenced. In point of fact, of those patients who developed this condition during gestation and after my initial examination, many (apart from those showing gross changes) were not even aware that bleeding existed. Of the rest, all were very vague as to what stage the marked changes occurred. It can be readily appreciated that this lack of awareness on the
patient, makes clinical assessment very difficult. Failing examination before conception, the examiner is forced to draw his own conclusions and rely on his own judgement.

Detailed questioning was often necessary to determine the condition of the gingiva before conception and this proved at times to be difficult in cases where migrant women had no command of English. However, ways and means were found.

The aim of the survey was to ascertain as to what extent gingivitis gravidarum did exist and in what form. As there are various factors which could influence the gingival condition during pregnancy, consideration was given to all pertinent items related to the patient's present and past history. In the existing clinical set up, it was impossible to investigate hormonal changes. However, this has been covered earlier in the thesis by reference to all relevant literature. After discussion with the medical staff, it was assumed that no patient suffered any nutritional deficiency, as a careful check was made by the hospital staff as to the patient's diet. Any inability to maintain an adequate diet was overcome by the almoners.

Because of the large number of migrants who attended this hospital it was possible to consider the effect that nationality could have on the final results.

A comparison was to be drawn between the ante natal
patients and a similar group of post natal patients. It was my intention to examine a large number of those seen ante nataly again after parturition, but here a major difficulty was encountered. Due to many reasons, including apathy, and despite the fact that appointments are routinely made by the hospital, only a very small percentage return for their post natal examination.
II. Method.

The examination consisted of a clinical appraisal of the oral mucous membrane and gingiva with the aid of a mouth mirror, a periodontal probe graduated in millimetres, a chip blower and a spot light. A sickle shaped explorer such as an Ash 54 was used for examining oral debris. A collapsible dental chair was kindly made available by the Preventive Dentistry Department of the Dental Hospital, and this enabled me to move easily to various departments. There is no dental surgery or equipment available at Crown Street Hospital. Each patient’s periodontal condition was scored using Russell’s method which refers to the clinical evidence of obvious mild gingivitis as well as signs of more advanced periodontal disease. The Debris and Calculus Indexes were also scored from which the Oral Hygiene Index was calculated. In the assumption that a condition of gingivitis does exist due to the gravid state, those manifestations of gingival disturbances as classified by Ziskin and Nesse were also scored. The following information was noted:—the patient’s age, nationality, number of previous pregnancies (including any miscarriages), age at each pregnancy, sex of previous children, last dental visit, frequency of visits, previous gingival disturbances, frequency of tooth brushing, number of weeks of gestation, and any relevant medical history. (See Figures One & Two.)

All examinations were carried out by me so as to ensure a standardisation of scoring.
FIGURE I.

UNIVERSITY OF SYDNEY

DEPARTMENT OF PREVENTIVE DENTISTRY

PERIODONTAL SURVEY HISTORY

NAME................................................................ HOSPITAL IDENTIFICATION ..............

DATE OF BIRTH........................................... AGE ............................................................

NATIONALITY ..........................................

NUMBER OF PREGNANCIES ........................................................

AGE AT PREGNANCY ....................................................................

SEX OF CHILDREN ....................................................................

SPECIAL PROBLEMS – PREGNANCY ........................................

MENSTRUAL ..............................................................................

DENTAL HISTORY – LAST VISIT ..............................................

FREQUENCY OF VISITS ................................................................

PREVIOUS GINGIVAL DISTURBLANCES ......................................

FREQUENCY OF TOOTHBRUSHING ..........................................

MALOCCLUSION ........................................................................

PRESENT ORAL STATUS .............................................................

GENERAL MEDICAL HISTORY ..................................................

............................................................................................
**FIGURE II**

**UNIVERSITY OF SOUTHERN CALIFORNIA**
**DEPARTMENT OF PRIMITIVE DENTISTRY**

**PERIODONTAL SURVEY CHART**

<table>
<thead>
<tr>
<th>Name</th>
<th>Age</th>
<th>Pregnancy (Weeks)</th>
<th>Hospital Number</th>
<th>Examiner</th>
<th>Date</th>
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</table>

**RUSSELL’S PERIODONTAL INDEX**

<table>
<thead>
<tr>
<th>Score</th>
<th>0</th>
<th>1</th>
<th>2</th>
<th>(4) X-ray only</th>
<th>6</th>
<th>8</th>
<th>X (Tooth Absent)</th>
</tr>
</thead>
</table>

<table>
<thead>
<tr>
<th></th>
<th>Central</th>
<th>Lateral</th>
<th>Cuspid</th>
<th>Bicuspid 1</th>
<th>Bicuspid 2</th>
<th>Molar 1</th>
<th>Molar 2</th>
<th>Molar 3</th>
</tr>
</thead>
<tbody>
<tr>
<td>Upper</td>
<td>Left</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Right</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Lower</td>
<td>Left</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Right</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Periodontal Index = Sum of individual scores
Number of teeth present

**ORAL HYGIENE INDEX**

<table>
<thead>
<tr>
<th>Debris Score</th>
<th>0, 1, 2, 3, X</th>
<th>Calculus Score</th>
<th>0, 1, 2, 3, X</th>
</tr>
</thead>
<tbody>
<tr>
<td>Right</td>
<td>Anterior</td>
<td>Left</td>
<td>Total</td>
</tr>
<tr>
<td>Upper</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Lower</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Sub-total</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Debris Index =
Calculus Index =
Oral Hygiene Index =

**PREGNANCY GINGIVITIS (GISKIN)**

<table>
<thead>
<tr>
<th>Score</th>
<th>0, 1, 2, 3, 4, 5</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Central</td>
</tr>
<tr>
<td>Upper</td>
<td>Left</td>
</tr>
<tr>
<td></td>
<td>Right</td>
</tr>
<tr>
<td>Lower</td>
<td>Left</td>
</tr>
<tr>
<td></td>
<td>Right</td>
</tr>
</tbody>
</table>
Epidemiological concepts had their beginnings many centuries ago. Before 400 B.C. Thucydides wrote of his belief in observing facts and judging by rule of reason. One basic idea at least has persisted from this early beginning down to the present—that is the concept that epidemiology is concerned with groups of people. Until recently much of our information on the prevalence and incidence of periodontal disease has been based on clinical observations, unsupported by adequate or reliable statistical data. Yet a knowledge of the extent of the problem is a basic necessity in the prevention and control of the disease.

Population indexes for periodontal diseases are numbers that define the relative status of a population on a graduated scale with definite upper and lower limits, for comparison with other populations classified by the same criteria and method.

No index represents a precision measurement as in physics; all rely to some extent upon the judgement of the examiner. Some may be determined very swiftly for large numbers of people, others require so much time as to limit the numbers that can be studied. Some require the use of bulky accessory aids such as X-Ray equipment, others are designed for a minimum of instruments.

The choice of an index in any specific situation should be based on the following:

1. The method should be sufficiently quantitative to provide a
measure of the degree of severity of the disease in population groups.

2. The method should be practicable for use in epidemiological studies of large populations and should have speed and simplicity of application rather than complexity even when the speed and the simplicity require sacrificing some details.

3. The technique to be employed must be within the abilities of those employing them, and general practitioners should be able to use it.

4. The recording should be suitable to reduction to a final score or index which may be subjected to statistical analyses.

5. The method of choice must have definite clear cut criteria which reduce the examiner's decisions to a minimum.

Arbitrary classifications, such as mild or severe are of little value without a clear definition of the meaning of these terms. (72) Because of the differences in the examination method employed for the assessment of the disease there have been wide variations in the findings of different workers. As there has been no agreement in the system of examination it is most difficult to know whether all the past reports from the various investigations indicate a real variation in the frequency of the disease between and within the various population groups.

The two systems at present in most use for the recording
of periodontal disease are the P.M.A. Index \(^{70}\) and the Periodontal Index (P.I.) \(^{95}\). Briefly P.M.A. Index of gingivitis was evolved by Massler and Schour and is a method of quantitatively assessing gingival disease and is based upon the examination of each individual gingival unit in the mouth, the papillary (P), marginal (M), and the attached (a) gingiva mesial to any given tooth.

The Periodontal Index of Russell, which has been used in this survey, is a more comprehensive index covering both gingivitis and periodontitis. The condition of the investigating tissues is estimated individually for each tooth, and is scored according to a progressive scale which gives relatively little weight to soft tissue inflammation and relatively great weight to destruction of bone. The score for an individual is the arithmetic average of the scores for the teeth in his mouth. The population score is the arithmetic average of the individual score for the persons examined.

Each tooth is scored according to the clinical condition of its supporting tissues. In the absence of overt inflammation in the free gingivae, or loss of function due to destruction of investing tissues, a tooth is considered to be negative and is assigned a score of zero. The scale provides for two stages of simple gingivitis, differing only to the extent of inflammation, and scored 1 and 2. A score of 6 is assigned when a periodontal pocket is demonstrated, except when tissue destruction is so far advanced that masticatory function has been impaired; in this
case the highest score 8, is given. If radiographs are studied it is sometimes possible to detect bone resorption prior to the appearance of a frank periodontal pocket. In the clinical test such cases would be assigned an intermediate score of 4.

Criteria for the Periodontal Score

Score 0. Negative. There is neither overt inflammation in the investing tissues nor loss of function due to destruction of supporting tissues.

Score 1. Mild Gingivitis. There is an overt area of inflammation in the free gingivae, but this area does not circumscribe the tooth.

Score 2. Gingivitis. Inflammation completely circumscribes the tooth, but there is no apparent break in the epithelial attachment.

Score 4. Not used in this clinical study.

Score 6. Gingivitis with pocket formation. The epithelial attachment has been broken and there is a pocket (not merely a deepened gingival crevice due to swelling in the free gingivae). There is no interference with the normal masticatory function, the tooth is firm in its socket, and has not drifted.

Score 8. Advanced destruction with loss of masticatory function. the tooth may be loose, may have drifted; may sound dull to
percussion with a metallic instrument; may be depressible in its socket.

The most difficult decision required of the examiner is usually that of distinguishing between negative gingivae and gingivae with mild gingivitis. A score for gingivitis is only assigned when the inflammation is clearly evident at first glance in good light. The problem of a questionable diagnosis is avoided by the rule, when in doubt, assign the lower score.

The index may be applied very swiftly and a minimum of equipment is necessary.

Used in this survey also, is the Oral Hygiene Index. It is composed of the Debris Index and Calculus Index. Each Index is based on numerical determinations representing the amounts of debris or calculus found on the buccal and lingual surfaces of each of three segments of each dental arch.

1. The segment distal to the right cuspid
2. The segment distal to the left cuspid
3. The segment mesial to the right and left first bicuspids.

Separate recordings are made for the buccal and lingual surfaces in recognition of the differences in oral hygiene that may exist between these surfaces. The individual indexes are derived from scores based on the fraction of tooth surface covered by debris or calculus. As there are considerable differences in the height
of clinical crowns of permanent and deciduous teeth, only fully erupted permanent teeth are scored. (A tooth is considered fully erupted when the occlusal or incisal surface has reached the occlusal plane.) Third molars and incompletely erupted teeth are not scored because of the wide variations in the heights of clinical crowns.

The two debris scores assigned to a segment are based on the buccal surface and lingual surface having the greatest surface covered by debris. The buccal score and the lingual score need not be taken from the same tooth. The surface area covered by debris is estimated by running the side of a sickle shaped explorer along the buccal, labial and lingual surfaces and noting the occlusal or incisal extent of the debris as it is removed from the tooth surface.

The method for scoring calculus is the same as that applied to debris with a specific score for subgingival deposits. The amount of calculus present is by visual examination and probing. Oral debris is the soft foreign matter adhering to the surface of the teeth. It consists of mucin, bacteria and food.

There are two scores for each segment in which there are fully erupted permanent teeth present, one score for the buccal surface, and one score for the lingual surface. The scores and criteria for oral debris are:
Score 0. No debris or stain present.

Score 1. Soft debris covering not more than one third of the tooth surface, or the presence of extrinsic stains without other debris regardless of surface area covered.

Score 2. Soft debris covering more than one third, but not more than two thirds, of the exposed tooth surface.

Score 3. Soft debris covering more than two thirds of the exposed tooth surfaces.

Criteria for Classifying Calculus

Dental calculus\(^{(14)}\) is defined as a deposit of inorganic salts composed primarily of calcium carbonate and phosphate mixed with food debris, bacteria, and desquamated epithelial cells. Dental calculus is divided into two types, differentiated primarily by location on the tooth in relation to the free gingival margin:

1. "Supragingival Calculus" - denotes deposits located occlusal to the free gingival margin and usually white to yellowish brown in colour.

2. "Subgingival Calculus" - denotes deposits located apically to the free gingival margin, which are usually light brown to black in colour because of inclusion of blood pigments.

When recording, the operator only notes deposits of hard calculus. Segments which have one or more permanent teeth standing are given two scores - one score for the buccal surface
segment and one for the lingual having the greatest accumulation of calculus. These two scores need not be taken from the same tooth.

The scores and criteria for oral calculus are:

0. No calculus present.

1. Supragingival calculus covering not more than one third of the exposed tooth surface.

2. Supragingival calculus covering more than one third, but not more than two thirds of the exposed tooth surface or the presence of individual flecks of sub-gingival calculus around the cervical portion of the tooth, or both.

3. Supragingival calculus covering more than two thirds of the exposed tooth surface or a continuous heavy band of sub-gingival calculus around the cervical portion of the tooth or both.

Calculation of Indexes.

To determine an individual's Debris Index, the debris scores were totalled and divided by the number of segments scored. The Calculus Index was arrived at by the same method. Oral Hygiene Index was found by adding the two scores. Since the Debris and Calculus Indexes each had a possible scoring of from 0. to 6., the Oral Hygiene Index ranged from 0. to 12.

Massier, Schour and Schopra,\(^{(71)}\) in 1950, suggested that
their study gave statistical proof that the examination of the anterior segments alone furnished a valid index of gingivitis of the entire mouth. However, for this survey, of course, the whole mouth was examined.

Ziskin's classification of gingivitis of pregnancy, as described earlier, was followed with one variation. No subdivision was made for Class 4.

Most patients presented themselves for their first medical examination shortly after the second month, although some, particularly the multiparae, did not come until late in their term. As a result, it is difficult to determine the actual time of onset of the gingivitis. The commencement of bleeding of the gingivae and changes in appearance are the only accurate data we can rely on.

To determine clinically whether any changes would take place and whether these anticipated changes would fall into any recognizable pattern, 141 patients were examined twice, and of these, 16 were examined a third time. So that I would not be influenced by anything that had gone before, patients examined for the second, third, or post-natal times, were done so without any reference to previous charts. For a control group, 75 post-natal patients were examined under identical conditions, at the same hospital. These were seen on the same day as they presented themselves for their final medical examination.
The two groups together, gave a total of 916 examinations carried out.

**TABLE 1.**

<table>
<thead>
<tr>
<th>Number of Patients Seen Once</th>
<th>Number of Patients Seen Twice</th>
<th>Number of Patients Seen Thrice</th>
<th>Number of Post-Natal Patients</th>
<th>Total Number of Examinations</th>
</tr>
</thead>
<tbody>
<tr>
<td>684</td>
<td>141</td>
<td>16</td>
<td>75</td>
<td>916</td>
</tr>
</tbody>
</table>

Where the patients were seen twice, it could have been a combination of first and second trimester, first and third, or second and third.

The group seen three times were seen in their first, second and third trimesters.
III. RESULTS.

The whole group generally showed poor oral hygiene, and many had badly neglected mouths with broken down teeth that had not been treated for many years. For some of the migrant women, this was their first dental examination. Most patients had some excuse - whether it be lack of finance to attend their local dentist, and reluctance to attend the Dental Hospital, overburdening with family duties and numerous young children to care for, and for those living in outlying suburbs, the added difficulty of poor transport. Some patients simply did not realise that treatment was needed, and others took the attitude, that if the discomfort became sufficiently annoying, the tooth could be extracted. This casual approach was more prevalent in the Australian group, who were aware of their bad oral condition, but who were not prepared to seek treatment. On the other hand, the migrant groups, whose teeth were basically sound, but who showed many gingival disturbances, without experiencing any pain, did not realise that treatment was needed.

Of the patients who were examined more than once changes in the gingival condition could be noted.

Forty patients showed a definite increase in gingivitis, this being either from no gingivitis gravidarum to the presence of it, or an increase of that which was present. Thirteen patients showed a definite reduction of the gingivitis in the ninth
month without having any treatment. This follows the observation of others (55) who have observed the same decrease during the last month. Of the seventy-five post-natal patients seen six to seven weeks after delivery, sixteen had been examined ante nataly. Of the sixteen, twelve had been classified as having gingivitis gravidarum. Two showed no change, and the remaining 10 showed a complete absence of the condition.

In the ante natal group, at the first examination, there were only thirty-one cases, or 4.5%, who had completely normal gingiva, with a Russell's Periodontal Index of zero. As there is a score if even one inflamed papilla is present this does eliminate many with only minor gingival disturbances. Of the 604, there were 335 who exhibited examples of gingivitis gravidarum. The gingivitis had commenced or increased after pregnancy began. The majority of these cases were of Ziskin's Class I and Class 2, i.e. with a tendency for the gingiva to bleed or to show a glossy appearance and a lack of stippling. The hypertrophic type, which some authorities found to be frequent, did not occur to such an extent in this survey, there being only twenty-four cases of it alone, or in combination with another type. As can be seen from Table XI, sixty-two patients exhibited more than one type of gingivitis gravidarum. There were only two cases of epulis in the whole group.

Norrin (79) is of the opinion that Ziskin's first three
classifications of gingivitis gravidarum may not, with certainty, be attributed to the pregnancy, but the hypertrophic type and epulis may. On the basis of my observations, I cannot agree with him. The general appearance of all cases of gingivitis fitted in with those described by Ziskin and Nesse. (134)

**TABLE II.**

Distribution of Type of Gingivitis

(Ziskin Classification)

At the First Examination

<table>
<thead>
<tr>
<th>Type</th>
<th>Number of patients in group</th>
<th>Total Number including combination*</th>
</tr>
</thead>
<tbody>
<tr>
<td>Class 1.</td>
<td>131</td>
<td>159</td>
</tr>
<tr>
<td>Class 2.</td>
<td>107</td>
<td>150</td>
</tr>
<tr>
<td>Class 3.</td>
<td>22</td>
<td>68</td>
</tr>
<tr>
<td>Class 4.</td>
<td>9</td>
<td>24</td>
</tr>
<tr>
<td>Class 5.</td>
<td>2</td>
<td>2</td>
</tr>
</tbody>
</table>

Combination: 62.

Total: 333.

Total Number of Ante Natal Patients Examined = 684.

* Combination refers to those cases exhibiting more than one type of gingivitis (Ziskin) in different areas of the mouth.
Most patients who had a gingivitis due to pregnancy did appear to have had some form of gingivitis or periodontitis beforehand. This ranged from very slight to very marked. In many instances, it was quite apparent from the overall picture, that there had been some gingival disturbance present, and that pregnancy, by some means, had aggravated the condition. Whilst there is generally a pre-existing gingivitis, authorities do claim that it can occur without any such conditions being present. There was usually no obvious reason why an increase in gingivitis should occur in specific locations in the mouth when there was already a generalised gingivitis present.

In certain patients who were examined more than once, there was a change in location, particularly if it were only mild, e.g. Ziskin Class 1, or Class 2. Bleeding had occurred in certain papillae at one stage, had regressed and commenced around other teeth. Whether this was because the patient had concentrated on better brushing in the indicated area is not certain. However, people are more inclined to brush less when bleeding occurs, so it is very unlikely that increased brushing actually took place.

Although the majority of the patients claimed they brushed their teeth, observation showed that very few carried out a proper brushing technique. On first examination some showed a mild gingivitis with a Ziskin classification of 1 or 2. When later
examined again, they showed that their gingivitis had acquired a proliferative or productive character. These were of a definite, progressive nature, and allowed more than one type to be present in the mouth at the same time. These are referred to in Table II as a combination. The occurrence of more than one type in the mouth is in agreement with Ziskin and Nesse. (134)

The oral hygiene of the mouth appeared to play an important role in some where the gingivitis coincided with large amounts of oral debris. In others, the oral hygiene was very good, with no calculus detectable, and a very low or zero debris score, and gingivitis gravidarum still occurred in these apparently clean areas.

Age.

The distribution of patients into age groups is shown in Table III and it can readily be observed that the age of the patient had no great bearing on the incidence of gingivitis gravidarum. It was present in all age groups, but the 26 - 30 year group showed the highest incidence, with a percentage of 56.2. It ranged from 28.5% in the 15 years and under, up to the maximum of 56.2%, and dropped to 34% in the oldest group. However the application of the Chi-Square test to this division of age showed that there was no statistical significance in this grouping.

Number of Pregnancies.

The number of pregnancies of each patient, including any
mis-carriages and abortions were noted from the hospital medical records, to determine if there was any factor which could influence the incidence of gingivitis gravidarum. However, from Table IV it can be seen that there is no specific pattern and once again the Chi-Square test showed no statistical significance in the frequencies of the group. The primaparæ formed the largest group and with those in their second or third pregnancy composed the majority of the patients.

Weeks of Gestation.

As can be seen from Tables V and VI the patients on their first examination have been divided into groups according to their weeks of gestation. The greatest incidence of gingivitis gravidarum occurred in the 25 - 28 week group. There is an increase in each four-weekly group, commencing at 8 weeks to a peak at 25 - 28 weeks with a decline to 32 weeks and a slight increase again to the last 4 weeks of pregnancy. The incidence of gingivitis gravidarum ranged from 35.2% in 0 - 8 week group, to a peak of 61.7% in the 25 - 28 week group and down to 49.1% in the 37 week-term group. The frequency of brushing and the Russell Index, Debris, Calculus and Oral Hygiene Index for each group is given in these tables. There was no relationship between the incidence of gingivitis gravidarum in Table V and the Russell Index. The Russell Index was highest in the 33 -36 week group which exhibited one of the lower incidences of gingivitis gravidarum. Table VI gives those without gingivitis gravidarum and shows the various Indexes for each four weekly group.
TABLE III.

Distribution of Age Groups of Ante Natal Patients
At Their First Examination.

<table>
<thead>
<tr>
<th>Age</th>
<th>Number In Group</th>
<th>Number With Gingivitis Gravidarum</th>
<th>Percentage With Gingivitis Gravidarum</th>
</tr>
</thead>
<tbody>
<tr>
<td>15</td>
<td>7</td>
<td>2</td>
<td>28.5</td>
</tr>
<tr>
<td>&amp; under</td>
<td>186</td>
<td>89</td>
<td>47.8</td>
</tr>
<tr>
<td>16 - 20</td>
<td>223</td>
<td>109</td>
<td>47.8</td>
</tr>
<tr>
<td>21 - 25</td>
<td>153</td>
<td>86</td>
<td>56.2</td>
</tr>
<tr>
<td>26 - 30</td>
<td>66</td>
<td>32</td>
<td>48.5</td>
</tr>
<tr>
<td>31 - 35</td>
<td>44</td>
<td>15</td>
<td>34.0</td>
</tr>
<tr>
<td>36 &amp; over</td>
<td>684</td>
<td>533</td>
<td>48.7</td>
</tr>
</tbody>
</table>

Other factors which were noted during the examination, and which proved to have no bearing on the subject were: six of previous children, menstrual disturbances, and previous medical history.
TABLE IV.

Incidence of Gingivitis Gravidarum Distributed

According to the Number of Pregnancies

(Including Miscarriage)

<table>
<thead>
<tr>
<th>Number of Pregnancies</th>
<th>Number in Group</th>
<th>Number with Ging. Grav.</th>
<th>Percentage with Gingivitis Grav.</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>273</td>
<td>129</td>
<td>47.2</td>
</tr>
<tr>
<td>2</td>
<td>161</td>
<td>90</td>
<td>55.9</td>
</tr>
<tr>
<td>3</td>
<td>108</td>
<td>45</td>
<td>41.1</td>
</tr>
<tr>
<td>4</td>
<td>60</td>
<td>32</td>
<td>55.0</td>
</tr>
<tr>
<td>5</td>
<td>34</td>
<td>10</td>
<td>29.4</td>
</tr>
<tr>
<td>6</td>
<td>22</td>
<td>13</td>
<td>59.1</td>
</tr>
<tr>
<td>7</td>
<td>9</td>
<td>6</td>
<td>66.6</td>
</tr>
<tr>
<td>8 or more</td>
<td>17</td>
<td>8</td>
<td>47.0</td>
</tr>
<tr>
<td>Total:</td>
<td>684</td>
<td>333</td>
<td></td>
</tr>
</tbody>
</table>
Brushing.

The patients were questioned as to the frequency of their brushing, and if there was any doubt, the lower score was taken. They were divided into three groups.

a) Those who brushed less than once a day. Several in this group claimed that they did not brush their teeth at all, and some only once a week.

b) Those who brushed once a day.

c) Those who brushed twice a day or more.

For the majority of cases a poor brushing technique was evident. In the whole ante natal group a higher brushing score was accompanied by a lower Russell Index, Debris, Calculus and Oral Hygiene Indexes. (Table VII.). In the group with pregnancy gingivitis, the Russell score was almost the same for those who brushed less than once a day and those who brushed once daily. The Debris, Calculus and Oral Hygiene scores were progressively lower with the increased brushing. (Table VIII). In the group without pregnancy gingivitis the Russell Index showed a reverse pattern with the highest score for the group which brushed twice or more daily. The Oral Hygiene score was lower in this group with the increased brushing as was the Debris score, but the Calculus did not follow this pattern. There were, however, on thirteen patients in the group who brushed less than once daily, compared with 106 and 232 in the other two groups. (Table IX.)
### TABLE V.

Division of Group into Weeks of Gestation, Showing Number and Percentage with Gingivitis Gravidarum and Distribution of Brushing Russell's, Debris Calculus and Oral Hygiene Indexes

<table>
<thead>
<tr>
<th>Weeks of Gestation</th>
<th>No. in Group</th>
<th>No. with G. G.</th>
<th>% with G. G.</th>
<th>Daily Brushing less than 1. 1.</th>
<th>2 or more Russell's</th>
<th>Debris Calculus</th>
<th>Oral Hygiene</th>
</tr>
</thead>
<tbody>
<tr>
<td>0-8</td>
<td>17</td>
<td>6</td>
<td>35.2</td>
<td>0 2 4</td>
<td>2.54</td>
<td>2.71</td>
<td>1.19</td>
</tr>
<tr>
<td>9-12</td>
<td>66</td>
<td>26</td>
<td>39.3</td>
<td>3 8 15</td>
<td>2.25</td>
<td>3.29</td>
<td>0.39</td>
</tr>
<tr>
<td>13-16</td>
<td>54</td>
<td>26</td>
<td>48.1</td>
<td>2 5 19</td>
<td>2.25</td>
<td>2.6</td>
<td>0.66</td>
</tr>
<tr>
<td>17-20</td>
<td>65</td>
<td>33</td>
<td>50.7</td>
<td>4 15 14</td>
<td>2.78</td>
<td>3.46</td>
<td>0.84</td>
</tr>
<tr>
<td>21-24</td>
<td>72</td>
<td>38</td>
<td>52.7</td>
<td>4 14 20</td>
<td>2.74</td>
<td>3.43</td>
<td>1.01</td>
</tr>
<tr>
<td>25-28</td>
<td>81</td>
<td>50</td>
<td>61.7</td>
<td>4 21 25</td>
<td>2.78</td>
<td>3.26</td>
<td>0.72</td>
</tr>
<tr>
<td>29-32</td>
<td>91</td>
<td>39</td>
<td>42.8</td>
<td>4 20 15</td>
<td>2.26</td>
<td>3.18</td>
<td>0.62</td>
</tr>
<tr>
<td>33-36</td>
<td>118</td>
<td>56</td>
<td>47.4</td>
<td>5 27 24</td>
<td>3.03</td>
<td>3.31</td>
<td>0.81</td>
</tr>
<tr>
<td>37 to term:</td>
<td>120</td>
<td>59</td>
<td>49.1</td>
<td>8 20 31</td>
<td>2.68</td>
<td>3.15</td>
<td>0.8</td>
</tr>
<tr>
<td>Total:</td>
<td>684</td>
<td>333</td>
<td>48.7</td>
<td>34 132 167</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

(Note: G.G. = Gingivitis Gravidarum).
<table>
<thead>
<tr>
<th>Weeks of Gestation</th>
<th>No. in Group</th>
<th>Daily Brushing</th>
<th>Russell's</th>
<th>Debris</th>
<th>Calculus</th>
<th>Oral Hygiene</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>less than 1.</td>
<td>1.</td>
<td>2 or more</td>
<td></td>
<td></td>
</tr>
<tr>
<td>0-8</td>
<td>11</td>
<td>0</td>
<td>5</td>
<td>6</td>
<td>2.46</td>
<td>1.91</td>
</tr>
<tr>
<td>9-12</td>
<td>40</td>
<td>1</td>
<td>8</td>
<td>31</td>
<td>1.57</td>
<td>2.4</td>
</tr>
<tr>
<td>13-16</td>
<td>28</td>
<td>1</td>
<td>9</td>
<td>18</td>
<td>1.55</td>
<td>2.63</td>
</tr>
<tr>
<td>17-20</td>
<td>32</td>
<td>1</td>
<td>14</td>
<td>17</td>
<td>1.26</td>
<td>2.11</td>
</tr>
<tr>
<td>21-24</td>
<td>34</td>
<td>1</td>
<td>8</td>
<td>25</td>
<td>0.9</td>
<td>2.32</td>
</tr>
<tr>
<td>25-28</td>
<td>31</td>
<td>3</td>
<td>9</td>
<td>19</td>
<td>1.38</td>
<td>2.84</td>
</tr>
<tr>
<td>29-32</td>
<td>52</td>
<td>3</td>
<td>14</td>
<td>35</td>
<td>1.34</td>
<td>2.49</td>
</tr>
<tr>
<td>33-36</td>
<td>62</td>
<td>1</td>
<td>18</td>
<td>43</td>
<td>1.2</td>
<td>2.04</td>
</tr>
<tr>
<td>37 to term:</td>
<td>61</td>
<td>2</td>
<td>21</td>
<td>38</td>
<td>1.42</td>
<td>2.54</td>
</tr>
<tr>
<td>Total:</td>
<td>351</td>
<td>13</td>
<td>106</td>
<td>232</td>
<td></td>
<td></td>
</tr>
<tr>
<td>TABLE VII</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Distribution of Brushing For The Whole Ante Natal Group Showing Incidence of Gingivitis Gravidarum, Russell's Index, Debris, Calculus and Oral Hygiene.</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>---</td>
<td>---</td>
<td>---</td>
<td>---</td>
<td>---</td>
<td>---</td>
<td>---</td>
</tr>
<tr>
<td>Frequency of Brushing per day.</td>
<td>No. in Group</td>
<td>No. with Ging.</td>
<td>% with Ging.</td>
<td>Russell's Debris</td>
<td>Calculus</td>
<td>Oral Hygiene</td>
</tr>
<tr>
<td>Less than 1 daily.</td>
<td>47</td>
<td>34</td>
<td>72.3</td>
<td>2.34</td>
<td>3.99</td>
<td>0.84</td>
</tr>
<tr>
<td>1 daily.</td>
<td>238</td>
<td>132</td>
<td>55.4</td>
<td>2.16</td>
<td>3.16</td>
<td>0.74</td>
</tr>
<tr>
<td>2 or more daily.</td>
<td>399</td>
<td>167</td>
<td>41.8</td>
<td>1.86</td>
<td>2.41</td>
<td>0.58</td>
</tr>
<tr>
<td>Total:</td>
<td>684</td>
<td>333</td>
<td>48.7</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

| TABLE VIII |
| Distribution of Brushing for Patients With Gingivitis Gravidarum. |
|---|---|---|---|---|---|
| Frequency of Brushing per day | No. in Group | Russell's | Debris | Calculus | Oral Hygiene |
| Less than 1 daily | 34 | 2.86 | 4.43 | 1.01 | 5.54 |
| 1 daily | 132 | 2.38 | 3.51 | 0.89 | 4.44 |
| 2 or more daily | 167 | 2.44 | 2.77 | 0.58 | 3.40 |
| Total: | 333 | | | | |

---

113
### TABLE IX

**Distribution of Brushing for Patients Without**

**Gingivitis Gravidarum.**

<table>
<thead>
<tr>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>less than 1. daily.</td>
<td>13</td>
<td>0.99</td>
<td>2.87</td>
<td>0.41</td>
<td>3.28</td>
</tr>
<tr>
<td>1. daily.</td>
<td>106</td>
<td>1.25</td>
<td>2.72</td>
<td>0.54</td>
<td>3.28</td>
</tr>
<tr>
<td>2 or more daily.</td>
<td>232</td>
<td>1.45</td>
<td>2.15</td>
<td>0.53</td>
<td>2.71</td>
</tr>
<tr>
<td>TOTAL:</td>
<td>351</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Gingivitis gravidarum was more prevalent amongst those who brushed their teeth less frequently, but, never-the-less, there were some whose brushing was very irregular, (less than once a day), and still did not show any evidence of the condition. Generally, the gingivitis gravidarum was least amongst those who regularly brushed their teeth twice a day or more. The few patients encountered with a Russell score of zero or close to it all came into this category. It was found that those who brush twice or more daily, and who comprise the largest group, exhibit the lowest incidence of gingivitis gravidarum. Those who brush less than once a day, and who comprise the smallest group
exhibit the greatest.

There is a statistical significance in the observed frequencies of the distribution of brushing, showing that this is an important factor.

From my observations it became apparent that the majority of these women had no conception of the need for adequate brushing carried out with a correct technique, and at the right time. Some of the more indigent gave as a reason for not brushing, their lack of funds to purchase tooth paste, others claimed they did not want to injure their few remaining teeth by wearing them out with brushing. Still others, with badly broken down teeth, anticipated having the remaining ones extracted, and so could not see the need for good oral hygiene. This attitude was prevalent amongst those who exhibited a lower standard of education and an apparent substandard social background. It should be realised that, while this clinic imposes a means test, it is available to all unmarried pregnant women. In this group could be seen the extremes — those with a good social background — good education and good oral training, and those who had been inmates of institutions, or living away from home, with lack of family care and interest. (As evidenced by young girls with anterior teeth missing, and no attempt made to restore them.) These girls exhibited a lack of education and a general lack of personal cleanliness. Where some had been committed to an institution, and examined again later, there was
generally an improvement in their oral hygiene.

**Nationality.**

The division of patients into national groups gave rise to some interesting observations. The groups were as follows:

a) Australasian — including New Zealanders. (All of European extraction.)
b) Italian
c) Greek
d) Maltese
e) United Kingdom
f) Middle Eastern
g) Other European
h) Miscellaneous — (those who did not fall into the above categories, including Australian born Chinese and part Aborigine).

The Italian, Maltese and Greek showed the highest incidence of *gingivitis gravidarum.* (See Table X.) This coincided with the fact that the greater portion of these people are in the habit of brushing only once a day. Compare this with the Australasian group, in which the greater number brushed twice a day or more. The Australasian group exhibited a much smaller percentage with *gingivitis gravidarum,* had the lowest Russell's Index, and the
lowest Oral Hygiene Index,

<table>
<thead>
<tr>
<th>Nationality</th>
<th>No. in Group</th>
<th>No. with Gingivitis Gravidarum</th>
<th>Percentage</th>
</tr>
</thead>
<tbody>
<tr>
<td>Australasian</td>
<td>456</td>
<td>191</td>
<td>41.9%</td>
</tr>
<tr>
<td>Italian</td>
<td>31</td>
<td>24</td>
<td>77.4%</td>
</tr>
<tr>
<td>Greek</td>
<td>50</td>
<td>36</td>
<td>72.3%</td>
</tr>
<tr>
<td>Maltese</td>
<td>28</td>
<td>21</td>
<td>75.0%</td>
</tr>
<tr>
<td>United Kingdom</td>
<td>34</td>
<td>16</td>
<td>47.1%</td>
</tr>
<tr>
<td>Middle Eastern</td>
<td>12</td>
<td>7</td>
<td>58.3%</td>
</tr>
<tr>
<td>Other European</td>
<td>63</td>
<td>34</td>
<td>53.9%</td>
</tr>
<tr>
<td>Miscellaneous</td>
<td>10</td>
<td>4</td>
<td>40.0%</td>
</tr>
<tr>
<td><strong>Total:</strong></td>
<td><strong>684</strong></td>
<td><strong>333</strong></td>
<td><strong>48.7%</strong></td>
</tr>
</tbody>
</table>

The following Tables – XI., XII., and XIII., show the relationship between nationality, Russell's, Debris, Calculus and Oral Hygiene Indexes, with regard to the number and percentage with Gingivitis Gravidarum. Tables XIV., XV., and XVI., deal with the National groups at their first examination, showing distribution of brushing.
**TABLE XI.**

National Groups of all Ante Natal Patients, showing Russell's, Debris, Calculus and Oral Hygiene Indexes.

<table>
<thead>
<tr>
<th>Nationality</th>
<th>No. in Group</th>
<th>Russell's</th>
<th>Debris</th>
<th>Calculus</th>
<th>Oral Hygiene</th>
</tr>
</thead>
<tbody>
<tr>
<td>Australasian</td>
<td>468</td>
<td>1.75</td>
<td>2.63</td>
<td>0.57</td>
<td>SD 3.2</td>
</tr>
<tr>
<td></td>
<td></td>
<td>SD 1.493</td>
<td></td>
<td></td>
<td>SE + 0.070</td>
</tr>
<tr>
<td></td>
<td></td>
<td>SE + 0.070</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Italian</td>
<td>31</td>
<td>2.89</td>
<td>3.72</td>
<td>0.93</td>
<td>4.7</td>
</tr>
<tr>
<td></td>
<td></td>
<td>SD 1.606</td>
<td></td>
<td></td>
<td>SD 1.786</td>
</tr>
<tr>
<td></td>
<td></td>
<td>SE + 0.288</td>
<td></td>
<td></td>
<td>SE + 0.321</td>
</tr>
<tr>
<td>Greek</td>
<td>50</td>
<td>2.32</td>
<td>2.96</td>
<td>0.53</td>
<td>3.44</td>
</tr>
<tr>
<td></td>
<td></td>
<td>SD 1.507</td>
<td></td>
<td></td>
<td>SD 1.614</td>
</tr>
<tr>
<td></td>
<td></td>
<td>SE + 0.213</td>
<td></td>
<td></td>
<td>SE + 0.228</td>
</tr>
<tr>
<td>Maltese</td>
<td>23</td>
<td>2.51</td>
<td>3.68</td>
<td>1.22</td>
<td>4.9</td>
</tr>
<tr>
<td></td>
<td></td>
<td>SD 1.809</td>
<td></td>
<td></td>
<td>SD 2.048</td>
</tr>
<tr>
<td></td>
<td></td>
<td>SE + 0.342</td>
<td></td>
<td></td>
<td>SE + 0.387</td>
</tr>
<tr>
<td>United Kingdom</td>
<td>34</td>
<td>2.11</td>
<td>2.92</td>
<td>0.85</td>
<td>3.75</td>
</tr>
<tr>
<td></td>
<td></td>
<td>SD 1.354</td>
<td></td>
<td></td>
<td>SD 1.648</td>
</tr>
<tr>
<td></td>
<td></td>
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<td></td>
<td></td>
<td>SE + 0.281</td>
</tr>
<tr>
<td>Middle Eastern</td>
<td>12</td>
<td>2.25</td>
<td>2.96</td>
<td>0.43</td>
<td>3.39</td>
</tr>
<tr>
<td></td>
<td></td>
<td>SD 2.57</td>
<td></td>
<td></td>
<td>SD 1.938</td>
</tr>
<tr>
<td></td>
<td></td>
<td>SE + 0.742</td>
<td></td>
<td></td>
<td>SE + 0.559</td>
</tr>
<tr>
<td>Other European</td>
<td>63</td>
<td>2.63</td>
<td>2.76</td>
<td>0.69</td>
<td>3.45</td>
</tr>
<tr>
<td></td>
<td></td>
<td>SD 1.75</td>
<td></td>
<td></td>
<td>SD 1.104</td>
</tr>
<tr>
<td></td>
<td></td>
<td>SE + 0.22</td>
<td></td>
<td></td>
<td>SE + 0.139</td>
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<tr>
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<td>10</td>
<td>2.2</td>
<td>3.48</td>
<td>1.15</td>
<td>4.65</td>
</tr>
<tr>
<td></td>
<td></td>
<td>SD 1.592</td>
<td></td>
<td></td>
<td>SD 1.819</td>
</tr>
<tr>
<td></td>
<td></td>
<td>SE + 0.503</td>
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<td></td>
<td>SE + 0.594</td>
</tr>
<tr>
<td>Total:</td>
<td>684</td>
<td>2.0</td>
<td>2.79</td>
<td>0.65</td>
<td>3.43</td>
</tr>
<tr>
<td></td>
<td></td>
<td>SD 1.09</td>
<td></td>
<td></td>
<td>SD 1.985</td>
</tr>
<tr>
<td></td>
<td></td>
<td>SE + 0.041</td>
<td></td>
<td></td>
<td>SE + 0.076</td>
</tr>
</tbody>
</table>

SD = Standard Deviation.  
SE = Standard Error.
TABLE XII

National Groups With Gingivitis Gravidarum, Showing
Russell’s, Debris, Calculus and Oral Hygiene Indexes.

<table>
<thead>
<tr>
<th>Nationality</th>
<th>No. in Group</th>
<th>Russell’s</th>
<th>Debris</th>
<th>Calculus</th>
<th>Oral Hygiene</th>
</tr>
</thead>
<tbody>
<tr>
<td>Austral-asian</td>
<td>191</td>
<td>SD 1.389</td>
<td>2.45</td>
<td>3.15</td>
<td>0.69</td>
</tr>
<tr>
<td>Italian</td>
<td>24</td>
<td>SD 1.591</td>
<td>3.13</td>
<td>3.91</td>
<td>1.04</td>
</tr>
<tr>
<td>Greek</td>
<td>36</td>
<td>SD 1.55</td>
<td>2.68</td>
<td>2.88</td>
<td>0.63</td>
</tr>
<tr>
<td>Maltese</td>
<td>21</td>
<td>SD 1.837</td>
<td>2.69</td>
<td>4.09</td>
<td>1.4</td>
</tr>
<tr>
<td>United Kingdom</td>
<td>16</td>
<td>SD 0.963</td>
<td>2.43</td>
<td>3.45</td>
<td>0.87</td>
</tr>
<tr>
<td>Middle Eastern</td>
<td>7</td>
<td>SD 1.27</td>
<td>3.2</td>
<td>2.49</td>
<td>0.38</td>
</tr>
<tr>
<td>Other European</td>
<td>34</td>
<td>SD 1.589</td>
<td>3.31</td>
<td>2.97</td>
<td>0.83</td>
</tr>
<tr>
<td>Miscellaneous</td>
<td>4</td>
<td>SD 1.992</td>
<td>3.62</td>
<td>3.83</td>
<td>1.41</td>
</tr>
<tr>
<td>Total:</td>
<td>333</td>
<td>SD 1.48</td>
<td>2.65</td>
<td>3.23</td>
<td>0.3</td>
</tr>
</tbody>
</table>

SD = Standard Deviation  
SE = Standard Error
<table>
<thead>
<tr>
<th>Nationality</th>
<th>No. in Group</th>
<th>Russell's</th>
<th>Debris</th>
<th>Calculus</th>
<th>Oral Hygiene</th>
<th>SD</th>
<th>SE</th>
</tr>
</thead>
<tbody>
<tr>
<td>Australasian</td>
<td>265</td>
<td>1.24</td>
<td>2.26</td>
<td>0.49</td>
<td>2.77</td>
<td>SD</td>
<td>SE</td>
</tr>
<tr>
<td>Italian</td>
<td>7</td>
<td>2.07</td>
<td>3.04</td>
<td>0.76</td>
<td>3.8</td>
<td>SD</td>
<td>SE</td>
</tr>
<tr>
<td>Greek</td>
<td>14</td>
<td>1.39</td>
<td>3.14</td>
<td>0.42</td>
<td>3.57</td>
<td>SD</td>
<td>SE</td>
</tr>
<tr>
<td>Maltese</td>
<td>7</td>
<td>1.95</td>
<td>2.45</td>
<td>0.66</td>
<td>3.11</td>
<td>SD</td>
<td>SE</td>
</tr>
<tr>
<td>United Kingdom</td>
<td>10</td>
<td>1.83</td>
<td>2.44</td>
<td>0.8</td>
<td>5.24</td>
<td>SD</td>
<td>SE</td>
</tr>
<tr>
<td>Middle Eastern</td>
<td>5</td>
<td>0.87</td>
<td>3.59</td>
<td>0.5</td>
<td>4.1</td>
<td>SD</td>
<td>SE</td>
</tr>
<tr>
<td>Other European</td>
<td>29</td>
<td>2.00</td>
<td>2.5</td>
<td>0.52</td>
<td>3.93</td>
<td>SD</td>
<td>SE</td>
</tr>
<tr>
<td>Miscellaneous</td>
<td>6</td>
<td>1.25</td>
<td>3.24</td>
<td>0.97</td>
<td>4.21</td>
<td>SD</td>
<td>SE</td>
</tr>
<tr>
<td>Total</td>
<td>351</td>
<td>1.37</td>
<td>2.53</td>
<td>0.53</td>
<td>2.91</td>
<td>SD</td>
<td>SE</td>
</tr>
</tbody>
</table>

SD = Standard Deviation
SE = Standard Error
### National Groups of all Ante Natal Patients, Showing

#### The Distribution of Brushing

#### Distribution of Brushing

<table>
<thead>
<tr>
<th>Nationality</th>
<th>No. in group</th>
<th>Less than 1 daily No.</th>
<th>Percent</th>
<th>Once No.</th>
<th>Daily Percent</th>
<th>Twice or more daily No.</th>
<th>Percent</th>
</tr>
</thead>
<tbody>
<tr>
<td>Australasian</td>
<td>456</td>
<td>25</td>
<td>5%</td>
<td>136</td>
<td>30%</td>
<td>295</td>
<td>65%</td>
</tr>
<tr>
<td>Italian</td>
<td>31</td>
<td>7</td>
<td>22.5%</td>
<td>16</td>
<td>51.3%</td>
<td>8</td>
<td>26%</td>
</tr>
<tr>
<td>Greek</td>
<td>50</td>
<td>4</td>
<td>8%</td>
<td>30</td>
<td>60%</td>
<td>16</td>
<td>32%</td>
</tr>
<tr>
<td>Maltese</td>
<td>28</td>
<td>6</td>
<td>21.5%</td>
<td>16</td>
<td>57%</td>
<td>6</td>
<td>21.5%</td>
</tr>
<tr>
<td>United Kingdom</td>
<td>34</td>
<td>2</td>
<td>6%</td>
<td>8</td>
<td>24%</td>
<td>24</td>
<td>70%</td>
</tr>
<tr>
<td>Middle Eastern</td>
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<td>0</td>
<td></td>
<td>6</td>
<td>50%</td>
<td>6</td>
<td>50%</td>
</tr>
<tr>
<td>Other European</td>
<td>63</td>
<td>2</td>
<td>3%</td>
<td>22</td>
<td>35%</td>
<td>39</td>
<td>62%</td>
</tr>
<tr>
<td>Miscellaneous</td>
<td>10</td>
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<td>10%</td>
<td>4</td>
<td>40%</td>
<td>5</td>
<td>50%</td>
</tr>
<tr>
<td><strong>Total</strong></td>
<td><strong>684</strong></td>
<td><strong>47</strong></td>
<td><strong>7%</strong></td>
<td><strong>238</strong></td>
<td><strong>35%</strong></td>
<td><strong>399</strong></td>
<td><strong>58%</strong></td>
</tr>
</tbody>
</table>
## TABLE XV

National Groups With Gingivitis Gravidarum

**Showing Distribution of Brushing**

<table>
<thead>
<tr>
<th>Nationality</th>
<th>No. in Group</th>
<th>Less than 1 daily No.</th>
<th>Less than 1 daily Percent</th>
<th>Once Daily No.</th>
<th>Once Daily Percent</th>
<th>Twice or more daily No.</th>
<th>Twice or more daily Percent</th>
</tr>
</thead>
<tbody>
<tr>
<td>Australasian</td>
<td>191</td>
<td>19</td>
<td>10%</td>
<td>63</td>
<td>33%</td>
<td>109</td>
<td>57%</td>
</tr>
<tr>
<td>Italian</td>
<td>24</td>
<td>6</td>
<td>25%</td>
<td>14</td>
<td>58%</td>
<td>4</td>
<td>17%</td>
</tr>
<tr>
<td>Greek</td>
<td>36</td>
<td>4</td>
<td>11%</td>
<td>22</td>
<td>61%</td>
<td>10</td>
<td>28%</td>
</tr>
<tr>
<td>Maltese</td>
<td>21</td>
<td>4</td>
<td>19%</td>
<td>11</td>
<td>52%</td>
<td>6</td>
<td>29%</td>
</tr>
<tr>
<td>United Kingdom</td>
<td>16</td>
<td>0</td>
<td>-</td>
<td>4</td>
<td>25%</td>
<td>12</td>
<td>75%</td>
</tr>
<tr>
<td>Middle Eastern</td>
<td>7</td>
<td>0</td>
<td>-</td>
<td>3</td>
<td>43%</td>
<td>4</td>
<td>57%</td>
</tr>
<tr>
<td>Other European</td>
<td>34</td>
<td>1</td>
<td>3%</td>
<td>13</td>
<td>38%</td>
<td>20</td>
<td>59%</td>
</tr>
<tr>
<td>Miscellaneous</td>
<td>4</td>
<td>0</td>
<td>-</td>
<td>2</td>
<td>50%</td>
<td>2</td>
<td>50%</td>
</tr>
<tr>
<td><strong>Total:</strong></td>
<td>333</td>
<td>34</td>
<td>10%</td>
<td>132</td>
<td>40%</td>
<td>167</td>
<td>50%</td>
</tr>
</tbody>
</table>
TABLE XVI

National Groups Without Gingivitis Gravidarum,

Showing Distribution of Brushing

<table>
<thead>
<tr>
<th>Nationality</th>
<th>No. in Group</th>
<th>Less than 1 daily No.</th>
<th>Percent</th>
<th>Once Daily No.</th>
<th>Percent</th>
<th>Twice or more daily No.</th>
<th>Percent</th>
</tr>
</thead>
<tbody>
<tr>
<td>Australasian</td>
<td>265</td>
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<td>2%</td>
<td>73</td>
<td>28%</td>
<td>186</td>
<td>70%</td>
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<td>Italian</td>
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<td>1</td>
<td>14%</td>
<td>2</td>
<td>29%</td>
<td>4</td>
<td>57%</td>
</tr>
<tr>
<td>Greek</td>
<td>14</td>
<td>0</td>
<td>-</td>
<td>8</td>
<td>57%</td>
<td>6</td>
<td>43%</td>
</tr>
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<td>Maltese</td>
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<td>2</td>
<td>29%</td>
<td>5</td>
<td>71%</td>
<td>0</td>
<td>-</td>
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<td>11%</td>
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<td>67%</td>
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<td>-</td>
<td>3</td>
<td>60%</td>
<td>2</td>
<td>40%</td>
</tr>
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<td>Other European</td>
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<td>3%</td>
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<td>31%</td>
<td>19</td>
<td>66%</td>
</tr>
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<td>Miscellaneous</td>
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<td>1</td>
<td>17%</td>
<td>2</td>
<td>33%</td>
<td>3</td>
<td>50%</td>
</tr>
<tr>
<td>Total:</td>
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<td>13</td>
<td>4%</td>
<td>106</td>
<td>30%</td>
<td>232</td>
<td>66%</td>
</tr>
<tr>
<td>No. of Weeks</td>
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<td>13-16</td>
<td>17-20</td>
<td>21-24</td>
<td>25-28</td>
<td>29-32</td>
</tr>
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<td>-------</td>
<td>-------</td>
<td>-------</td>
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<td>-------</td>
</tr>
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<td>15</td>
<td>20</td>
<td>16</td>
<td>31</td>
<td>23</td>
</tr>
<tr>
<td>Italian</td>
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<td>1</td>
<td>0</td>
<td>3</td>
<td>5</td>
<td>4</td>
<td>2</td>
</tr>
<tr>
<td>Greek</td>
<td>0</td>
<td>3</td>
<td>3</td>
<td>4</td>
<td>6</td>
<td>1</td>
<td>5</td>
</tr>
<tr>
<td>Maltese</td>
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<td>2</td>
<td>3</td>
<td>3</td>
<td>3</td>
<td>3</td>
<td>3</td>
</tr>
<tr>
<td>United Kingdom</td>
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<td>2</td>
<td>3</td>
<td>1</td>
<td>3</td>
<td>3</td>
<td>0</td>
</tr>
<tr>
<td>Middle Eastern</td>
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<td>0</td>
<td>1</td>
<td>1</td>
<td>1</td>
<td>1</td>
<td>3</td>
</tr>
<tr>
<td>Other European</td>
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<td>1</td>
<td>1</td>
<td>4</td>
<td>6</td>
<td>3</td>
</tr>
<tr>
<td>Miscellaneous</td>
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<td>26</td>
<td>26</td>
<td>33</td>
<td>38</td>
<td>50</td>
<td>39</td>
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</table>
### TABLE XVIII

Distribution of National Groups Without Gingivitis Gravidarum According To Weeks of Gestation

<table>
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<tr>
<th>No. of Weeks</th>
<th>0-8</th>
<th>9-12</th>
<th>13-16</th>
<th>17-20</th>
<th>21-24</th>
<th>25-28</th>
<th>29-32</th>
<th>33-36</th>
<th>37-term</th>
</tr>
</thead>
<tbody>
<tr>
<td>Australasian</td>
<td>7</td>
<td>32</td>
<td>24</td>
<td>25</td>
<td>25</td>
<td>25</td>
<td>38</td>
<td>45</td>
<td>44</td>
</tr>
<tr>
<td>Italian</td>
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<td>2</td>
<td>0</td>
<td>1</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>1</td>
<td>0</td>
</tr>
<tr>
<td>Greek</td>
<td>0</td>
<td>2</td>
<td>3</td>
<td>2</td>
<td>2</td>
<td>1</td>
<td>0</td>
<td>1</td>
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</tr>
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<td>0</td>
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<td><strong>Total:</strong></td>
<td>11</td>
<td>40</td>
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<td>32</td>
<td>34</td>
<td>51</td>
<td>52</td>
<td>62</td>
<td>61</td>
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From Table XI it is seen that the Australasian Group had the lowest Russell Index in the whole ante natal group, and this coincided with the lowest Oral Hygiene score. A fairly close relationship is observed in this Table of higher Russell scores with higher Oral Hygiene scores. The Other European Group showed a higher Russell score than most, yet its Oral Hygiene Index is only 0.2 above the mean and it exhibited 53.9% with pregnancy gingivitis compared with the mean of 48.7%. The Italian, Greek and Maltese groups, had high Russell scores and were also those with the highest gingivitis gravidarum scores.

The mean Russell score for the whole ante natal group was 2.0 with an Oral Hygiene score 3.43. On dividing them into those with gingivitis gravidarum and those without, the Russell Index for the former was 2.65 with an Oral Hygiene score of 3.99. The latter's Russell Index score was 1.37 and the Oral Hygiene 2.91.

Table XIV shows the brushing habits of all the ante natal patients by division into national groups. As was shown earlier, gingivitis gravidarum was more prevalent amongst those who brushed less frequently, and the Italian, Greek and Maltese groups fall into this category. The majority of these people brushed once a day. As they had a tendency to brush less frequently than for example the Australasian group, they
would be more prone to gingivitis even without pregnancy. Combine this less frequent brushing with a food factor of a large ingestion of farinaceous material which many consumed, and there is an ideal combination for gingival irritation.

The Australasian and United Kingdom groups who were in the habit of brushing more frequently, were amongst those with the lowest incidence of pregnancy gingivitis, and they were the two groups with the lowest Russell scores. However, 62% of the Other European group brushed twice or more daily, and still had the second highest Russell score. This did not coincide with the pattern exhibited by the other groups. They did, however, have an incidence of gingivitis gravidarum not greatly in excess of the mean of the whole ante natal group. The groups who brushed more frequently were generally the ones who exhibited less gingivitis gravidarum, and the fact that certain groups exhibited much higher figures for it seemed to be directly attributable to their brushing habits.

In Table XVII and XVIII can be seen the distribution of each national group into its various weeks of gestation according to whether or not there was a gingivitis gravidarum present.

Post Natal Patients

The group of seventy five post natal patients was drawn from the Post Natal Clinic of the same hospital, and was
chosen at random. The same examination procedure was carried out for these patients as for the ante natal group, and the same scoring indexes used. Table XIX shows a comparison of the scoring indexes of the ante natal and post natal groups. The Russell Index for the post natal group was much lower than for the ante natal, yet the Debris and Oral Hygiene Indexes were much higher.

The Russell Index is not an indicator of increase or decrease of gingivitis gravidarum. Therefore it can only be stated that the post natal group exhibited less gingivitis than the ante natal group even with the presence of increased oral debris. This could imply that debris is not the only factor involved. Whether the absence of pregnancy in the post natal group is the cause is open to question and cannot be determined by the Russell score.

Of the sixteen patients seen both ante and post natally, twelve of these had been diagnosed ante nataly as having gingivitis gravidarum. Post nataly, ten showed an amelioration of the condition, and only two remained unchanged. Although these ten still exhibited some gingivitis, it was far less, and could not be described as a typical Ziskin-type gingivitis. It must be remembered that the oral habits of these patients had not altered — nor had they had any treatment. This was only six to seven weeks after parturition.
### Table XIX

Relationship of Ante Natal and Post Natal Group Showing

**Russell's, Debris, Calculus and Oral Hygiene Indexes**

<table>
<thead>
<tr>
<th>Number in Group</th>
<th>All Ante Natal Patients</th>
<th>Patients with Ging. Grav.</th>
<th>Patients without Ging. Grav.</th>
<th>Post Natal Group</th>
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<tr>
<td></td>
<td>604</td>
<td>233</td>
<td>371</td>
<td>75</td>
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<table>
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<th>Russell's Index</th>
<th>SD 1.09</th>
<th>SD 1.66</th>
<th>SD 1.65</th>
<th>SD 1.59</th>
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<tr>
<td>SE = 0.041</td>
<td>SE = 0.031</td>
<td>SE = 0.077</td>
<td>SE = 0.134</td>
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</tr>
<tr>
<td>Debris Index</td>
<td>2.79</td>
<td>3.23</td>
<td>2.33</td>
<td>5.45</td>
</tr>
<tr>
<td>Calculus Index</td>
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<td>0.80</td>
<td>0.53</td>
<td>0.57</td>
</tr>
<tr>
<td>Oral Hygiene Index</td>
<td>SD 1.935</td>
<td>SD 1.902</td>
<td>SD 1.902</td>
<td>SD 1.59</td>
</tr>
<tr>
<td>SE = 0.076</td>
<td>SE = 0.104</td>
<td>SE = 0.101</td>
<td>SE = 0.134</td>
<td></td>
</tr>
</tbody>
</table>

SE = Standard Deviation. SE = Standard Error.
INCIDENCE OF GINGIVITIS GRAVIDARUM ACCORDING TO:

- AGE
- WEEKS OF GESTATION

**Graph a:**

- Percentage With Gingivitis Gravidarum

Age in Years

0-12 13-20 21-28 29-36 37 and over

**Graph b:**

Percentage With Gingivitis Gravidarum

0 1 2 3 4 5 6 7 8

Weeks of Gestation

0-8 9-12 13-16 17-20 21-24 25-28 29-32 33-36 37 to term

**Figure III**
FIGURE V

DISTRIBUTION OF BRUSHING OF NATIONAL GROUPS

Brushing:
- less than once daily
- once daily
- twice or more daily

%}

Whole Group
Australasian
Italian
Greek
Multiple
United Kingdom
Middle Eastern
Other
European
Misc.
FREQUENCY OF BRUSHING OF NATIONAL GROUPS WITH GINGIVITIS GRAVIDARUM

W H O L E  A N T E  N A T A L  G R O U P

A U S T R A L A S I A N

Number of patients

Whole Brushing Group: Patients With Gingivitis Gravidarum

- less than once daily
- once daily
- twice or more daily

FIGURE VII
SCORING OF INDEXES FOR ANTE NATAL AND POST NATAL GROUPS

INDEX SCORES

All Ante Natal Patients With Gingivitis With Granuloma Without Gingivitis With Granuloma Post Natal

Russell's Calculus Debris Oral Hygiene

FIGURE XI
IV. SELECTED CASE HISTORIES.

1. Mrs. C., an Italian aged 32 years, was first seen at 28 weeks and was a primipara. She brushed her teeth twice daily and claimed that she had had no bleeding present, prior to about 12 weeks gestation.

When examined, she showed a marked hypertrophy around most of her teeth. All teeth were present. No calculus was visible with a light and none could be detected with a probe. The condition affected her mastication because of the bleeding, and there was some pain when food rubbed over the gums. From this period until delivery which was at 43 weeks, there was an increase of the hypertrophy both labially and lingually. This was more marked on the lower anteriors than elsewhere. Mrs. C. was seen several times during the pregnancy, and over the last weeks the bleeding became less as the enlargements increased.

The hypertrophy was not an isolated process, but was part of a generalised condition of proliferation. The margins of practically all the gingivae were swollen and hyperaemic and scarlet red in colour.

Generally, the patients at the hospital were not given dental treatment, but in this case, advice was sought on the
means of treatment. The patient was shown how to carry out improved gingival stimulation with a very soft tooth brush, but claimed that even this soft brush caused discomfort, although she did persevere.

Her mouth was examined five days after the birth, and at this stage showed some marked regression, although it was not complete. The bleeding had ceased completely, and there was no soreness.

Her medical history showed no serious illnesses, and her period of gestation, apart from being three weeks longer than usual, was uncomplicated. She was a normally developed woman, and well nourished, with, however, a national leaning toward a large amount of farinaceous material in her diet.

Because of this type of diet, and the inability to brush well, it is possible that these hypertrophied areas formed an ideal site for food impaction, and the collection of sordes which acted as constant irritants, with a resultant irritation of the condition.

2. Mrs. J., aged 19 years, was Australian and a primapara. She attended regularly for dental treatment and brushed her teeth three times daily. At her first examination at nine weeks, her Russell's Index was 0.25 with only
two upper first molars showing any periodontal condition.

When examined at twenty-five weeks her Russell's Index gave a score of 2.0 with a gingivitis around every tooth. There was considerable bleeding in the anterior region. Her Oral Hygiene Index had shown some increase. In both examinations there was no calculus detectable.

She was again examined at 37 weeks when there was a reduction in the Russell's Index to 1.75. All teeth still showed gingivitis, but the score on some teeth had dropped from 2 to 1, and there were fewer areas of bleeding.

3. Mrs. G., aged twenty-two years was an Australian and a primipara. She was seen at my surgery and attended regularly, every six months for treatment. Her brushing was twice daily.

When examined at sixteen weeks, she showed an oral condition no different from when examined before her pregnancy commenced. Her Russell's Index was 0.54 and her oral condition was very good. Although there were a few areas of gingivitis, there had been no bleeding from any of them.

When examined again at thirty-five weeks, there was bleeding from around the lower anterior teeth, the gingivae showing raspberry red gums. Her Oral Hygiene score remained
basically the same as at the previous examination.

4. Mrs. S., was also seen at my surgery. She was aged twenty-nine years, in her third pregnancy, and was Australian born. She attended for treatment every six to nine months. A jacket crown was present around the upper right lateral incisor.

When examined at thirteen weeks and thirty-three weeks, an inflammatory reaction had taken place around the gingival margin of the crown, and the area bled very easily. Although the crown had been in place for several years, it apparently did not cause any observable clinical irritation prior to the pregnancy. No other gingival area showed any reaction during the pregnancy. When examined four weeks post-natally, the inflammation had subsided and no bleeding was evident.

5. Mrs. A., an Australian was aged twenty-eight years. She was now in her eleventh pregnancy, her first child being born when she was sixteen. She only attended a dentist when she had pain. Her brushing was twice daily.

An examination was made at twenty-four weeks. The oral condition was poor, with roots remaining where the crowns of two teeth had decayed away. The Russell's Index was 3.05 and there was some gingivitis which appeared to be due to pregnancy super-
imposed on a long standing gingival disturbance.

At her next examination at 36 weeks, there was a deterioration of the condition, which now showed hypertrophy in areas which previously presented only bleeding or loss of stippling. The amount of bleeding had increased considerably. The patient stated that a similar occurrence took place with as many of her previous pregnancies as she could remember, and that post partum, the condition always disappeared.

6. Mrs. De. V., an Italian, primipara, aged twenty-two years, who brushed her teeth twice daily. Although under treatment with a dentist at present, she only sought treatment when necessary.

When examined at ten weeks, there was some gingivitis present, but none that could be classified as due to pregnancy.

A further examination took place at twenty-four weeks, when more than half of her teeth showed gingival changes. There was a combination of Ziskin Class 2, Class 3 and Class 4. Her Russell's Index had increased from 1.2 to 3.21 and her Oral Hygiene Index had increased only slightly.

Another examination was made at thirty-six weeks. The Russell's Index was 3.32 and there was a slight increase in the gingival changes in certain areas showing more proliferation,
whilst others had remained the same. The Oral Hygiene Index was basically unchanged.

Photographs of typical examples of gingivitis gravidarum amongst the patients examined are illustrated.

Included is one of a multipara in an advanced stage of gestation, showing a completely normal gingiva.
AUSTRALIAN AGED 21 YEARS, THIRD PREGNANCY, BRUSHING THREE TIMES A DAY. 33 WEEKS GESTATION. NORMAL GINGIVA.

AUSTRALIAN, AGED 25 YEARS, THIRD PREGNANCY, BRUSHING TWICE DAILY. 20 WEEKS GESTATION. BLEEDING AROUND LOWER LEFT FIRST & SECOND PREMOLARS. PRESENT OVER SEVERAL WEEKS.
MALTESE, AGED 39 YEARS, 24 WEEKS GESTATION, BRUSHING TWICE DAILY, POOR ORAL HYGIENE. ADVANCED PERIODONTITIS BLEEDING AROUND LOWER ANTERIORS, ONLY PRESENT OVER LAST SIX WEEKS.

ENGLISH, AGED 20 YEARS, PRIMAPARA, 36 WEEKS GESTATION, BRUSHING ONCE DAILY. SMALL EPULIS GRAVIDARUM BETWEEN FIRST AND SECOND PREMOLARS, PRESENT ONLY DURING PREGNANCY PATIENT WAS UNCERTAIN OF EXACT TIME OF OCCURRENCE.
AUSTRALIAN, AGED 24 YEARS, PRIMAPARA, 20 WEEKS GESTATION, BRUSHING ONCE DAILY. SWELLING AND OEDEMA, LOSS OF STIPPLING, MAINLY AROUND ANTERIOR TEETH WHICH HAD INCREASED MARKEDLY OVER PREVIOUS SIX WEEKS.

ITALIAN, AGED 19 YEARS, PRIMAPARA, 28 WEEKS GESTATION, BRUSHING LESS THAN ONCE DAILY. BLEEDING AROUND ANTERIOR TEETH AFTER COMMENCEMENT OF PREGNANCY.
AUSTRALIAN, PRIMAPARA, AGED 32 YEARS, TEN WEEKS GESTATION, BRUSHING TWICE DAILY. INFLAMMATION AROUND MOST OF THE ANTERIORS WITH INFLAMMATORY SWELLING BELOW LOWER LEFT LATERAL AND CUSPID. PRESENT TWO WEEKS.

AUSTRALIAN, AGED 21 YEARS, SECOND PREGNANCY, 18 WEEKS GESTATION, BRUSHING TWICE DAILY. "RASPBERRY RED" GUMS AROUND LOWER ANTERIORS. PRESENT FOR AT LEAST FOUR WEEKS.
ITALIAN, AGED 32 YEARS, PRIMAPARA, 32 WEEKS GESTATION, BRUSHING TWICE DAILY. MARKED HYPERPOTROPHY AROUND ALL TEETH. PRESENT THREE WEEKS.

SAME PATIENT, FORTY WEEKS GESTATION.
5. DISCUSSION

The oral tissues and teeth must be considered an intimate part of the human organism and the oral cavity is analogous to other body cavities with their associated organs. They are all governed by the same physiologic principles and the same physicochemical laws, with a common source of nutrition. As the oral tissues are in natural relation with the external environment and are subject to mechanical, chemical and bacterial insults that are rarely, if ever, experienced by other body cavities, they are unusually prone to disease.

These tissues are sensitive indicators of the general health status of the individual, and consequently changes in these structures are frequently the first indication of subclinical disease, processes or altered findings in other organ systems. This is particularly true of nutritional deficiencies, gastrointestinal disturbances, blood dyscrasias, certain anaemias and endocrine dysfunctions. (25)

This uniqueness of the tissues of the oral cavity have made it an ideal field for research and investigation.

In this study it was possible to examine a group of women in all stages of gestation at the Women's Hospital, Crown Street, Sydney. The use of a portable dental chair and a small spot light
allowed me easy access to all departments. As many of the patients lived in outlying districts, they attend other clinics after the initial examination and do not return to this hospital until the last few weeks or the time of the actual confinement. This meant that it was not possible to follow through with second and third visits of as many patients as I would have desired. Although I was anxious to examine post nataly many interesting ante natal patients, it was impossible to do so in most cases as, despite the routine appointment made for all confinements by the hospital, an extremely small number bother to attend, and the two cases of epulis were not seen post nataly to ascertain whether there had been regression or not.

The six hundred and eighty-four ante natal patients examined formed a sufficiently representative group to determine the prevalence of gingivitis gravidarum. It must be emphasized that these patients were not selected, but were taken at random and examined, as they presented themselves at the hospital, with the exception of those who came from my own surgery, and who were all the pregnant women who came for treatment during the same period. No patient was rejected unless she was edentulous or possessed less than six teeth.

Most patients were apathetic towards dental examinations, particularly those with poor oral health and of those who were examined more than once, very few sought the treatment which had
been advised. In many cases, where I suggested that the patient consult her own dentist, I was informed later by the patient, that the practitioner was unwilling to treat her during her pregnancy. This unfortunate attitude from some members of the profession shows a complete lack of understanding of the condition and needs to be overcome.

Difficulty was sometimes experienced in obtaining all the information required where the migrant women did not have a full command of English. However, they were often accompanied by the husband or a friend who could interpret, and at other times an interpreter was available from the hospital staff.

The classification by Ziskin and Nesse\(^{(134)}\) is possibly the best method available for identification of the various gingival disturbances in pregnancy, but it is by no means the ideal. Too much emphasis must be placed on the patient's information as to her gingival condition prior to pregnancy, and too much is left to the examiner's interpretation of any changes which may have taken place. There is no accurate means of measuring actual gingival change such as colour changes, commencement or increase in bleeding, or changes in volume. All gingival disturbances seen during the survey were covered by Ziskin's classification. The Class 5 of Ziskin and Nesse, which is an epulis, was retained, although Tiilila\(^{(111)}\) does not agree that a typical epulis gravidarum is an exaggeration of gingivitis gravidarum.

I have discussed epulis under a separate section, but never
the less I have retained it as Class 5 for the purpose of comparison in this investigation. Because of the low prevalence of epulis gravidarum, percentages are misleading, and an impossibly larger number of pregnant women would be needed to get a true picture of the incidence. This is evidenced by Amies, who examined 1,700 pregnant women in all stages and did not find even one true case of epulis.

The gingivitis gravidarum encountered in these patients ranged from the localization of inflammation of a few gingival papillae, lack of stippling, raspberry red gums, generalised hypertrophy and finally to epulis. The prevalence of hypertrophic gingivitis did not appear to be as high in this survey as in some, such as Ziskin, who found 70% hypertrophic of all patients affected. Hilming, noted a lower percentage. As a hypertrophic gingivitis in non-pregnant women may continue into pregnancy, one is not justified in diagnosing all such conditions as arising from pregnancy without further investigation.

Russell’s Periodontal Index, whilst being suitable for determining the gingival state, was only partly effective in showing gingival changes in pregnancy. If a high score already existed around a tooth, there was no method of indicating a superimposed inflammation or hypertrophy. It was quite effective in comparing the gingival condition of the various groups and the ante natal and post natal groups.
The score for the Russell Index is heavily weighted for periodontal breakdown, but as gingivitis gravidarum does not affect the deeper tissues, this scoring method is only partially effective. It is necessary to differentiate between gingivitis in pregnancy and gingivitis attributable to pregnancy.

The examiner, in an investigation of this type is faced with five types of patients, namely: a) those who, prior to pregnancy evinced no signs of gingival disturbance, and who, during the pregnancy, showed no change, b) those who evinced no signs of gingival disturbance before pregnancy, and who developed a gingivitis during its course, c) those with a gingivitis before pregnancy, and showed no change during gestation, d) those with a gingivitis before pregnancy, and who showed a change in gingival condition during its course, and e) those with a gingivitis before pregnancy and who showed some special gingivitis during pregnancy.

To diagnose these conditions accurately, the examiner should have access to the patients prior to pregnancy. It can readily be appreciated that in a large scale investigation, this is an impossible prerequisite. What is needed is some means of actual measurement or a series of standardised photography, or, in short, a method which will show even small macroscopic changes. A patient may show a Ziskin Class 1 or 2 condition, for example, at her first visit, and at a later examination, may show an increase which still lies within the same
classification. Using the scoring of Russell or Ziskin there is no way of indicating this.

The Debris, Calculus and Oral Hygiene Indexes were a good indication of the oral state. Generally, the Oral Hygiene Index was related to the Periodontal Index. This was not true for the post natal group, where the Russell Index was lower, but the Oral Hygiene score was higher.

The number of pregnancies and the age of the mother had no bearing and gave no indication of being an etiological cause. This is confirmed by Gompertz, (43) and Mugnier. (78) This can also be compared with the work of Schour and Massler (100) in a survey of a non-pregnant group of 270 nurses, twenty to thirty-four years old. They found that there was no significant increase in the amount of gingivitis with an increase in age.

Ringsdorf, Powell, Knight and Cheraskin, (91) have shown that the twenty-five to thirty year olds have significantly less bleeding than the younger group. Erb and Brzezinsky, (31) found however, that their greatest prevalence was amongst the twenty-six to thirty year old of Israeli-born women, and twenty-one to twenty-five year old group among the immigrants.

Whilst it is not possible to state the time of onset of gingivitis in pregnancy, as it appears to vary greatly in each individual, it was observed in those examined more than once,
that there was a notable increase during gestation, and many exhibited amelioration in the ninth month. This has also been noted by Hilming.\(^{(55)}\)

The direct relationship between the frequency of brushing and the incidence of gingivitis gravidarum shows that the condition can be minimised by better oral hygiene. This alone is unlikely to prevent the condition, but it could be a major factor in controlling its severity. The brushing techniques of most of the patients left much to be desired.

Whilst some national groups exhibited a higher percentage of the condition, it did not appear to be due to any inherent physical condition. Rather was it a combination of poor oral hygiene and ingestion of a highly farinaceous diet. These particular people had not been in the country very long. Horsnell and Packer,\(^{(57a)}\) however, found no significant difference in the Periodontal or Oral Hygiene Indexes between Australians and those of European origin. This was probably due to their selection of patients as they excluded those with limited English.

Russell,\(^{(94)}\) has shown that periodontal disease, once present in an individual, is conditioned by his social background. People with better educational attainments tend to exhibit periodontal disease in the milder stages. This was also my observation in the course of examination with most of the patients from all
countries including Australia. The Italians, Maltese and Greeks exhibited a high percentage of gingivitis gravidarum and most of them came from the poorest areas of their countries.

Fraser (36) also noted a high incidence of the condition in the lower economic group.

From the patients studied and from the literature available, nutritional and vitamin deficiencies can generally be ruled out as a cause of the condition. It also appears that most local factors can be discounted as the only cause. Since the condition usually improves so rapidly after parturition without any treatment being carried out, and without any extra care being exercised by the patient, one must look for systemic causes, possibly hormonal, particularly as the local conditions remain the same except that the body has reverted to its non-gravid state.

A condition of gingivitis gravidarum has been discussed throughout this thesis and the patients divided into groups of those with and without the condition even though the ideal method of diagnosis and measurement has not been found.

At the present time the initiating cause of changes which often take place in the oral cavity during pregnancy is still uncertain. Although the results showed 48.7% of the women examined had gingivitis gravidarum, a more objective form of accurate diagnosis and measurement could give a different result.
It is practically impossible to differentiate between gingivitis of pregnancy and a simple gingivitis without the knowledge that the patient is pregnant or not.

Notwithstanding the limitations of the methods of diagnosis and measurement of gingival changes, a clinical impression remains that changes do take place in the oral cavity during the period of gestation, even though the assessment cannot be regarded as completely accurate. However, it is only to this extent that one is justified in saying that a gingivitis of pregnancy exists since there are no specific clinical and histopathologic changes in the gingiva which are exclusive to pregnancy.
SUMMARY AND CONCLUSION.

1. The subject of gingivitis in pregnancy has been discussed by many over the years. The conflicting thoughts regarding its etiology makes one realise that there is enormous scope for research in order to obtain more conclusive results.

2. An epidemiological study of six hundred and eighty four pregnant women and seventy five post natal patients was carried out in Sydney, and showed that there was a higher incidence of gingivitis in the pregnant group when scored by the Russell Periodontal Index.

3. The Russell Periodontal Index, whilst indicating the periodontal status of the gingival state of patients, is heavily weighted for more advanced periodontal disease and is not an adequate scoring method for assessing gingival changes during pregnancy.

4. Until a more accurate means of diagnosing and scoring the gingival changes during pregnancy is found, and until a means is devised whereby groups of women could be examined before, during and after pregnancy, the present methods of measurement must be used, despite their shortcomings.
5. The observed clinical changes diagnosed as gingivitis gravidarum are not unique to pregnancy and can also be observed in the non-gravid state.

6. In the course of this survey of six hundred and eighty-four pregnant women, three hundred and thirty-three were diagnosed as having gingivitis gravidarum according to Ziskin’s classification. As the diagnosis relies on information obtained from the patient, and leans heavily on the examiner’s opinion as to what changes have occurred, these figures must be treated with some caution.

7. The Russell Periodontal Index of the 684 ante natal group was 2.00 and of these only 31 exhibited a score of zero. The post natal group of 75 had an Index score of 1.49.

8. In those diagnosed as gingivitis gravidarum, no statistical significance could be attributed to age, number of pregnancies or weeks of gestation, although there was some diminution of the condition in the ninth month.

9. The frequency of brushing was directly related to the incidence of the gingivitis, showing that those who brushed more frequently were less inclined to suffer from the condition.
The Greek, Maltese and Italian groups exhibited the highest incidence of gingivitis and these were the groups which brushed less frequently.

10. No clinical test was carried out as to the part played by hormonal changes during pregnancy, but a full review of the pertinent literature has been included.

11. There were no obvious cases of nutritional deficiency amongst these patients, but the highly farinacous diet of some groups did appear to cause irritation due to deposited debris.

12. The importance of local factors in gingivitis in pregnancy cannot be minimised but the role played by systemic disturbances due to changes in hormonal balance must be considered. Obviously, no single factor is responsible for the onset and continuance of this condition. As the result of an altered functional and regulatory mechanism of the body following the implantation of the ovum, it is possible that hormonal changes could cause exaggerated tissue changes in those mouths where local factors are already present.
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