BURNING MOUTH SYNDROME :
CLINICAL PRESENTATION AND MANAGEMENT

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DECLARATION

The material contained in this thesis is entirely original and the analysis of all data was undertaken by the author with the exception of the Cattell's 16PF Form C questionnaire which is licensed for use only by persons with a training in psychiatry or psychology.

Parts of the work of this study have been presented at scientific meetings and have been published in the following publications.

1. Abstracts of Papers Presented at Scientific Meetings

(i) Aetiological factors in burning mouth syndrome: A prospective study. Journal of Dental Research. 1987. 66. 851 (with P-J Lamey) ABSTRACT NO 150.

(ii) Psychological aspects of burning mouth syndrome. Journal of Dental Research. 1987. 66. 851 (with P-J Lamey and P E Reeve) ABSTRACT NO 151.

(iii) Microbiological and Glycaemic aspects of burning mouth syndrome. Journal of Dental Research. 1987. 66. 851 (with P-J Lamey, J Gibson. T W MacFarlane. L P Samaranayake and M A Boyle). ABSTRACT NO 152.

(iv) The burning lip component of burning mouth syndrome. Journal of Dental Research. 1989. 68. 1017 (with P-J Lamey and T Ferris) ABSTRACT NO 1205.

2. Papers

(i) Atypical burning mouth syndrome. Contact Dermatitis. 1987.17. 242-243 (with P-J Lamey and A Forsyth).

(ii) Prospective study of aetiological factors in burning mouth syndrome. British Medical Journal. 1988, 296, 1243-1246 (with P-J Lamey).

(iii) Burning mouth syndrome: psychological aspects. British Dental Journal. 1988, 165. 256-260 (with P-J Lamey and P E Reeve).

(iv) Oral carriage of Candida species and coliforms in patients with burning mouth syndrome. Journal of Oral pathology, 1989, 18, 233-235 (with L P Samaranayake, P-J Lamey and T W MacFarlane).

3. Book Chapter

Refractory burning mouth syndrome. In Non-responders in gastroenterology. Ed. G. Dobrilla New York: Raven Press. (with P-J Lamey) (Publication Date September 1990).

S U M M A R Y

A review of the literature on Burning Mouth Syndrome (BMS) revealed that many aetiological factors for the condition had been proposed. Nevertheless the relative role of individual factors had rarely been assessed and no prospective study previously had been reported. Therefore the aim of the study was to further understand the aetiology of BMS and to devise a treatment protocol. The design of the study specifically avoided the approach of other authors which had focused attention only on their particular specialist areas. As a result treatment was often of a fragmented nature with multiple referrals to practitioners in different specialties. The past literature was further confused by the inclusion of patients with specific conditions in which the patient may report a burning sensation, such as erosive lichen planus and geographic tongue. These conditions characterised by possible mucosal changes have their own specific treatment regimes and are distinct from BMS where patients have no visible oral changes. Based on a knowledge of the literature a prospective study of 150 patients with BMS was undertaken. Care was taken to ensure that no patients that had visible oral changes were included.

It became clear that patients with BMS gave a varied history and therefore did not constitute a homogenous group. The validity of recognising subtypes of BMS was evaluated as it could have prognostic implications. A classification system was introduced such

that patients who said their burning was absent on waking, developed as the day progressed and was maximal in the evening were termed Type I BMS. Others who reported burning which was present on waking and was constant throughout the day were termed Type 2 BMS. In both Type I and Type 2 BMS burning was unremitting being present every day. Less commonly patients reported variable burning which was not necessarily present each day, these patients being classified as Type 3 BMS. Thirty five per cent of patients were classified Type I, 54% Type 2 and 11% Type 3.

In order to quantify the severity of symptoms a linear analogue scale was employed. This allowed a baseline assessment of the severity of patients' initial symptoms and allowed progress during treatment to be quantified.

Other data collected and analysed included the oral site of burning, duration of symptoms, age, sex, sleep pattern, home and social circumstances and cancerphobia. Haematological investigations included serum ferritin, corrected whole blood folate, serum vitamin B12, blood film and haemoglobin. Biochemical investigations were undertaken to detect undiagnosed diabetes mellitus and for 67 patients assays for Vitamin B1, B2, B6 and Vitamins A, C, D and E were completed.

The oral flora was sampled and stimulated parotid salivary flow rates were estimated. Dentures, if worn, were assessed for factors contributing to overloading of the oral tissues in particular tongue restriction and

excessive vertical dimension were noted. Parafunctional activity was investigated.

Psychological aspects of BMS have tended to dominate the literature. In this study one third of patients were assessed using the Cattell's 16 PF Form C questionnaire. The results from this component of the study indicated that 60% of the sample had psychological abnormalities.

A structured treatment protocol was developed so that any detected abnormalities were managed appropriately. Using the linear analogue scale to assess outcome of treatment 60% of patients with BMS were cured and sustained improvement in symptoms was reported by a further 11% with a minimum follow up period of 18 months.

Whilst 54% of patients assessed with the Cattell's 16 PF Form C questionnaire as being psychologically disabled were cured compared to 68% of patients who were assessed as psychologically normal, those patients with a Type 2 history of BMS particularly when anxiety was present were significantly less likely to have a successful outcome. This finding supports the opinion that patients with BMS should not be regarded as a homogeneous group.

The results of the study have placed in context the relative importance of organic factors including haematological, biochemical, microbiological and prosthodontic elements and non-organic factors such as psychological status. For the first time a prospective analysis of a cohort group has permitted justification for

attention to be paid to all potential aetiological factors, since only 47% of patients cured were cured as a result of treatment of one factor alone. However, replacement dentures, management of parafunctional activity, vitamin B1 and vitamin B6 replacement therapy and the use of the tricyclic antidepressant, Dothiepen (Prothiaden) were shown to be the most useful in treatment. The precise role of all aetiological factors is fully discussed and the importance of taking cognisance of psychological, social and domestic circumstances and cancerphobia is stressed. Finally suggestions for future avenues of research are proposed.

GENERAL INTRODUCTION

Patients with Burning Mouth Syndrome (BMS) have previously been difficult to treat effectively and this has largely been due to lack of a prospective scientific approach to study. A clear picture of aetiological factors has not emerged in part due to authors frequently focusing attention on their own specialised area of clinical interest in the field. Consequently treatment was often unsatisfactory, resulting in a poor patient response which necessitated multiple referrals to practitioners in different specialities. In addition, the literature has caused further confusion by including references to patients with specific conditions which have a burning sensation of the oral cavity as a symptom. These conditions such as erosive lichen planus and geographic tongue are entirely distinct from BMS and are characterised by visible mucosal changes which have their own specific treatment regimes. On the other hand, patients with BMS have no visible oral changes, a feature which allows a diagnosis of BMS to be made. Much avoidable distress has resulted from multiple referrals to specialists who were not familiar with BMS as a separate disease entity and in whose patients the oral mucosa looks normal, the tendency has been to dismiss the condition as of no consequence. This can be particularly distressing to those patients who describe intolerable burning and as a result they, and their practitioners, believe that the symptoms must be psychological in origin and this aspect,

with some exceptions, has tended to dominate the literature.

The purpose of the present study was to review the state of current knowledge concerning aetiological factors in BMS. Using this information the first prospective study to investigate all known aetiological factors was devised so that a structured and effective management regime could be developed. In addition to providing successful therapy such a regime could be efficient with resources by utilising only appropriate investigations and avoiding unnecessary referrals. From the data collected global studies could also be undertaken to assess the frequency of given aetiological factors in specific populations.

Finally, by an understanding of the relative importance of the individual aetiological factors and assessing difficulties in management, problem areas which would form the basis of continuing research have been identified.

CHAPTER I

AETIOLOGICAL FACTORS IN BURNING MOUTH SYNDROME

1.1 INTRODUCTION

The symptom of a burning sensation affecting the mouth is widely recognised and is referred to in many standard textbooks of oral medicine (Dolby, 1975: Chisholm et al. 1978: Gayford and Haskell, 1979: Harris and Davies in Jones and Mason, 1980: Brightman in Lynch, 1984: Scully and Cawson, 1987), oral pathology (Pindborg, 1980: Shafer, Hine and Levy, 1983), and prosthodontics (Anderson and Storer, 1981: Watt and MacGregor, 1983: Hickey, Zarb and Bolender, 1985). However, a review of the world literature reveals that the complaint of a burning sensation of the oral mucosa is poorly researched and there are few papers in which a scientific approach to a study of the condition has been adopted and there have been no prospective studies.

Historically many diverse terms have been used when describing a burning sensation affecting the mouth. These terms include burning tongue, (Engman, 1920; Schroff, 1935; Fox, 1935: Ziskin and Moulton, 1946; Elfenbaum, 1969), glossodynia, (Ziskin and Moulton, 1946; Massler 1951: Kruger and Reynolds, 1965; Coles, 1966; Elfenbaum, 1969: Glick et al, 1976; Oles, 1979; Brightman

in Lynch, 1984), glossalgia, (Keil, 1970; Brightman in Lynch, 1984), glossopyrosis, (Birch, 1969; Elfenbaum, 1969; Dolby, 1975; Harris and Davies in Jones and Mason 1980), sore tongue, (Goss, 1973; Gayford and Haskell, 1979; Scully and Cawson, 1987), burning mouth (Richman and Abarbanel, 1943; Massler, 1951; Brody and Nesbitt, 1967; Schoenberg, 1967; Basker, Sturdee and Davenport 1978; Main and Basker, 1983; Zegarelli, 1983), stomatodynia, (Richman and Abarbanel, 1943), stomatopyrosis, (Schoenberg et al, 1971), oral dysaesthesia (Harris, 1975; Sutton, 1982; Scully and Cawson, 1987), and burning mouth syndrome, (Silverman, 1975; Grushka, 1983; Lamey et al, 1986). Not all authors in their description of the patients' complaint of a burning sensation affecting the oral mucosa in fact describe comparable conditions. The use of the term burning mouth syndrome relates only to patients who on clinical examination have an apparently healthy oral mucosa but when other terms are used patients who have an obvious abnormality of the oral mucosa are included since there are a number of oral conditions with which a complaint of burning is sometimes associated. Such conditions include desquamative gingivitis, erosive or atrophic lichen planus, benign migratory glossitis (geographic tongue) and occasionally chronic atrophic candidosis. These conditions are characterised by clinical and histopathological changes in the appearance of the oral mucosa and should be excluded when a diagnosis of burning mouth syndrome is proposed.

Factors which have been incriminated in the

aetiology of burning mouth syndrome include haematological deficiencies such as iron deficiency anaemia, vitamin B12 deficiency, folic acid deficiency, sideropenia, vitamin B complex deficiences, candidal infections, hormonal changes, endocrine abnormalities, salivary gland dysfunction, denture design discrepancies, allergies to foodstuffs and dentrificies, overuse of denture cleansers or mouthwashes and psychological factors.

These factors are dealt with in detail in the subsequent sections of this Chapter which is concluded by a discussion of the complex inter-relationship between many of them. Since more than one aetiological factor may be involved in the pathogenesis of the condition a structured approach to diagnosis and subsequent treatment is therefore necessary for individual patients.

1.2 SYMPTOMS ON PRESENTATION

An interesting feature of patients with burning mouth syndrome is that the symptoms on presentation are similar. Typically patients complain either of burning on waking which persists throughout the day or of a burning sensation which develops during the day and is maximal by evening. Disturbance with sleep is not a feature of burning mouth syndrome. These general statements have been derived from the literature which is now presented chronologically.

Engman (1920) reported a condition which he described as burning tongue. He studied nine patients, 11 of whom were female with no abnormality of the mucosa of the tongue, of note was that each of the patients had a

marked fear of cancer.

In another study of burning sensations of the tongue Schroff (1935) indicated that the tip and lateral aspects of the tongue were most commonly affected. The clinical condition of the patients' tongue was not discribed but the complaint was aggravated in some cases by hot, spicy or acid foods or liquids.

The description given by patients with a burning sensation of the tongue was investigated by Fox (1935). He indicated that patients' abnormal oral sensations were variously described as "burning", "itching", "boring", "stinging", "feeling of sand in the mouth", or "bad taste". The burning was usually continuous but did not interfere with sleep. There were no pathologic changes visible in the group of patients studied who were usually middle aged women who subjectively were said to have a neurotic tendency.

Ziskin and Moulton (1946) described the most frequent oral site affected as the tongue although any part of the mouth could be affected. The typical patient was the postmenopausal woman, unduly emotionally disturbed over her oral condition and complaining of nervousness, depression, hopelessness, insomnia or alluding to significant cancerphobia. The sensation was persistent and unbearable, even though it may have been present for years. Younger female patients rendered menopausal by surgical procedures and a few men were also affected, but the condition did not affect children. There were no visible oral lesions on examination with the

tongue and mucous membranes appearing normal. Frequently patients attributed their discomfort to some coexistent dental problem, for example the recent provision of dentures, sharp tooth surfaces or of margins of restorations or edentulous spaces which subconsciously attracted the tongue.

Hormonal aspects of a burning sensation of the mouth were studied by Massler (1951). That study reported the findings on 86 patients experiencing the climacteric and concluded that their burning sensation usually occurred at various sites including the side, tip and ventral surface of tongue, buccal mucosa and the faucal region. Cancerphobia was a conspicuous feature and significant numbers of patients either could not wear a recently constructed prosthetic appliance, or associated their symptoms with adjacent dental restorations.

Schoenberg (1967) reviewed the previous literature and agreed with the documented descriptions of the symptoms of burning but also indicated that in terms of severity patients might describe the burning sensation as intolerable. However the complaint was not physically incapacitating and typically the burning sensation increased in severity as the day progressed. As had been reported by Fox (1935), Schoenberg (1967) noted that the complaint did not interfere with sleep.

Harris (1975) expanded on the previous observations of other authors and added further comment on patients daily experience of the burning sensation. He reported that the burning may not be present on waking,

may be unilateral or bilateral and was often relieved by eating or drinking. This finding was in contrast to the report of Schroff (1935) but is consistent with symptoms given by patients who have physically evident conditions of the tongue such as geographic tongue which may lead to the complaint of burning but in which the burning sensation is usually made worse by eating and drinking. Fear of cancer was stated as the patient's greatest concern. More recently Grushka (1983) also stated that patients with a burning sensation of the mouth are not usually wakened from sleep with the complaint.

1.3 HAEMATOLOGICAL DEFICIENCY

It has been recognised that deficiency states such as sideropenia, iron deficiency anaemia, pernicious anaemia and folic acid deficiency can result in oral mucosal discomfort described as a burning sensation. The oral consequences of such deficiencies have been described fully by Hjorting-Hansen and Bertram (1968), Faccini (1968), Hardisty and Weatherall (1974), Dolby (1975), Brooke and Seganski (1977), Basker et al. (1978), Main and Basker (1983) and Zegarelli (1984). Collectively these studies concluded that if deficiencies were of sufficient duration visible changes could occur in the oral tissues but the complaint of a burning sensation could be present when no obvious mucosal changes were evident.

The mechanism by which these various deficiency states might give rise to burning symptoms is uncertain. Wintrobe et al (1981) suggested that in the case of sideropenia and iron deficiency anaemia that depletion of

iron dependent enzymes such as cytochrome oxidase could result in functional epithelial changes. In the case of vitamin B12 and folic acid deficiency, morphological changes in oral epithelial cells similar to changes described in bone marrow and red cell precursors have been observed.

Brooke and Seganski (1977) in their study stated that of 55 cases of patients complaining of oral discomfort but with no mucosal abnormality, 53% were iron deficient. 4% were folic acid deficient and 44% had no detectable haematological deficiency.

Basker et al. (1978) reported that the causative factors in 21 patients with the complaint of burning mouth included haematological deficiencies in 7 patients. Of this group, one patient was vitamin B12 deficient and 6 patients were deficient in folic acid. In 3 of these 7 patients (14%) the haematological deficiency was the sole cause of burning as judged by the clinical response to appropriate replacement therapy.

In a follow-up study, Main and Basker (1983) reported on a further 37 patients with burning mouth syndrome and found haematological deficiencies in almost 40% of the group. The deficiencies again identified were of folic acid, vitamin B12 and iron. In less than half these cases however were these deficiencies deemed to be prime causative factors in the burning sensation.

Zegarelli (1984) in his analysis of 57 patients complaining of a burning sensation of the oral mucosa reported only one patient who was nutritionally deficient.

This patient was subsequently shown to have pernicious anaemia. One criticism of this study however is the heterogenous nature of the group under study. Thus in many of these patients mucosal abnormalities such as geographic tongue and lichen planus were evident. These patients must therefore be excluded from those who are diagnosed as having burning mouth syndrome.

Deficiency in vitamins of the B complex group has also been thought to result in symptoms of burning mouth in some patients. Biskind (1946) showed that there was an intimate relationship between the vitamin B complex status and the level of oestrogens. Assay of vitamin B complex levels in these patients was however lacking. Massler (1951) suggested that the majority of postmenopausal women suffer from vitamin B complex deficiency but his study lacked quantitative data. In his series of 86 postmenopausal patients who presented with oral symptoms, including burning, approximately one-third responded quickly to vitamin B complex replacement therapy. Elfenbaum (1969) also suggests a possible link between what he described as glossodynia and vitamin B complex deficiency.

Lamey et al (1986) showed that in a group of 70 patients with burning mouth syndrome 28 patients were deficient in vitamin B1 B2 or B6 or a combination of these vitamins. In addition these patients had otherwise normal routine haematological parameters, normal stimulated parotid flow rates and no significant isolates of candidal species. In this group of patients 27 had

normal vitamin B complex profiles. Fifteen patients were, therefore, excluded from the study because of known aetiological factors being present. This study also compared the clinical response of patients to appropriate vitamin replacement therapy. Deficient and non-deficient patients were given vitamin B1 (300 mg per day), vitamin B2 (20 mg per day in two divided doses), or vitamin B6 (150 mg per day in three divided doses) but not in a double blind cross over trial. Eighty-eight per cent of patients with burning mouth syndrome and proven vitamin deficiency were asymptomatic at three months, whereas in the non deficient group no patients were asymptomatic and only 7% showed some improvement at three months.

1.4 MICROBIOLOGICAL ASPECTS

The pathogenic role of the oral flora in patients with the complaint of burning mouth syndrome has been poorly documented. Cahn (1936) used the term denture sore mouth to describe the diffusely inflammed mucosa seen under a maxillary complete denture although this condition is not usually accompanied by discomfort. Denture sore mouth occurs only rarely in the mucosa of the mandibular denture bearing area (Budtz-Jorgensen, 1974). Since few patients experience discomfort with this condition this term has largely been replaced by the terms denture stomatitis and chronic atrophic candidosis (Budtz-Jorgensen and Bertram, 1970 a & b; Jones, 1976).

Denture induced trauma, lack of denture hygiene and infection with Candida albicans or other yeast-like organisms have been reported as the main causative agents

in denture stomatitis (Cawson, 1963; 1966; Turrell, 1966a; Ritchie et al, 1969; Davenport, 1970; Budtz-Jorgensen, 1971; Allison and Douglas, 1973; Olsen, 1974; Budtz-Jorgensen, Stenderup and Grabowski, 1975; Gale et al, 1975; Budtz-Jorgensen, 1976). The fitting surface of the upper denture and not the palatal mucosa has been shown to be the predominant site of candidal colonization (Davenport, 1970; Olsen, 1974). Since erythematous change is a feature of denture stomatitis and not all patients experience discomfort, patients with this condition do not, by definition, have burning mouth syndrome.

Bartels and Blechman (1962) estimated the oral candidal carriage in subjects with a clinically normal healthy oral mucosa to be between 33% and 40% when mixed saliva was sampled. Budtz-Jorgensen and Bertram (1970a) showed that 40% of apparently healthy denture wearing controls carried yeast-like organisms when microbial sampling was carried out using an impression method. These figures are substantially higher than the 18% quoted by Turrell (1966a) in a review of four papers where the swab technique of candidal isolation was used. Williamson (1972) showed that non-denture wearing carriers of candidal species, with a clinically normal oral mucosa, had a diurnal variation in candidal count with an early morning peak, whereas denture wearing increased the candidal count and reversed the diurnal curve.

Arendorf and Walker (1980) compared the detection rates of Candida albicans carriage using imprint cultures, salivary samples, impression cultures and epithelial

smears. They investigated healthy non-denture wearing dentate patients and found that 44.4% carried Candida albicans using imprint culture, 29.6% using whole salivary samples and 13% using impression cultures. A similar detection rate was found when either the imprint culture technique or an epithelial smear method was employed. Females were more frequent carriers of candidal species than males and smokers predominated over non-smokers.

Arendorf and Walker (1979) used the imprint culture technique since, at that time, it was the most sensitive method of candidal carriage detection to compare the incidence of Candida albicans carriage in healthy denture wearers, patients with denture stomatitis and healthy dentate controls. Of healthy denture wearers, 55.6% carried candidal species and 100% of patients with denture stomatitis carried Candida albicans. Wearing dentures overnight was associated with increased candidal colonization and the likely development of denture stomatitis.

Samaranayake et al (1986) compared the imprint culture method of sampling of Arendorf and Walker (1980) and a method using a 60 second oral rinse of sterile phosphate buffered saline. They reported similar frequencies of Candida albicans carriage in healthy patients using the two methods but suggested that the oral rinse was simpler to perform, could also be used to detect carriage of Staphylococcus aureus and coliforms and was a satisfactory method to use in patients with xerostomia.

Brooke and Seganski (1977) reported that 7 of

their 55 patients complaining of burning mouth had candidal species isolated using the smear method.(13%). Zegarelli (1983) reported that 2 patients (3.5%) in his heterogeneous group of 57 burning mouth patients carried candidal species with no oral signs. Alteras & Cojocaru (1969) isolated Candida albicans in 10.5% of their 180 patients suffering from glossodynia. The significance of the carriage of yeasts in these studies is unclear since sampling methods were not recorded in two of the three reports and the carriage incidence was lower than that reported from the mouths of normal healthy individuals (Bartels and Blechman, 1962: Turrell, 1966a; Budtz-Jorgensen and Bertram, 1970a: Arendorf and Walker, 1979; 1980).

There are no reports of the oral carriage of organisms other than yeasts in patients with burning mouth syndrome.

1.5 CLIMACTERIC FACTORS

Ziskin and Moulton (1946) were the first to report that typically a patient suffering from the complaint of a burning mouth was postmenopausal. They confirmed that oestrogen supplementation in postmenopausal women increased the cornification of the oral mucosa but that it did not give sustained relief from symptoms. They concluded that psychological factors played a more important role.

Richman and Abarbanel (1943) reported that in their opinion there was an association between stomatodynia and xerostomia in climacteric patients. They also suggested that in addition to relief from the

characteristic hot flushes with suitable hormone replacement therapy (estradiol or diethlylstilbestrol), the oral symptoms also tended to disappear. To substantiate their hypothesis a study correlating visual gross signs in the oral cavity with subjective symptoms, and buccal mucosal biopsies was undertaken. Their findings suggested that oral symptoms did not appear until 6 months after cessation of menstruation. At that time the mucosa became pale pink, symptoms of burning or dryness appeared and, histologically, atrophy of the mucosa became evident. In time and without hormone therapy the mucosa appeared greyer, further microscopic signs of atrophy were evident and burning and dryness became an increasingly common complaint. Progressive atrophy of the oral mucosa occurred and in some patients a particularly intense burning sensation was reported. Histologically the oral changes were similar to those affecting the postmenopausal vulvovaginal tract. They stated that histological changes in the oral and vaginal epithelium could be reversed, and symptomatic relief achieved, if there was early treatment by buccal injections of estradiol dipropionate or oral administration of diethylstilbestrol.

In a broadly comparable animal study in oophorectomized monkeys Zisken, Blackberg and Slanetz (1935) described similar reversal of histological changes in the oral mucosa following oestrogen administration. In the following year Zisken (1937) confirmed these oestrogen dependent histological changes in the oral epithelium of women.

To obviate the need for oral mucosal biopsies, Ziskin and Moulton (1948) studied the cytology of the oral mucosa of women during the various phases of the menstrual cycle. Their study concluded that there was a correlation between serum oestrogen levels and the degree of cornification of the oral epithelium. However, independently both Iusem (1950) and Trott (1958) were unable to demonstrate this correlation. Nevertheless Iusem (1950) felt that there was a relationship between serum oestrogen levels and the maturity of cells.

Massler (1951) reported a series of 86 climacteric patients with oral symptoms, of whom approximately one-third responded to vitamin B complex replacement therapy. In the same study, one third of patients responded to oestrogen replacement therapy and the remaining patients were improved by combined vitamin and hormone therapy.

Litwack, Kennedy and Zander (1970) reported that the buccal mucosa of squirrel monkeys was altered in oestrogen deficient animals. Hormonal therapy in surgically oopherectomized animals reversed these changes. These oral changes could be demonstrated histologically at four months post-oopherectomy. In the light of their own findings, the authors commented on the previous reports which suggested histological changes were found to occur slowly in the oral mucosa after the menopause and that unless studies used sensitive techniques to investigate patients in the postmenopausal period then the conclusions drawn might be misleading.

Hertz et al (1971) considered the oral symptoms of the menopause to be largely psychological in origin (see 1.9). They did however find that stimulated parotid salivary flow was significantly lower in postmenopausal women which they conceded could account for some of the symptoms of burning in the oral cavity.

A more direct study investigating hormonal effects on the oral cavity was that of Pisanty, Rafaely and Polishuk (1975). They reported that topical applications of a placebo, or oestrogen and progesterone or oestrogen alone, all resulted in increased salivary secretion, mucosal regeneration and some improvememt in oral symptoms. They concluded therefore that there was no specific effect of female sex hormones alone on the symptom of burning and that repeated massage with any suitable ointment base would be effective.

In an attempt to obtain demographic data on the relationship between a burning sensation of the mouth and the climacteric Basker et al. (1978) studied patients attending a Menopausal Research Clinic. A variety of oral symptoms were recorded in 26% of patients and seventy-three per cent of these patients complained of a burning mouth. The figure was markedly lower than the 80 out of 86 climacteric women reported by Massler (1951). Basker et al (1978) speculated that the difference between these two studies might be explained by differences in cultural background. In addition, if the incidence of burning mouth syndrome (BMS) was as high as Massler (1951) reported then BMS would have become a widely recognised

feature of the menopause which is presently not the case. In the study of Basker et al (1978) 24 patients with oral symptoms were treated with replacement oestrogen and progesterone therapy simulating the natural pre-menopausal ovarian cycle. 9 of the women became symptom free. 8 noted symptomatic improvement and 7 reported no change after 6 months treatment. Cytological investigation of vaginal epithelium in these patients showed a marked increase in cornification. however. they reported a minimal increase in cornification of the palatal mucosa. The authors concluded that oestrogen deficiency was a relatively uncommon cause of burning mouth syndrome and study of the relationship between oral symptoms and hormone therapy needed further development.

In a further attempt to obtain demographic data. Ferguson et al (1981) undertook a postal questionnaire survey of 145 oophorectomized women. Overall 17.9% of such women reported a burning sensation affecting the tongue or lips. but it was not clear whether these patients had BMS. Additional findings were that there was no temporal reduction in the incidence of oral symptoms after oophorectomy and oestrogen therapy did not improve the patients oral symptoms including burning. In contrast systemic symptoms such as flushing showed a linear reduction reaching statistical significance 5 years after surgery. The authors postulated that many women undergoing the climacteric had vasomotor disturbances due to oestrogen deficiency which in turn may lead to neurosis. and an increased prevalence of oral symptoms

such as a burning sensation. However no patients in that study were screened for organic or denture disturbances and therefore such patients cannot be regarded as having symptoms solely attributable to neurosis.

Subsequently, Main and Basker (1983) reported that only 3 of their 37 burning mouth patients had burning associated with the menopause.

1.6 SALIVARY FLOW

Salivary flow rate has been shown to decrease with age (Becks and Wainwright, 1943; Bertram, 1967; Mason and Chisholm, 1975). Kullander and Sonesson (1965) showed that there was a marked decrease in the rate of both resting and stimulated submandibular flow in postmenopausal women, although this was not confirmed by Ericson (1968). However Ericson (1969) did confirm a reduction in stimulated parotid secretion with age, although individuals may have different responses to various salivary stimuli, and therefore results obtained from sialometric methods using different stimuli may not be comparable. Ericson (1971) further demonstrated that maximal secretory rate is directly proportional to the size of the gland and varies greatly between individuals. Heft and Baum (1984) found no significant differences in stimulated or unstimulated parotid flow rate related to age.

Since burning mouth syndrome occurs predominantly in postmenopausal women, reduction in salivary flow has been suggested as a possible cause of the burning (Elfenbaum, 1969). Hertz et al (1971) showed that the

parotid salivary flow rate (both resting and stimulated) was reduced in menopausal women thus giving objective support to subjective feeling of dryness and burning of the mouth in this group. However Glick et al (1976) in a study of resting mixed saliva showed no differences in flow rate between postmenopausal women who had burning tongues and those who did not. These findings were similar to those reported earlier by Karshan et al (1952). Glick et al (1976) did show however that there were differences in composition of the saliva of postmenopausal women with the complaint of a burning tongue and asymptomatic individuals. Protein, potassium and phosphate were significantly higher in women with burning tongues and the authors considered that the phosphate concentration reflected hormonal changes as reported by Ben-Aryeh et al (1976).

Xerostomia, can be caused by many factors, in addition to age, including pathological conditions of the salivary glands and can also be idiopathic. Mason and Chisholm (1975) reviewed the clinical consequences of xerostomia fully and indicated that, irrespective of aetiology, patients could complain (in addition to dryness) of a burning sensation, sore tongue or generalised oral discomfort. Factors thought to contribute to xerostomia include mental stress, anxiety and psychopathological emotional states (Bates and Adams, 1968; Brown, 1970), drug therapy, pernicious anaemia, iron deficiency anaemia, diabetes mellitis, various vitamin and hormonal deficiencies as well as systemic

disorders such as Sjögrens syndrome which may involve the salivary glands.

1.7 CONTACT STOMATITIS

Since burning mouth frequently occurs in relation to events such as the provision of new dentures, patients dentists and physicians often assume that allergy to the acrylic resin of the denture base to be the cause.

It is generally accepted that allergy to acrylic (polymethylmethacrylate) is rare, Devlin and Watts (1984) in a review article cast considerable doubt on acrylic resin or its various constituents as being capable of causing sensitivity reactions. They cite many papers in which acrylic allergy was suspected but considered that often the criteria on which diagnosis was made were ill-defined. Thus frequently the signs of allergy were those of stomatitis, and poorly designed patch testing techniques in which pressure was applied to the skin during testing may have caused false positive results. They concluded that mechanical irritation can often mislead the patient, medical and dental practitioner into an erroneous diagnosis of acrylic allergy. However they believed chemical irritation from high level of residual monomer could under certain circumstances cause a burning sensation (Turrell, 1966b).

High levels of residual acrylic monomer in dentures can remain in self cured acrylic and in heat cured acrylic particularly if incorrectly cured or when a short curing cycle is used (Fisher, 1956; McCabe and Basker, 1976; Austin and Basker, 1980). Unfortunately

full testing for residual monomer requires destruction of the denture base and therefore a cause and effect relationship can be difficult to establish. If a high level of residual monomer is suspected the level can be reduced by enclosing the denture in plaster of paris, boiling for 30 minutes and cooling slowly (Anderson and Storer, 1981). Immersion in water is less effective as residual monomer in heat cured acrylic is very resistant to removal by this method (Austin and Basker, 1980).

Acrylic allergy should only be considered as an aetiological factor in burning mouth syndrome following elimination of all other local and systemic factors, and only then when confirmed by patch testing. In addition cutaneous patch testing in the diagnosis of hypersensitivity reactions confined to the oral mucosa may not be reliable due to immunological differences in the response of skin and of oral mucosa, the effect of the oral flora and the presence of saliva (Greenberg, in Lynch 1984).

Fisher (1975) believed that reactions to chemical contact allergens in the mouth were rare due to rapid dispersal and absorption of the allergens through well-vascularised mucosa, a short period of contact with the oral mucosa and dilution and removal of the potential allergens by saliva.

Ali et al (1986) reported 22 patients who had a complaint of burning sensation related to the denture bearing area of the palate present only when wearing the prosthesis and resolving within minutes or hours of its

removal. These patients did not have BMS since the mucosa had visible changes, namely inflammation, vesiculation and ulceration. Five of these patients were found to be allergic to methyl methacylate monomer and high levels of free residual monomer in their acrylic dentures by skin patch testing.

1.8 DIABETES MELLITUS

Sheppard (1942) reviewed the literature on oral symptoms and diabetes mellitus prior to 1942 and discussed the possible link between the two conditions. He suggested that the relationship was a relic of the preinsulin era and that since nearly all known cases of diabetes mellitus are treated and controlled the mouth and tongue of diabetic patients should differ little from those of non-diabetic individuals.

Sheridan et al (1959a and b) suggested however that specific oral symptoms may serve as sensitive clinical indicators of abnormal glucose tolerance. Occasional oral burning was reported in 9 patients out of 26 with abnormal glucose tolerance curves. Chinn et al (1965) reported an investigation of 45 consecutive dental patients whose oral complaints were burning, dryness or gingival tenderness. Thirty one patients reported burning, 2 dryness and 6 a combination of the two symptoms. Twelve of the 45 patients (27%) had abnormal glucose tolerance tests consistent at that time with the criteria for diabetes mellitus.

Chinn et al (1965) also reviewed the study of Sheridan et al (1959a) which had noted that the incidence

of previously unrecognized diabetic patients depended on the sensitivity of the method used, i.e. urinalysis 0.19 to 1.20 per cent, blood sugar 6%. They suggested that if the oral symptoms under consideration were truly a more sensitive test of diabetes mellitus than the classical systemic symptoms then a more sensitive test than the standard oral glucose tolerance test might be employed as an indicator for patients with oral symptoms who may develop diabetes later in life. They proposed that the cortisone glucose tolerance test might be useful in the detection of prediabetes although accepted that this was questionable. Of the 20 patients analysed in their sample 10 showed no improvement in oral symptoms when glycaemic control was instituted and only 2 had complete remission.

Brody, Prendergast and Silverman (1971) examined 142 patients with a variety of oral complaints. Sixty two of these patients complained of burning and of these 39% had an abnormal glucose tolerance test. These authors reitterated that oral symptoms may be a good indicator of early or undiagnosed diabetes mellitus. They suggested that the poor response of oral symptoms to institution of glycaemic control was due to irreversible basement-membrane changes in blood vessels of the oral cavity which is a change which occurs early in diabetic patients.

In contrast, Goss (1973), citing no evidence, stated that the complaint of sore tongue in patients with poorly controled diabetes resolved with improved glycaemic

control. He also claimed that the complaint of sore tongue in patients with controlled diabetes was harder to deal with.

Other than the suggestions put forward by Brody et al (1971) of the aetiological factors linking the complaint of a burning sensation of the mouth to diabetes mellitus it was unchallenged until Basker et al (1978) put forward three reasons for the association. Firstly, since insulin increases the rates of glycogen, lipid and protein synthesis and maintains a balance between the anabolic and catabolic processes within the oral mucosa, the oral tissues may be less resistant to wear and tear. Secondly, xerostomia was a common symptom of diabetes and thirdly oral candidal infections were relatively common in diabetic patients. In this study 110 patients being treated for overt diabetes mellitus were questioned directly about oral symptoms. A dry mouth was reported by 40% of patients, whereas burning mouth or abnormal tastes were reported by only 10% of patients. They also noted that in several cases burning was associated with a particular diabetic therapy. For example burning disappeared in one patient when an oral preparation was replaced by insulin and for an other when the type of insulin was changed. In 21 patients presenting with burning mouth, diabetes mellitus was an initially unrecognised causative factor in 1 case.

Other authors have reported on the association between diabetes mellitus and burning mouth. Thus Oles (1979) reported a case of prediabetic glossodynia which

responded to dietary control. Zegarilli (1984) in his study of 57 patients with burning mouth found no cases of diabetes mellitus in 39 patients who had non fasting blood glucose determination. Finally Main and Basker (1983) who used urinalysis to estimate glucose status detected one case of undiagnosed diabetes mellitus among their patients complaining of a burning mouth.

1.9 PSYCHOLOGICAL ASPECTS

As with much of the literature on the complaint of burning mouth the literature on psychological aspects is fragmented and it is difficult to draw comparisons between studies. Nevertheless a chronological perspective has been adopted to present the available data. Engman (1920) reported 11 patients. 9 female and 2 male who presented with a burning sensation of the tongue, and all of whom had a marked fear of cancer.

Schroff (1935) listed psychological causes. cancerphobia and mental strain amongst factors which caused burning mouth. Landa (1945) believed that cancerphobia was an effect rather than a cause of patients oral symptoms and that since the menopause was associated with nervous depression. irritability and melancholia this allowed a symptom such as burning mouth to develop at the slightest provocation. Treatment he suggested should include dealing with local factors such as ill fitting dentures and lack of saliva. systemic factors via diet and psychological therapy by sympathetic handling and management of the patient with reassurance being

particularly important.

Ziskin & Moulton (1946) described the typical burning mouth patient as the postmenopausal woman, unduly emotionally disturbed over her oral condition, who complained of nervousness, depression, hopelessness or insomnia or had marked cancerphobia. They described their group of patients for whom local and systemic causes had apparently been excluded. A long initial interview to evaluate the patient's psychiatric status was stated as important and an attempt was made to see whether there was an association between oral symptoms and with emotional factors. Shorter review visits were used to observe change in mental attitude. Reassurance they said was important, and in support of this noted only 1 of their 14 middle aged women required psychotherapy. In addition to the factors already mentioned, they found hypochondriacal tendencies, chronic excessive worry and marked dissatisfaction with life among their patients. Ten of the 14 patients appeared grossly neurotic and 13 of the 14 indicated lack of sexual gratification, due to being widowed, unmarried or having invalid husbands. They concluded that there was a pronounced relationship between alleviation of oral symptoms and improvement in emotional status.

Massler (1951) described climacteric symptoms as often being accompanied by emotional changes, and stated that patients often transferred internal tensions into subjective symptoms. He believed that while many of the patients symptoms had an emotional basis they were based

upon real physical changes and they could be alleviated by proper management of the patient. Thus the findings of Massler (1951) are at odds with those of Ziskin and Moulton (1946) in that he disagreed that all symptoms were purely psychogenic. However he felt that the emotional status contributed to the severity of the symptoms. Massler (1951) concluded that the majority of such patients oral symptoms responded to vitamin B complex and oestrogen therapy (1.5).

The question of reassurance in a clinical setting was also emphasised by Kruger and Reynolds (1965); and Coles (1966) who stressed the importance of taking time over the taking of the history. They suggested that patients obtained much well being from someone just listening in an unhurried clinical setting.

Brody and Nesbitt (1967) reported on their assessment of patients with burning mouth where no identifiable organic factor had been found. Dealing with 6 patients only they concluded that the patients superficially appeared to be kind and sensitive but their psychotherapeutic interviews revealed hostile and angry feelings about an environmental situation which were resistant to hypnotherapy. They concluded that their patients with burning mouth demonstrated suppressed hostility and a marked oral aggresive tendency and overcompensated by suppressing any emotions or actions inconsistent with their self image. This complex psychological profile probably contributes to making treatment difficult.

Schoenberg (1967) studied 25 patients with burning mouths and no apparent organic aetiology and suggested that these patients were depressed as a result of psychological stress. This stress resulted from the real or threatened loss of a loved person, valued object or bodily function and that "symptom formation" served the purpose of suppressing the cause by experiencing a real symptom. In addition he felt that the physical symptom increased interest on the part of family, friends and the dentist which satisfied dependency needs. Suggested treatment should include listening carefully , encouraging verbal expression and offering simple explanations to social difficulties. Antidepressant drugs and psychotherapy were useful adjuncts.

In a later study Schoenberg et al (1971) looked at a further 21 patients, of whom 17 were judged to be clinically depressed, the remaining 4 showing covert signs of depression. Onset of symptoms in 40% of the patients was related to a specific oral procedure. Personality testing was completed for 15 of the patients using the Minesota Multiphasic Personality Inventory (MMPI). Nine patients had high scores on the depression scale and 4 on the hypomania scale. All had a preoccupation with loss and inability to accept or integrate loss with their accompanying feelings. Reassurance was an essential part of therapy, however, actual psychotherapy was difficult.

Hertz et al (1971) believed that subjective xerostomia contributed to the complaint of burning mouth, and that this subjective xerostomia was primarily

psychogenic occurring in menopausal or postmenopausal women who had suffered in the past from oral conflicts. These conflicts they considered were reactivated in the menopause when confronted with problems of adjustment to their altered status.

Zucker (1972) listed anxiety states, hypochondriacal reactions, conversion reactions, tic or masochistic reactions. as differential diagnoses of psychogenic tongue symptoms. Anxiety was suggested as a cause of tongue thrusting against the teeth in the tense individual particularly when the patient was subjected to emotional stress.in some cases this response may become habitual. and he also suggested that bruxism had a similar aetiology (see 1.10). Hypochondriacal reactions were also reported as the likely cause of depression reported by Schoenberg et al (1971). Masochistic reaction resulted in the attraction of the tongue to a dental fault and thus compulsively subjecting the tongue to painful stimuli. Reassurance. a sympathetic approach on the part of the clinician and avoidance of excessive treatment measures were necessary for successful treatment of patients with burning mouth. The additional comment was made that patients could be handled appropriately and adequately by the practitioner if he or she was aware of psychological influences. This role of reassuring the patient and adopting a sympathetic approach is reiterated by a number of authors, (Basker et al 1978; Lowental and Pisanti, 1978; Kaner, 1980; Lowental, 1981; Gobetti, 1981).

Harris (1975) suggested that a crucial test

separating organic symptoms from psychosomatic ones was the relation of symptoms to eating and drinking. Organic pains were made worse by eating and drinking, psychosomatic were relieved by eating, drinking or perhaps gum chewing. The author did not however assess this statement scientifically.

Basker et al (1978) reported that cancerphobia and anxiety were causative factors in 5 of their 21 treated patients.

Beck, Kaul and Weaver (1979) discussed the recognition and management of the depressed dental patient, mentioning the role of depression in burning mouth. They also believed the dentist has a role in counselling patients but advised referral for psychiatric or psychological treatment if there appeared to be an increased potential for suicide.

Ferguson et al (1981) attributed oral complaints including burning related to climacteric symptoms, in the absence of physical disturbances, to oral vasomotor changes which occurred as a result of changes in oestrogen levels, similar changes which presumably intracranially were thought to be responsible for accompanying features such as neurosis.

Grushka (1983) highlighted the problem of whether patients with a chronic pain personality perceive burning or whether the burning itself creates the chronic pain personality in patients with burning mouth.

Subsequently Main and Basker (1983) reported that anxiety with or without cancerphobia was a significant

factor in 20% of their 37 patients with burning mouth.

In the study carried out by Zegarelli (1984) on 57 burning mouth patients, 21 (37%) were diagnosed as having burning of psychogenic origin, 14 were postmenopausal females, 5 associated the onset with extensive dental care and 3 with the loss of a loved one. In addition, a further 6 patients (11%) had multiple co-existent causes which included a psychogenic element.

Grushka, Sessle and Miller (1987) confirmed the findings of Schoenberg et al (1971) regarding depression but also concluded that BMS patients differed from age-sex matched controls in that they were more concerned with bodily function than with being emotionally repressed, angry, distrustful, anxious and socially isolated. They concluded that such patients were therefore typical of chronic pain patients suffering from reactive depression.

Van der Ploeg et al (1987) investigated 154 patients using the Speilberger State-trait Anxiety Inventory (STAI-Form Y) Dutch adaptation to measure state and trait anxiety and the Depression Adjective Check List (DACL) Dutch adaptation to measure state and trait depression, in patients complaining of a burning sensation of the mouth. The DACL is a questionnaire designed to measure bodily and somatic reactions to stress and the authors also used the Dutch personality inventory to measure neurotic lability, neurosomatic lability and introversion - extraversion. It is unclear however whether patients with visible oral changes were also included in this study which therefore may not strictly

have been of BMS patients. Nevertheless the study concluded that state and trait anxiety scores were significantly higher than for healthy persons, mean depression scores indicated serious depression, and there was a higher frequency of somatic reactions to stress and higher scores for neuroticism and neurosomatic lability than in the general population.

In a UK study Browning et al (1987) investigated 25 patients with BMS using the Montgomery Ashberg Depression Scale and the General Health Questionnaire and compared them with a control group of 25 patients with a painful oral condition for which there was an identifiable organic cause. That paper concluded that 44% of BMS patients had an associated psychiatric disorder compared to 16% in the control group.

Emotional stress leading to clenching, grinding and other parafunctional habits causing discomfort under dentures is discussed in section 1.10.

1.10 DENTURES

In complete denture wearers a design fault which increases the level of functional stress or which restricts normal function of the circumoral or lingual musculature may lead to a burning sensation of the oral mucosa (Basker et al 1978). Main and Basker (1983) reported in their study of 37 patients complaining of a burning mouth that in 50% of patients the causative factor in the condition related to errors in denture design. Previous sections in this Chapter have highlighted how

non-denture factors might also give rise to a burning sensation affecting the oral cavity.

Stansberry (1928) considered that when healthy oral tissues began to atrophy the underlying cause might be a nutritional deficit brought about by an impaired blood supply. He hypothesised that the older the patient the less readily their oral tissue can accommodate to deviations from normal and the less quickly it accommodates to the functional load imparted by dentures.

Clearly, the oral mucosa of the denture bearing areas can be subjected to stresses for which it was not intended (Stansberry, 1928; Thomson, 1968a & b; Thomson, 1971). The oral mucosa assumes the role of the periodontal membrane, lost on extraction of the teeth, in transferring the functional forces to the underlying bone (Thomson, 1968a).

The effect of denture wearing on the oral mucosa has been investigated histologically and shown conflicting findings (Ostlund, 1958; Hedegard, 1962; Kapur and Shklar, 1963; Van Scotter and Boucher, 1965; Turck, 1965; Al-Ani, Shklar and Yurkstas, 1966; Carlsson, Thilander and Hedegard, 1967; Markov, 1968; Nedelman, Gamer and Bernick, 1970; McMillan, 1971/72; Van Mews, Pinkse-Veen and James, 1975; Jani and Bhargava, 1976; Watson, 1978; and Watson and MacDonald, 1982). The reasons for the conflicting findings can be attributed to the variation in biopsy site chosen, the type and design of denture provided and the method of mucosal sampling and tissue analysis. In one study Pendleton (1951) found similar

mucosal changes in edentulous areas of the jaws in patients whether or not they wore dentures.

Watson (1978) in a small study of post-mortem subjects showed that the morphology of the oral epithelium was most variable in the region of the alveolar crest. He also showed that the surface contour varied most in the region of the palatal rugae, and therefore chose to analyse the mucosa in the first molar region between the alveolar crest and the midline. This site was used in the investigations of Van Scotter and Boucher (1965); Van Mews et al (1975); Jani and Bhargava (1976); and Watson and MacDonald (1982). Watson (1978) showed that in the non-denture bearing mucosa of healthy males, epithelial thickness increased with age, the ratio of basement membrane length to surface length was not related to age and the thickness of the stratum corneum increased with age. Whereas wearing dentures prevented the age related increase in thickness of the epithelium and of the stratum corneum. there was a reduction in the ratio of basement membrane length to surface length related to length of denture experience and keratinization tended to change from complete orthokeratosis to incomplete orthokeratosis. These changes were evident 1 year after insertion of complete dentures in patients with no previous denture experience.

Additionally Watson and MacDonald (1982) showed in healthy adult males that there was no effect on epithelial thickness, epithelial morphology, stratum corneum thickness, or degree of keratinization by smoking in both

denture wearers and non denture wearers. They suggested that a flatter basement membrane in denture wearers indicated an adaptive change to surface loading, and the denture produced a thinner less highly keratinized stratum corneum. Van Mews et al (1975) investigated both male and female adult subjects and produced similar findings regarding the effect of denture wearing on the epithelium-connective tissue interface. However they showed no differences in thickness of epithelium between denture wearers and non-denture wearers. Jani and Bhargara (1976) using adult male subjects with no systemic conditions likely to affect the oral mucosa studied the effects of well constructed dentures on mucosa not previously subjected to denture wearing. They showed that there was an increase in thickness of epithelium with an increase in the keratin layer in denture wearers. Van Scotter and Boucher (1965) investigated male post-mortem subjects and found no direct correlation between age and thickness of stratum corneum or between the thickness of the keratin layer and length of time the denture was worn.

The crest of the maxillary alveolar ridge was investigated by Kapur and Shklar (1963); Turck (1965) and Nedelman et al (1970). Kapur and Shklar (1963) showed that on examining biopsies from the premolar region before and twelve weeks after immediate denture insertion there was an increase in width of the stratum corneum. Parakeratosis changed to hyperkeratosis and there was a reduction in the connective tissue infiltrate. They concluded that dentures stimulated keratinization. No

indication of the sex of their subjects was given. Turck (1965) studied partially dentate patients over 50 years of age of both sexes with no systemic disease. He examined the mucosa from edentulous ridges of patients who had and who had not worn partial dentures and from tissue surrounding natural teeth. The epithelial thickness from the crest of the ridge did not differ in non-denture or denture wearing patients. There was however variation in keratinization in edentulous non-denture supporting ridges. The basement membrane in the edentulous areas with or without dentures showed marked irregularities even when the epithelium was fully keratinized and the underlying connective tissue was not inflammed. There was normally a mild cellular infiltrate in both non-denture and denture bearing edentulous areas. Ageing and wearing well fitting dentures did not seem to influence the gross histologic picture. Ill-fitting dentures however caused lack of keratinization an irregular shape to the basement membrane and thickening of the epithelium. The connective tissue also showed all the characteristic signs of chronic inflammation.

Nedelman et al (1970) studied male and female patients aged between 31 and 80 years. They found chronic infiltration of lymphocytes and plasma cells in the lamina propria of edentulous non-denture bearing specimens regardless of age sex or duration of the edentulous condition. Insertion of dentures resulted in a decrease of the cellular infiltrate. In non-denture bearing mucosa, the stratum corneum became thicker, but the

thickness was dependent upon the length of time the mucosa was edentulous. They also showed that the insertion and wearing of a denture reduced the thickness of the stratum corneum.

Hedegard (1962) and Carlsson et al (1967) examined labial mucosa in the upper central incisor region. Hedegard (1962) studied the mucosa of healthy patients who had not previously worn a partial denture, and compared this with biopsy specimens of patients who either had an immediate denture inserted or patients with no denture provided after the extraction of the central incisors. He reported that the patients without dentures showed normal keratinization of the mucosa. In the group with immediate dentures the stratum corneum had a parakeratotic appearance and the lamina propria had a high level of cellular infiltration compared with the non-denture group who showed little subepithelial inflammation three months after tooth extraction. Carlsson et al (1967) studied the oral mucosa of healthy men and women aged between 20 and 62 years. They demonstrated a reduction in keratinization of the oral epithelium in some patients and increased inflammatory reaction in the subepithelial connective tissue in some of their immediate denture group while histological changes in the mucosa of the non-denture group were negligible.

In a further histological study of a different site of the oral mucosa Ostlund (1958) investigated biopsy specimens from the hard palate in the region of the vibrating line in male and female subjects. In males

the palatal mucosa was qualitively slightly thicker than females. Wearing dentures caused thinning of the strateum corneum and parakeratosis developed.

Exfoliate cytology was used by Al-Ani et al (1966); Markov (1968); and McMillan (1971/1972) to investigate mucosal changes. Al-Ani et al (1966) examined mucosal biopsies from men and women aged between 11 and 85 years and compared denture wearers with non-denture wearers. Biopsy sites investigated were the hard palate and left and right buccal mucosa and they found no significant differences in the oral mucosa between smokers and non smokers. The palatal but not the buccal mucosa was affected by denture wearing, there being a decrease in keratinization in the palatal tissues. Markov (1968) investigated male and female patients, and concluded that the degree of keratinization of oral mucosa under dentures is inversely proportional to the length of time dentures were worn, there being a greater degree of keratinization in men than women. Keratinization was not correlated with age, but the degree of keratinization was greater in smokers than non-smokers. In addition keratinization was noted to be increased if dentures were removed at night.

Differing histological changes were found in the studies of McMillan (1971/1972) who sampled palatal tissue in males and females aged betweeen 3 and 77 years. He concluded that the tissue response to denture wearing was variable between individuals and not related to the clinical appearance of the mucosa. In addition, under dentures the mucosal surface may be as well keratinized as

when no dentures were worn. The reduction in palatal keratinization under dentures was considered to be related neither to the age of the subject nor to the length of time for which dentures were worn.

It would appear therefore that the effects of denture wearing per se on the histology of underlying mucosa are still somewhat equivocal. Nevertheless histology appearance may have little relationship to clinical symptoms. However there is much evidence to support the comments of Basker et al (1978) that in complete denture wearers a design fault which increases the level of functional stress or which restricts normal function of the circumoral or lingual musculature may well promote a burning sensation.

Wright (1929) suggested that the cause of a burning sensation under a denture can be related to an unbalanced occlusion disturbing the soft tissues and correction of the fault can relieve the patient's discomfort. Wright (1933) further commented that any denture must be correctly made to maintain the health of the underlying mucous membrane.

Pendleton and Glupker (1935) and Pendleton (1937) concluded that the tissues of the mouth reacted favourably to artificial dentures in those regions receiving a well distributed load from the functional stresses and unfavourably in those areas where the load was less well distributed.

Thomson (1968b) described intolerance to dentures in the absence of obvious clinical signs. He described

the discomfort as varying from a continuous irritation of the soft tissues to a pain of a throbbing or burning nature. The discomfort could be localised, variable in location or diffuse within the denture-bearing areas of the mouth the most common sites being the crests of the alveolar ridges and the anterior part of the palate. Irritation of the tongue and lining mucosa of the lips was sometimes an accompanying feature. The symptoms appeared within periods ranging from a few minutes to several hours after the dentures were inserted and were relieved by removing the dentures. Patients tended to remove their dentures when the discomfort became intolerable and when social circumstances allowed. Thomson (1968b) also described excessive loading of the denture-supporting tissues as the basic cause of these symptoms although systemic factors may contribute to lowered resistance to irritating processes in the supporting oral tissues.

Excessive loading of the denture-bearing tissues could be brought about either by denture design faults or as a result of excessive non-functional loading of the dentures or by a combination of both these factors (Thomson, 1971). In consequence Thomson (1968a) suggested that treatment should be aimed at dealing with the excessive load or improving the supporting tissues.

Factors other than denture design may also contribute to overloading of the oral mucosa for example excessive non-functional loading can occur as a result of clenching or grinding of the teeth sometimes referred to as bruxism. Tishler (1928) coined the term "occlusal

habit neurosis" in describing grinding or pressing of the teeth together with great force practiced during sleep, periods of worry, excitement and unusual stress. Although not the only cause, the condition could possibly be due to a traumatic occlusion adjustment of which often eliminated the habit.

Boyens (1940) suggested that patients were often not aware of bruxism or clenching and only realized it when they had been informed of it by the dentist or by their associates. Autosuggestion may be helpful in eliminating nocturnal habits.

Leof (1944) believed that normal tooth contact occured during mastication and swallowing only. He agreed with the suggestion that the patient was usually unaware of a clenching habit but that it occurred when the patient was tired or angry, emotionally disturbed or concentrating on a work problem. The problem was further compounded by habits such as lip biting, cheek biting, sucking on the teeth, sucking of the lips, cheeks or fingers, finger nail biting and pressure with the tongue on the cheeks. Treatment involved making the patient aware of their habit including informing the family in order to point out the habit and thus make use of conscious control to reduce clenching. Relaxation before sleeping was useful in reducing nocturnal grinding. Kimball (1954) referred to clenching in complete denture wearers and suggested occlusal trauma, including an excessive vertical component, was an important contributing factor. It was proposed that symptoms could

be reduced by using fully extended bases to spread the load over as wide an area as possible.

Nadler (1957), Perry et al (1960) and Thomson (1971) highlighted the importance of stress, concentration, studying and watching television as occasions when clenching and grinding may occur. The additional involvement of habits such as pipe and pencil chewing were also discussed.

Stress leading to increased muscular activity (Yemm, 1971) and hence increased clenching have been described by Perry et al (1960) and Yemm (1972).

Whether the occlusal surfaces of teeth contact during normal function has been the subject of some dispute, Gottlieb (1947) found that natural teeth functioned for less than 1 hour/day. Jankelson (1953) suggested that teeth do not contact during mastication only on deglutition.

Yurkstas and Emerson (1954), Anderson and Picton (1957), Sheppard and Markus (1962) and Brewer (1963) showed that complete dentures did contact on chewing. Brewer (1963) further showed that the type of contact varied with the jaw relationship, cusp form, occlusion and type of food eaten. Non-masticating waking contacts varied from 150 per hour in some patients to 1,500 per hour in others. Sleeping contacts totalled 3 to 15 minutes per night for some patients to as much as 2 hours 30 minutes in others. Sheppard and Markus (1962) found that actual tooth contact did not exceed 11.7 minutes per day.

Thomson (1965) described attrition of acrylic teeth on complete dentures of patients who habitually grind or clench their teeth as wear characterized by sharp angles and flat polished facets on the occlusal surfaces which was very different from the wear caused by function during mastication. This clenching wear pattern was often associated with loads in excess of the tissue tolerance resulting in soreness of the supporting tissues. Thomson (1968a) described these polished facets as an indication of a tooth-grinding habit.

Generalized discomfort of the denture-bearing tissues can be reduced by paying attention to certain design features of complete dentures. The load transmitted to the tissues during mastication can be decreased by reducing the area of the occlusal table so that smaller amounts of food are contacted and less masticatory work is required (Rapp, 1954; Frechette, 1955; Kydd, 1956; Mumford and Storer, 1962; Wehner, Hickey and Boucher, 1967; Ortman, 1971; Bearn, 1973; Roedema, 1976; Roedema, 1979). In this context, narrow posterior teeth penetrate food more easily (Thomson, 1968a) and thus may help to reduce transmitted masticatory loads.

Another factor which can lead to overloading of oral denture bearing tissues is lack of freeway space due to excessive occlusal face height. (Kimball, 1954; Lawson, 1959; Mack, 1964; Thomson, 1968 (a); Thomson, 1968 (b); Hickey et al, 1985).

Excessive loading of the denture bearing tissues can be minimised by redistributing the load by utiliziing

the maximum mucosal supporting area that the anatomy of the mouth will allow, thus reducing masticatory load per unit area. (Kimball, 1954; Lawson, 1959; Mumford and Storer, 1962; Thomson, 1968a; Thomson, 1971; British Society for the Study of Prosthetic Dentistry, 1981). Lawson (1959) reviewing design errors in cases of complete denture failure showed that under-extension of the bases was the commonest error and commented that very few dentures utilized more than a small fraction of the supporting area available. In many cases the areas could have been doubled. Thomson (1967) reported that an increase in support area of 50% could be achieved in complete dentures with underextended bases.

Thomson (1971) highlighted the anatomical unsuitability of the oral tissues used to support complete dentures. In normal function, no heavy load is placed upon the oral epithelium. With complete dentures the oral epithelium is made to serve the same purpose as the periodontal membrane which was adapted to provide support for natural teeth. Thomson (1967) measured the denture support area available in 20 edentulous patients selected at random. The mean denture-bearing area in the edentulous maxilla was 22.96cm^2 and 12.25cm^2 in the mandible. Watt et al (1958) measured the amount of periodontal membrane available for support in each jaw. Their sample showed that in both the maxilla and mandible 45cm^2 was available. This highlights the support deficit for complete dentures when these figures are compared to those of Thomson (1967), particularly when the mandible is

considered.

Better load distribution might be achieved by relief of certain areas (Kimball, 1954; Thomson, 1968(a); Thomson, 1971), the use of resilient liners (Kimball, 1954; Storer, 1962; Bates and Smith, 1965; Thomson, 1971; Makila, 1976; Schmidt and Smith, 1983), or selective impression techniques (Thomson, 1971; Watt and MacGregor, 1983).

1.11 CONCLUSIONS

Much of the evidence presented in this Chapter for the role of aetiological factors in the complaint of a burning sensation of the mouth is conflicting. It is likely that this is due in part to burning mouth symptoms in an individual patient being multifactorial. Also, some authors rather than investigating the full range of previously known aetiological factors have focused their attention on a narrower field usually of their own specialist interest.

Silverman (1975) summarised his interpretation of the literature. He believed that there was an impairment in the cellular integrity of the tissues in their response to microtrauma from either the external environment, such as from a denture, or from a disturbance of the internal environment, such as disease of the hormone system or the circulation. These factors in turn were thought to affect the steady-state metabolism for cellular repair and homeostasis causing oral discomfort such as a burning sensation.

The management of patients with burning mouth

syndrome is further complicated by the inter-relationship between many of the aetiological factors. Reduced salivary function, iron deficiency and the presence of dentures all increase the chances of candidal species colonization. Stress, diabetes mellitus, and the menopause can all reduce salivary function. Reduced salivary function increases the potential for dentures to induce trauma and also reduces their retention. Denture difficulties and oral discomfort may lead to dietary insufficiency or psychological problems resulting in further oral discomfort.

These are some examples of the effect a number of factors might have. If taken individually they may have little significance but in combination might be contributory factors to a burning sensation. Even if physical causes are treated, psychological factors might cause the condition to persist.

This review highlights that it is important when dealing with patients with symptoms of a burning mouth to adopt an ordered comprehensive approach to management by considering a number of aetiological factors simultaneously in a sympathetic and reassuring manner.

CHAPTER 2

DESIGN OF THE STUDY / MATERIALS AND METHODS

2.I. PATIENT SELECTION

For the purpose of this prospective study a "Burning Mouth Syndrome" clinic was established and held on one session per week in the Oral Medicine Unit. All patients were seen jointly by the author and the same member of staff of the Oral Medicine Unit.

One hundred and fifty patients who were referred to the Department of Oral Medicine and Pathology and the Department of Prosthodontics, Glasgow Dental Hospital and School between 1st October 1984 and 31 January 1986 with a complaint of burning in the mouth and a normal appearance to the oral mucosa were included in the study.

2.2 HISTORY AND EXAMINATION

The patient's history was often complex and in order to maintain consistency of recording clinical and laboratory data a standard pro-forma was used (appendix 2). It was felt important that two persons should be present, one to question the patient and record the information and one who could act as an observer to note the patient's general demeanour and detect any abnormal oral habits such as clenching or tongue thrusting.

The history taking and examination was carried out in a conventional dental surgery where privacy could be

maintained. It became apparent during the study that full details were often obtained only after a number of visits when the patient's confidence had been gained. This reinforced and justified the need for the same clinicians to be present.

At the first visit an overall view of the patient's complaint together with personal data was recorded. In addition, haematological, biochemical and microbiological investigations were performed. Stimulated parotid salivary flow rates were recorded and denture design assessed. At subsequent visits in the light of the findings further questioning took place and appropriate therapy or further investigations were instituted as necessary.

2.3 PATIENT DATA

Patient's name, hospital number, sex, date of birth, date of first appointment and source of referral were recorded.

The site of burning, duration of symptoms, and pattern of burning were recorded as it became clear early in the study that burning mouth patients did not in terms of their history, constitute a homogenous group. A classification system was introduced such that patients who said their burning was absent on waking developed as the day went on and was maximal in the evening were termed Type 1 Burning Mouth Syndrome. In contrast patients who reported burning which was present on waking and constant throughout the day were termed Type 2 BMS.

In both Type 1 and Type 2 the burning was unremitting being present every day. Finally and less commonly patients reported variable burning which need not be present every day and these patients were classified as Type 3 BMS. The relationship of symptoms to eating and drinking, the ingestion of irritant foods or cooling drinks was recorded.

In order to quantify the severity of symptoms patients were asked to choose a value on a linear analogue scale which represented their degree of discomfort (Bond, 1984). They were told that a figure of zero represented no discomfort, and a figure of 10 represented symptoms that were intolerable and could not be worse. This allowed an assessment of the severity of patients' initial symptoms and the progress of treatment to be made as well as allowing quantification of inter-patient comparisons.

Climacteric symptoms, drug history, sleep pattern, smoking, history of diabetes mellitus or of depressive illness were recorded. Home and social circumstances were questioned and the patients were asked to score these on a 0 to 10 analogue scale, zero being "things could not be worse" ten representing "things could not be better".

2.4 HAEMATOLOGICAL INVESTIGATIONS

A 35-40 ml sample of venous blood was withdrawn from the ante cubital fossa using a 50 ml syringe. This sample was then immediately divided as follows: 10 millilitres was delivered to each of two plain glass containers for the estimation of serum vitamin B12 and

serum ferritin. Four mililitres was delivered to each of two plastic vials containing potassium EDTA, which prevents clotting, one for the corrected whole blood folate estimation and the other for full blood count and film . In addition 2.5 millilitres was placed in a container with fluoride oxalate for a plasma glucose estimation (section 2.5). The assays of serum ferritin, corrected whole blood folate and serum vitamin B12 were undertaken for the identification of specific deficiencies and glucose estimations were taken to detect undiagnosed diabetes mellitus.

The haematological investigations were completed by the Department of Haematology, Gartnavel Hospital, Glasgow, Scotland. An automated Coultercounter model S was used to determine full blood counts and differential blood films.

The haemoglobin level (Normal adult range : male 13-18 g/dl, female 11.5-16.5g/dl) and the mean cell volume (MCV) (Normal range 78-99 fl) give an indication of (i) the presence of anaemia or polycythaemia, (ii) macrocytosis suggesting pernicious anaemia or folate deficiency, and (iii) microcytosis suggesting iron deficiency.

It has been reported that iron deficiency can be assessed most accurately by estimation of the serum ferritin level; this is reduced if iron stores are low (Jacobs, et al , 1972; Walters, Miller and Worwood, 1973; Lipschitz, Cook and Finch, 1974; Mazza, et al 1978; Worwood, 1980; Allan in Macleod, Edwards and Bouchier,

1987). For this reason serum ferritin was chosen in preference to estimations of serum iron and total iron binding capacity (Young and Hicks, 1965). Serum ferritin was estimated using a radioimmunoassay (Corning method), with a normal range of 12-300 ng/ml, although in the presence of inflammatory disease ferritin levels up to 25 ng/ml would suggest iron deficiency.

Folic acid deficiency is most accurately determined by estimating the corrected whole blood folate which acts as an indicator of total stores. Corrected whole blood folate was estimated by radioimmunoassays (Normal range 75-400 mg/ml). Low serum vitamin B12 suggests the presence of pernicious anaemia particularly in conjunction with a raised MCV signifying Macrocytosis. Serum Vitamin B12 was also determined by radioimmunoassay techniques. (Normal range 175-800 pg/ml). (Allan in Macleod, Edwards and Bouchier, 1987).

2.5 BIOCHEMICAL INVESTIGATIONS

Five millilitres of venous blood was withdrawn and placed in a container with fluoride oxalate and plasma glucose estimated by a specific enzymatic assay. (Normal range 2.80 - 6.0 mmol/L).

For patients with an elevated random blood glucose estimation a standard oral glucose tolerance test was performed. This involved asking the patient to attend when he/she had fasted overnight. A 2.5 ml sample of venous blood was withdrawn as described above followed by the oral administration of 75 grams of glucose dissolved in water. Further samples of venous blood were withdrawn at

30 minute intervals over a 2 hour period. and the plasma glucose as these times measured.

The aim of the test is to investigate the patient's ability to respond to loading with glucose. In a normal patient the initial plasma glucose level will be within the normal range. Following administration of glucose orally there will be a rise to a maximum of 9.5 mmol/L at about 1 hour. By 2 hours the plasma glucose level will have returned to normal in response to insulin produced by the pancreas as a result of the increased plasma glucose level.

In the diabetic the initial plasma glucose level is often higher than normal and after glucose loading blood levels greatly exceed the normal response. and may exceed the renal threshold provoking glucosuria. In patients with diabetes mellitus. the 2 hour plasma glucose level remains elevated at greater than 11.5 mmol/L (Baird in Macleod. Edwards and Bouchier. 1987).

A proportion of the patients within the study group had assays for the following Vitamins : Vitamin A. B1. B2. B6. C. D and E. It was not possible to assess all patients as the facility for measurement was subsequently withdrawn. A 10 ml venous blood sample was collected using a lithium-heparin container. The sample was centrifuged and plasma and red cells separated. Plasma retinol (Vitamin A) and alpha-tocopherol (Vitamin E) were assayed using high performance liquid chromatography (Catignani and Bieri, 1983), ascorbate (Vitamin C) by spectrophotometry (Sauberlich, et al 1976), and 25-OH D3

(Vitamin D) by competitive protein binding (Belsey, De Luca and Potts, 1974) after preparative chromatography (Boyle et al, 1977). Washed and lysed red cells were used to indirectly assay thiamine (Vitamin B1), riboflavine (Vitamin B2) and pyridoxine (Vitamin B6) by the increase in activation effect of added vitamins on the red cell thiamine-dependant transketolase (E.C. 2.2.1.1.), riboflavine-dependent glutathione reductase (E.C. 1.6.4.2.) and pyridoxine-dependent aspartate amino transferase (E.C. 2.6.1.1.) respectively (Williams, 1976; Bayoumi and Rosalki, 1976).

These techniques are in routine use and standard age-related reference values have been achieved by the Scottish Vitamin Referrence Laboratory, who are particularly concerned with the nutritional status of the elderly, for a West of Scotland population. (Normal values plasma vitamin A 1.0-2.8 µmols/L, plasma vitamin C >12 µmols/L, plasma vitamin D 10-50 ng/ml and plasma vitamin E 14-39 µmols/L. Red cell activation effect normal values vitamin B1 <25%, vitamin B2 <23% and vitamin B6 <20%). These Vitamin assays were performed at the Biochemistry Department, Stobhill Hospital, Glasgow, Scotland.

2.6 MICROBIOLOGICAL INVESTIGATIONS

Since the oral tissues in burning mouth syndrome appear essentially normal the concentrated rinse culture method was chosen as a sensitive method of sampling the oral flora. Samaranayake et al (1986) have shown that this method was simple to perform, was equally sensitive to the

imprint culture technique and was superior in quantifying yeast, coliform and staphylococcus counts. It also has the advantage of being simpler to sample the oral flora in patients with reduced salivary function.

The oral rinse was performed by supplying the patient with 10 ml of sterile phosphate buffered saline (PBS; 0.1M, pH7.2) in a universal container and asking them to rinse the mouth thoroughly for 60 seconds. The mouth rinse was then returned to the universal container and sent to the laboratory for microbiological analysis.

The oral rinse was then concentrated by centrifugation at 1700 g for 10 min., and the deposit was suspended in 1 ml of sterile PBS to obtain the concentrated mouth rinse. A spiral plater (Spiral Systems Marketing Limited, Maryland, USA) was used to deliver twenty five microlitres of each rinse sample onto the following media : Sabouraud's Dextrose agar (candidal count), Mannitol Salt Agar (Staphylococcus aureus count) and MacConkey's agar (coliform count). The Sabouraud's plates were incubated for 48 hours while the other plates were incubated for 24 hours at 37° C. The number of colony forming units (cfu) of yeasts or bacteria in each plate was enumerated using a Gallenkamp Colony Counter (Gallenkamp, Leicestershire, England) after which the number of cfu per ml of the sample was quantified.

For the purposes of standardisation with a reference population the patients in the study were compared with a control group of age and sex matched individuals. The control individuals in addition were also

matched for denture status and denture wearing pattern.

2.7 STIMULATED PAROTID SALIVARY FLOW RATES

Stimulated parotid salivary flow rates were employed as a measure of salivary function, using the method described by Mason and Chisholm, (1975). The patient was seated comfortably in the dental chair and the procedure explained. A suction cup device was applied over the duct openings of the right and left parotid glands. The suction cup device was first described by Carlson and Crittenden in 1910 and has been modified many times (Mason and Chisholm, 1975). It is often used in the routine collection of parotid saliva and its methods of use has been well documented (Diamant et al 1957; Shannon et al, 1962; and Mason and Chisholm, 1975.) Suction was provided by means of a dental aspirator unit.

One millilitre of 10% citric acid was applied to the dorsum of the tongue from a 2 ml syringe, and the patient asked to rinse the solution around the mouth before swallowing. Saliva was collected over a 1 minute period in graduated collecting tubes.

The volume collected was compared to the normal ranges for age and sex published by Mason and Chisholm (1975).

2.8 ASSESSMENT OF DENTURE DESIGN

Since assessment of the design of dentures is in some aspects subjective every attempt was made to record only faults that were clinically relevant to any particular patient's complaint. In addition to correction of the most relevant faults any replacement denture was

constructed in full accordance with standard principles of prosthodontics (Working party report of the British Society for the Study of Prosthetic Dentistry, 1981). For patients with complete dentures, particular attention was paid to design features that might contribute to (a) overloading of the denture bearing tissues and (b) tongue restriction. These features are for (a) lack of base extension and excessive occlusal face height and for (b) buccolingual tooth position, level of occlusal plane and lack of freeway space. In addition, features which might lead to movement of the bases and hence trauma to the underlying tissues were noted. These were lack of fit of the bases, lack of retention, lack of muscle balance, faults in the recording of the horizontal component of the jaw relationship and occlusal imbalance.

For patients with partial dentures particular attention was paid to the adequacy of the support of the dentures particularly if the denture was mucosa-borne. Retention was assessed and any occlusal errors were noted, particularly errors which created an excessive occlusal face height and prevented natural posterior tooth contact.

Denture cleansing habits were questioned and the cleanliness or otherwise of dentures was assessed subjectively. Finally the occlusal surfaces of the dentures were examined for attrition facets which might indicate the presence of a parafunctional habit. During interviews the patients were observed, note being made of any habits such as clenching, grinding or tongue thrusting in addition to directly asking patients about the

possibility of such habits.

The relationship to provision or modification of dentures to the onset of symptoms was also recorded.

2.9 ASSESSMENT OF PSYCHOLOGICAL FACTORS

During history taking, a subjective assessment of depression and cancerphobia was made, specifically attention was paid to problems within the patient's domestic and social circumstances, particularly the presence of invalid spouses or children who were being cared for by the patient and the degree of satisfaction the patients had with their domestic surroundings. Another factor that emerged as possibly being relevant was knowledge of a friend or relative who had had oral carcinoma. and patients were asked specifically if they were concerned that the burning sensation represented cancer.

In order to have an objective assessment of psychological status every third patient was asked to complete the Cattell's 16PF Form C questionnaire (Cattell, Eber, and Tatsuoka, 1970), prior to initiating treatment. Every third patient was chosen because such a psychological assessment was time consuming, this time had to be found seperately. Nevertheless, this objective assessment totalled 47 patients which is likely to be representative of the whole group. The advantage of this particular questionnaire was that it had already been employed to investigate patients in the dental environment (Reeve et al, 1982; Reeve, Watson and Stafford, 1984;

Watson and Reeve. 1985; Watson et al. 1986). Responses to the questionnaire items generate raw scores on 16 bi-polar scales (Table 2:1) which provide a comprehensive psychological profile of the individual. These scores can be converted to standard scores (stens) which are plotted on a profile sheet. An individual's profile can be compared to those of relevant populations. Equally a mean profile for a particular sample can be calculated and compared with that for the general population.

The completed Cattell's 16PF Form C questionnaires were analysed by Mr Peter E Reeve (Department of Applied Psychology, University of Wales Institute of Science and Technology, Cardiff, Wales).

2.10 INVESTIGATIONS OF ALLERGY TO FOODSTUFFS AND DENTAL MATERIALS

Patients for whom an allergy to foodstuffs or dental materials was suspected, were referred for appropriate patch testing to a consultant dermatologist, Dr Angela Forsyth (Contact Dermatitis Investigation Unit, Belvidere Hospital, Glasgow, Scotland). Patch testing was reserved for patients in whom the burning sensation was related to the denture bearing mucosa and for whom the results of all other investigations were negative, and to Type 3 BMS patients in whom all other investigations were negative.

The testing consisted of applying allergens recommended by the International Contact Dermatitis Group to the patients' back and removal after 48 hours. Examinations for reactions were made at 48 and 96 hours.

Allergens selected depended on the history given by the patient but were made up from the Chemtec dental battery, the standard European series of allergens (Fregert, 1981) and a food additives battery including flavouring agents and essential oils (Lewis et al, 1989). A positive reaction (Type IV delayed hypersensitivity) was indicated by erythema underneath the test patch at 48 or 96 hours. Urticaria after 1 hour indicated a mildly positive reaction of doubtful significance to the patient's symptoms.

2.11 SAMPLING DIFFICULTIES

As with any clinical study it is very difficult to obtain every desired investigation on every patient. There are a variety of reasons for this including difficult venepuncture, sample damage or loss during transit to the laboratory, problems with processing and patients being unwilling to submit to repeat tests. Despite every effort being made in this study to obtain complete data for all patients, this was not possible, for example glucose estimations were only available for 128 patients and oral rinse for 128 patients, although it is important to emphasise that these represented different individuals, and stimulated parotid flow rates could be estimated for only 114.

Factor	A person with a low score is described as:	A person with a high score is described as:
A	Reserved, detached, critical, cool	Outgoing, warmhearted, easy-going, participating
B	Less intelligent, concrete-thinking	More intelligent, abstract-thinking, bright
C	Affected by feelings, emotionally less stable, easily upset	Emotionally stable, faces reality, calm
E	Humble, mild, obedient, conforming	Assertive, independent, aggresive, stubborn
F	Sober, prudent, serious taciturn	Happy-go-lucky, heedless, gay, enthusiastic
G	Expedient, a law to himself, bypasses obligations	Conscientious, persevering, staid, rule-bound
H	Shy, restrained, diffident, timid	Venturesome, socially bold, uninhibited, spontaneous
I	Tough-minded, self-reliant, realistic, no-nonsense	Tender-minded, dependent, over-protected, sensitive
L	Trusting, adaptable, free of jealousy, easy to get on with	Suspicious, self-opinionated, hard to fool
M	Practical, careful, conventional, regulated by external realities, proper	Imaginative, wrapped up in inner urgencies careless of practical matters, bohemian
N	Forthright, natural, artless, sentimental	Shrewd, calculating, worldly, penetrating
O	Placid, self-assured, confident, serene	Apprehensive, worrying, depressive, troubled
O1	Conservative, respecting established ideas, tolerant of traditional difficulties	Experimenting, critical, liberal, analytical, free-thinking
O2	Group-dependent, a 'joiner' and good follower	Self-sufficient, prefers own decisions, resourceful
O3	Casual, careless of protocol, untidy, follows own urges	Controlled, socially precise, self-disciplined, compulsive
O4	Relaxed, tranquil, torpid, unfrustrated	Tense, driven, overwrought, fretful

Table 2:1 The meaning of the 16PF questionnaire (form C)

CHAPTER 3

PATIENT DATA - RESULTS

3.1 SEX

In total the study group comprised 150 patients of whom 131 (87.33%) were female and 19 (12.67%) male, 6.89:1 (See Figure 3:1).

3.2 AGE

The age of patients at clinical presentation is shown in Table 3:1 and illustrated in Figure 3:2 where they are grouped into five year age bands. The age range of patients varied from 31 years to 85 years with a mean of 59 years.

Of the 131 female patients 104 (79%) were postmenopausal by enquiry, 19 (14%) had symptoms of the climacteric and 8 (6%) had normal menstrual cycles (Figure 3:3).

3.3 SITE OF BURNING

The number of patients affected by burning at particular sites is shown in Table 3:2 and Figure 3:4. The tongue was the most commonly reported site, followed by the palate and upper alveolus or upper denture bearing tissues, the lips, and lower denture bearing tissues or lower alveolus. Less commonly, the buccal mucosa, throat and floor of mouth were affected. In total 60% of patients complained of burning at more than one of these sites. Generalised oral burning was present in 7.33% of the sample.

3.4 SOURCE OF REFERRAL

Patients with burning mouths were referred to Glasgow Dental Hospital and School from four sources. General dental practitioners referred 76 patients (50.67%). 33 patients (22%) attended without referral (self-referral) the majority of these having attended the Dental Hospital previously, 32 patients (21.33%) were referred by general medical practitioners and 9 patients (6%) were referred by hospital consultants outwith the dental hospital (Figure 3:5).

3.5 DURATION OF SYMPTOMS

The duration of symptoms at presentation are shown in Figure 3:6.

3.6 SYMPTOM PATTERN

Table 3:3 shows the pattern of burning reported by patients. Where known the symptoms were present in the following percentages: burning present on wakening 43.53%, symptoms becoming worse as the day progressed 70.74%, maximum discomfort in the evening 56.55%, burning that caused wakening 23.30% and burning that had changed the patients' sleep pattern 23.44%.

By grouping various symptoms together it was possible to classify 145 patients according to Type 1, 2 or 3 BMS.

Type 1. Those patients who presented with the "classical symptoms", i.e. no burning on wakening, burning then developing and becoming more intense as the day progressed and being most severe in the evening. This

pattern of burning wakened patients from sleep in a few cases.

Type 2. Patients whose symptoms did not fit the pattern of Type 1 but had burning present each day, often on wakening and persistent throughout the day. Again this pattern of burning wakened a few patients from sleep.

Type 3. Those patients whose symptoms followed no pattern and were intermittent. not being present each day.

Fifty one patients (35%) were classified Type I, 78 (54%) Type 2 and 16 (11%) Type 3).

Table 3:4 and Figure 3:7 shows the relationship between burning symptoms and eating and drinking. This relationship was known for 147 patients. Burning was associated with eating and drinking in 51.70% of these patients. In these patients burning was frequently made worse by the ingestion of hot. spicy foods or hot drinks such as coffee and tea. In addition some patients avoided vinegar and citrus fruits and drinks.

Where symptoms were relieved by drinking or eating it was frequently cold drinks that were responsible. Some patients who wore dentures obtained relief by running them under a cold tap before replacement in the mouth.

3.7 SEVERITY OF SYMPTOMS AT PRESENTATION

One hundred and forty-two patients were able to indicate the severity of the burning sensation using the linear analogue scale (Table 3:5 and Figure 3:8). Forty five patients (31.69%) described the symptoms as intolerable (score of 10) and only 7 (3.52%) scored their symptoms less than 5. Ninety patients (63.38%) indicated a

severity score of 8 or more.

3.8 MEDICAL HISTORY

3.8.1 Drug History

One hundred and two patients (68%) were receiving some form of regular medication prior to presentation. 26 patients (17.33%) were taking tranquillisers, 48 (32%) sleeping tablets, and 9(6%) antidepressants. Three patients (2%) were on medication for diabetes mellitus and 65 (43.33%) were receiving other forms of regular medication (Figure 3:9).

3.8.2 Smoking

One hundred and forty-six patients were questioned about their smoking habits. Forty patients (27.39%) admitted to smoking. The number of cigarettes/day or the equivalent for pipe smokers is shown in Figure 3:10. The ratio of females to males in this group of smokers was 5.66:1 compared to 6.89:1 in the study group as a whole.

3.8.3 History of Diabetes Mellitus

Four patients (2.66%) were known to have diabetes mellitus on presentation 3 were being treated with medication (see 3.8.1) and 1 by dietary control.

3.8.4 History of Depressive Illness

One hundred and forty-six patients were questioned about a history of depressive illness; 43 patients (29.45%) indicated that they had been or were being treated for depression. Five (11.62%) of these patients were taking antidepressant drugs on presentation, representing 55.5% of patients who were receiving this

type of medication.

3.8.5 Sleep pattern

One hundred and thirty four patients were questioned about their pattern of sleep. Incomplete data was recorded for 8 of these patients. Sixty seven patients described sleeping normally. Of these patients 44 had an entirely normal sleep pattern. The remaining patients either had difficulty getting to sleep, early morning wakening, burning that caused wakening or burning that had changed their sleep pattern. (Table 3:6).

The sleep patterns of the 59 patients who described sleeping abnormally are shown in Table 3:7.

Twenty seven patients (20.14%) of the 134 questioned indicated that burning caused wakening from sleep. Six (8.95%) of those described their sleep as otherwise normal and 21 (31.34%) of those described their sleep as abnormal.

3.9 DOMESTIC AND SOCIAL CIRCUMSTANCES

The linear analogue score indicated by 143 patients is shown in Figure 3:11.

Features elicited from patients which affected their satisfaction with their domestic and social circumstances included living with an incapacitated spouse or handicapped child (a surprisingly common finding - 20%), dissatisfaction with their home or its locality and fear of vandalism.

Although there is presently no comparable clinical data the clinical impression gained at interview was that a score of less than eight represented significant

unhappiness. Thirty nine of those questioned responded in this way (27%). (Table 3:8).

3.10 CONCLUSIONS

The results of this study confirmed previous studies which showed that burning mouth syndrome affects predominately postmenopausal women. The mean age, 59 years, was remarkably similar to the findings of Basker et al (1978) and Main and Basker (1983) who reported a mean age of 60 years, and by Schoenberg et al (1971) who cited a figure of 58 years. The mean age reported in an earlier study by Schoenberg (1967) was 54 years.

The tongue was confirmed as the most commonly affected site. The site hierarchy was similar to that reported by Main and Basker (1983) for their general dental practice and Leeds and Birmingham dental hospital surveys except that in the present study the throat was marginally more commonly affected than the buccal mucosa.

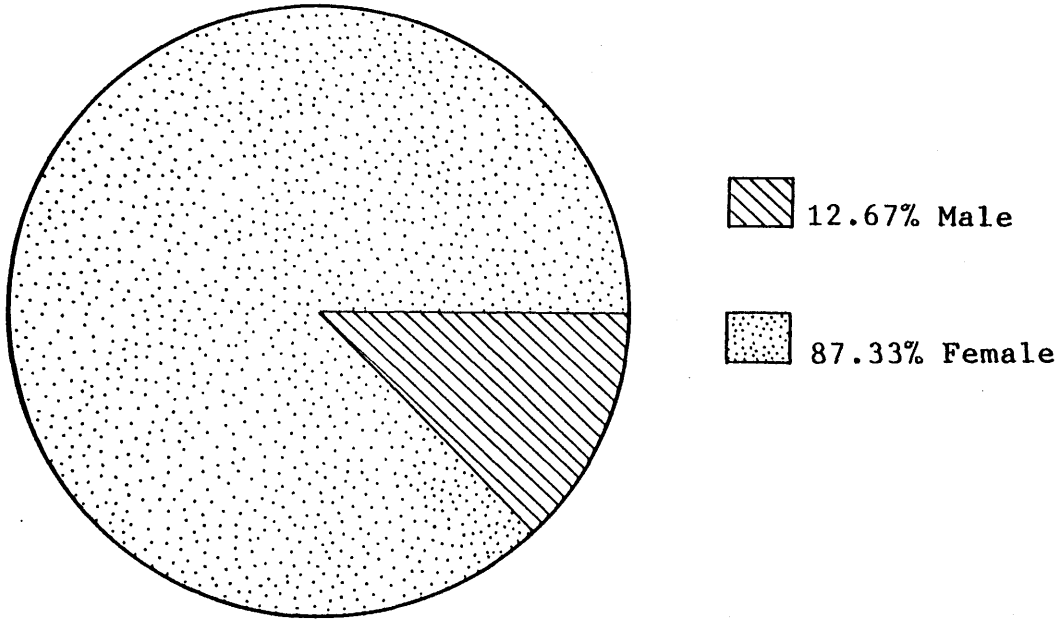


Figure 3:1 Female : Male ratio

Age in Years	Number of Males	Number of Females
31-35	0	2
36-40	0	6
41-45	1	5
46-50	1	10
51-55	1	15
56-60	4	18
61-65	4	31
66-70	5	20
71-75	2	13
76-80	1	10
81-85	0	1

Table 3:1 Age at presentation

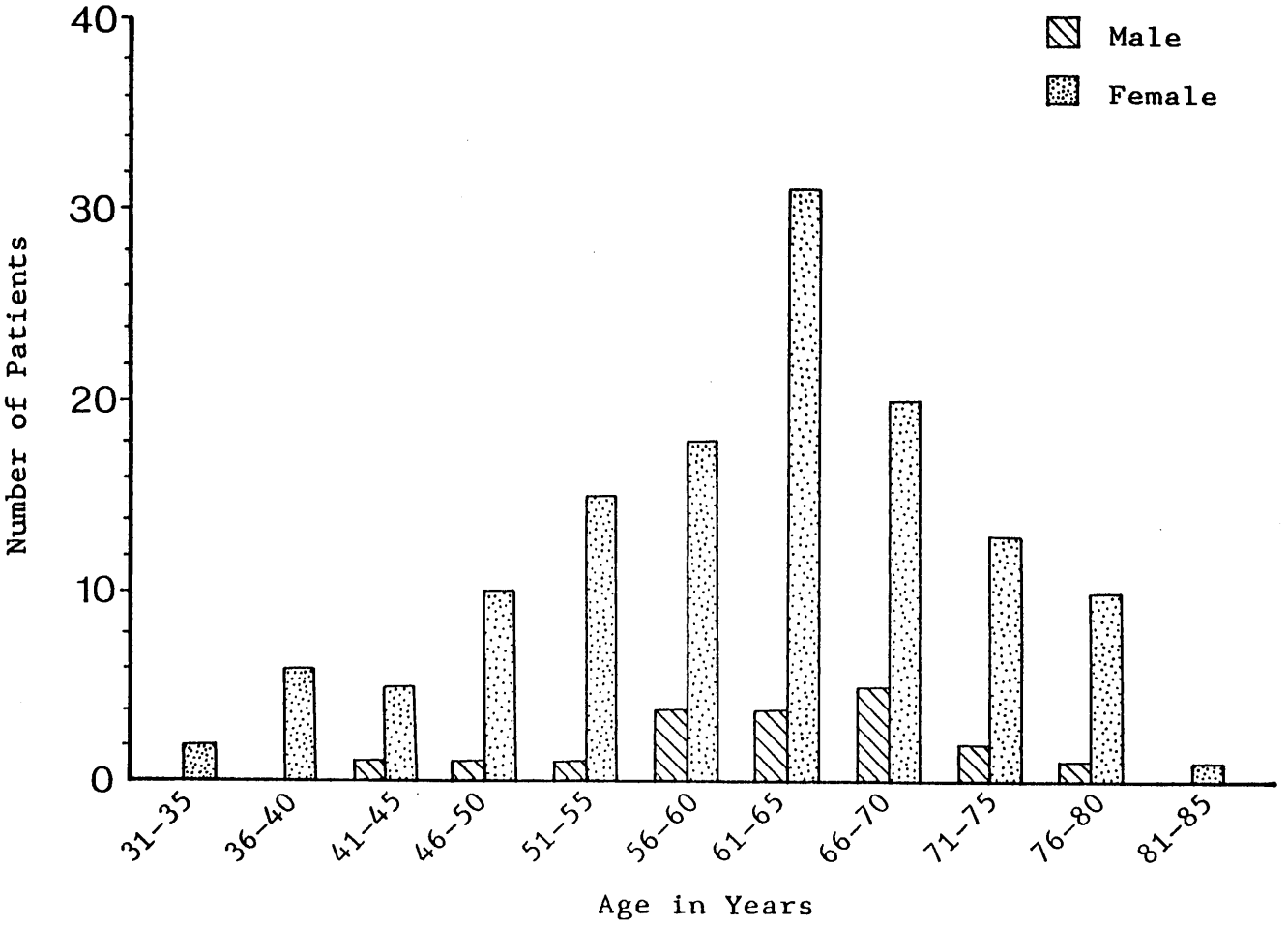


Figure 3: 2 Age at presentation

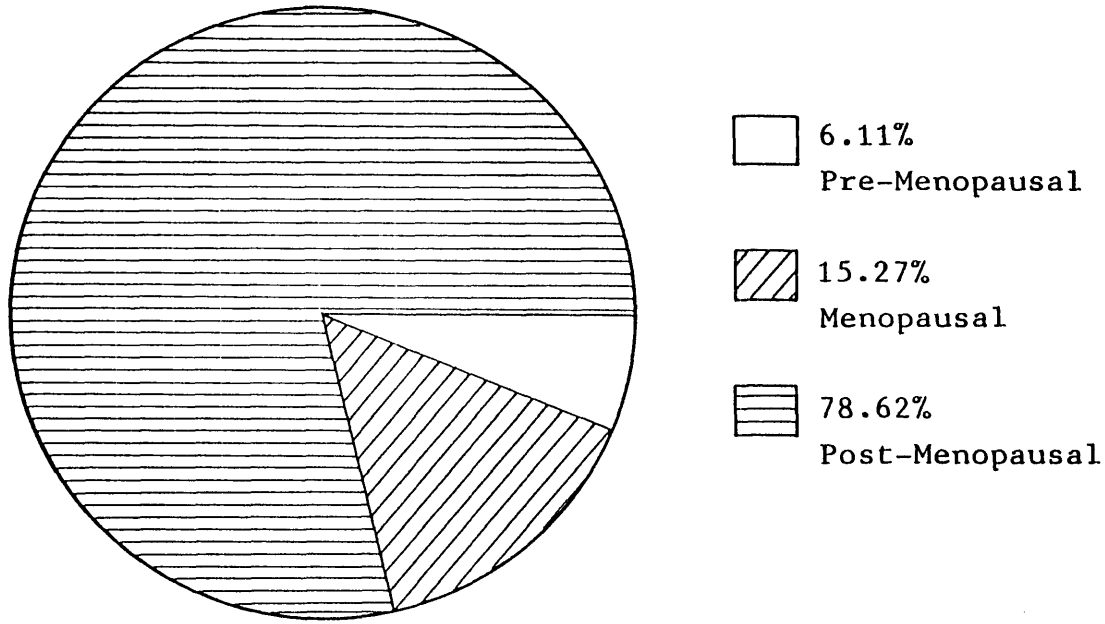


Figure 3: 3 Menopausal status

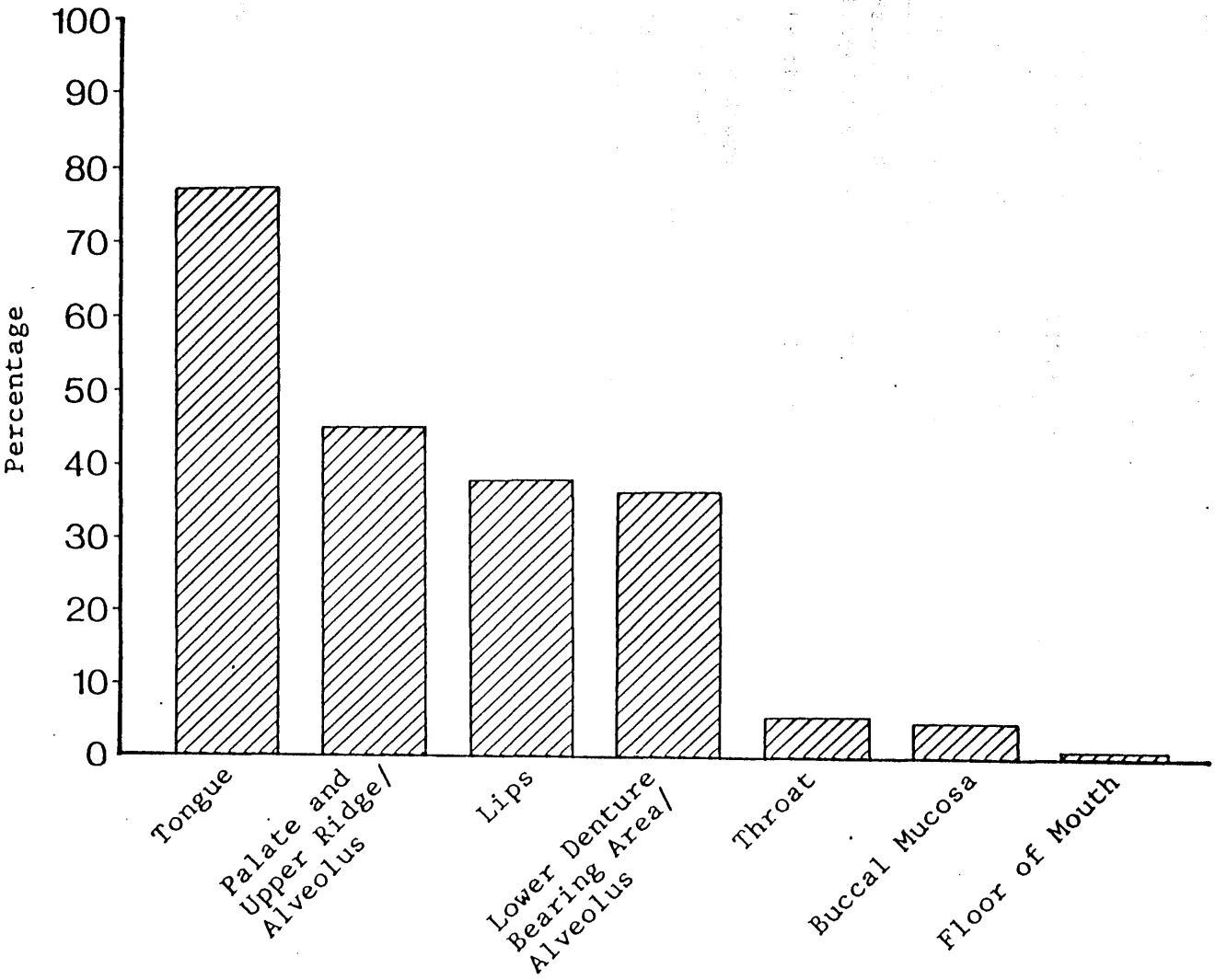


Figure 3:4 Site of burning

Site	Number of patients	Percentage of total	Only site (number)	Percentage of total sample
Tongue	116(100%)	77.33	41	27.33
Dorsum	5(4.31%)	3.33	1	0.66
Tip	15(12.93%)	10.00	6	4.00
Lateral borders	9(7.75%)	6.00	6	4.00
Tip and lateral borders	16(13.79%)	10.66	7	4.66
Tip and dorsum	10(8.62%)	6.66	4	2.66
All three sites	61(52.58%)	40.66	17	11.33
Palate and Upper Ridge/Alveolus	67(100%)	44.66	6	4.00
Palate	20(29.85%)	13.33	3	2.00
Upper Ridge/ alveolus	7(10.44%)	4.66	1	0.66
Both	40(59.70%)	26.66	2	1.33
Lips	58(100%)	38.66	8	5.33
Upper	4(7.01%)	2.66	2	1.33
Lower	10(15.78%)	6.66	2	1.33
Both	44(77.19%)	29.33	4	2.66
Lower Denture Bearing Area or Lower Alveolus	55	36.66	5	3.33
Buccal Mucosa	8	5.33	0	0
Throat	9	6.00	0	0
Floor of mouth	2	1.33	0	0

Table 3:2 Site of burning

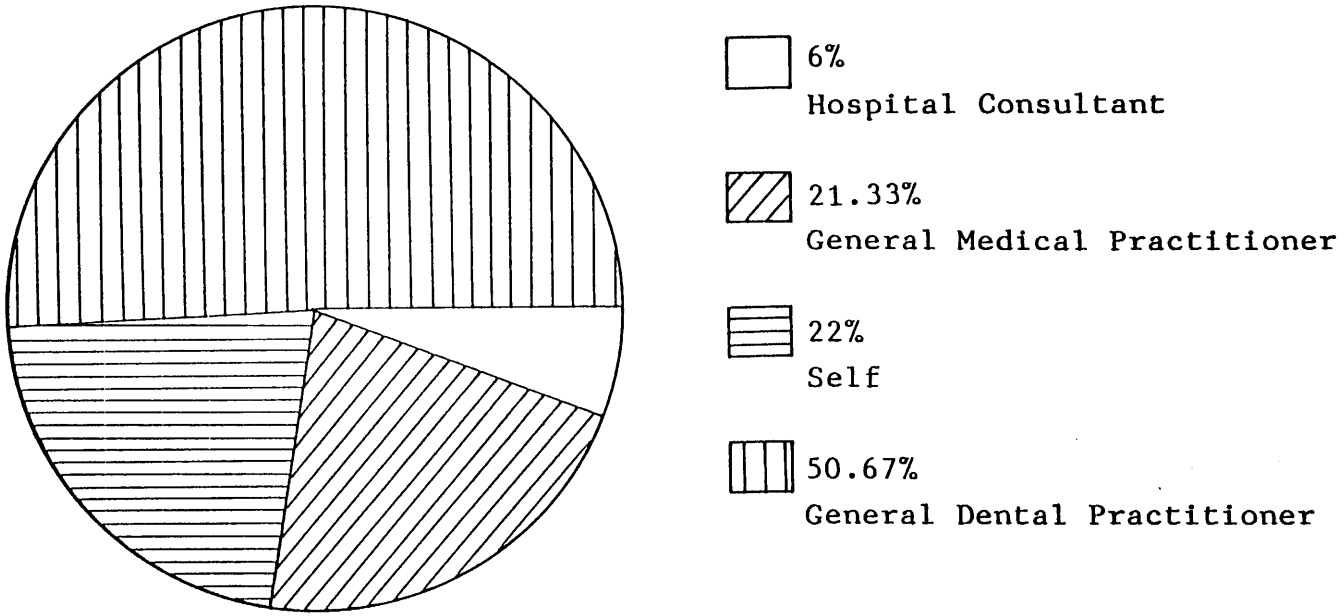


Figure 3: 5 Source of referral

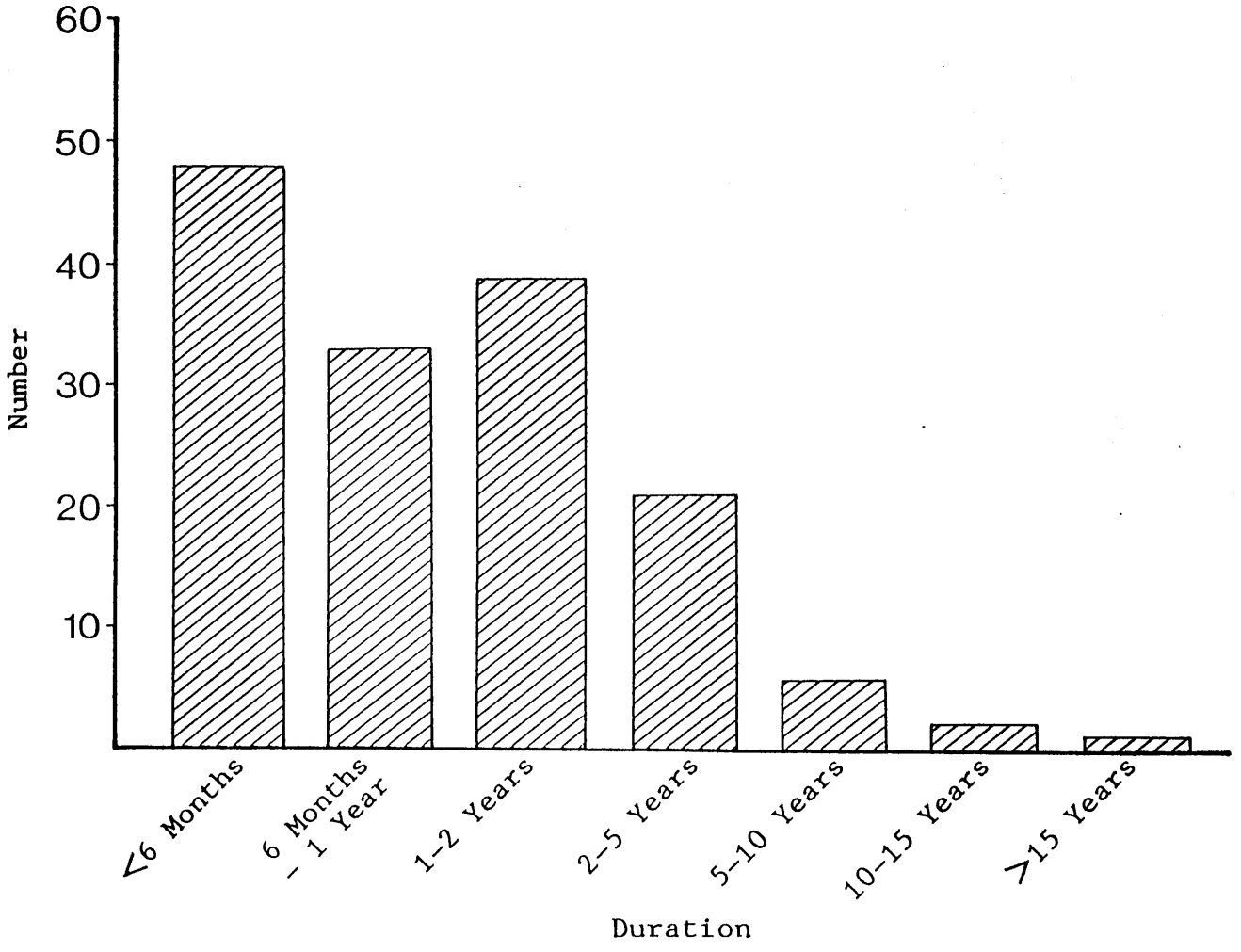


Figure 3:6 Duration of symptoms at presentation

	YES (NUMBER)	NO (NUMBER)	NOT KNOWN (NUMBER)
PRESENCE OF SYMPTOMS ON WAKING	64	83	3
SYMPTOMS BECOMING WORSE AS DAY PROGRESSED	104	43	3
MAXIMUM DISCOMFORT IN THE EVENING	82	63	5
SYMPTOMS CAUSED WAKENING FROM SLEEP	31	103	16
SYMPTOMS CHANGED SLEEP PATTERN	30	99	21

Table 3:3 Symptom Pattern

	Number of Patients
Symptoms not associated with eating or drinking	71
Symptoms improved by eating or drinking	21
Symptoms made worse by eating or drinking	47
Symptoms either improved or made worse by eating or drinking	8
Not known	3

Table 3: 4 Association of symptoms with eating or drinking

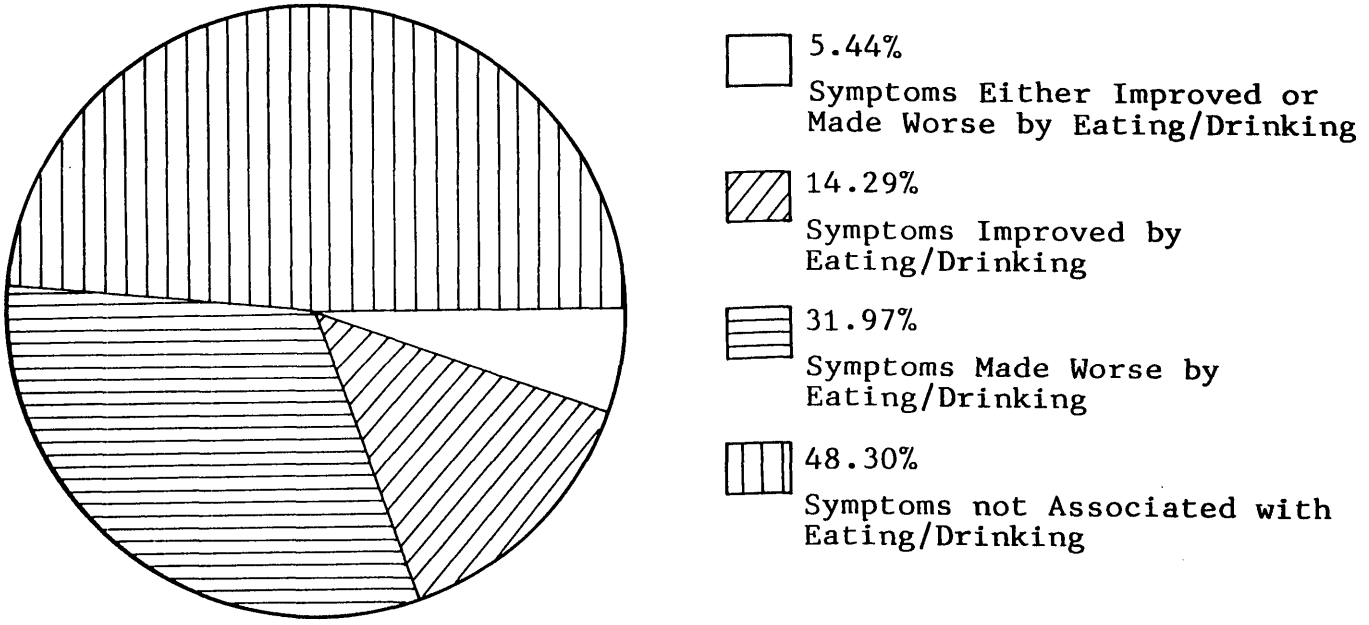


Figure 3: 7 Association of symptoms of BMS with eating and/or drinking

SEVERITY SCORE	NUMBER OF PATIENTS
3	1
4	6
5	19
6	4
7	22
8	30
9	15
10	45

Table 3:5 Number of patients indicating a particular severity score at presentation (n =142)

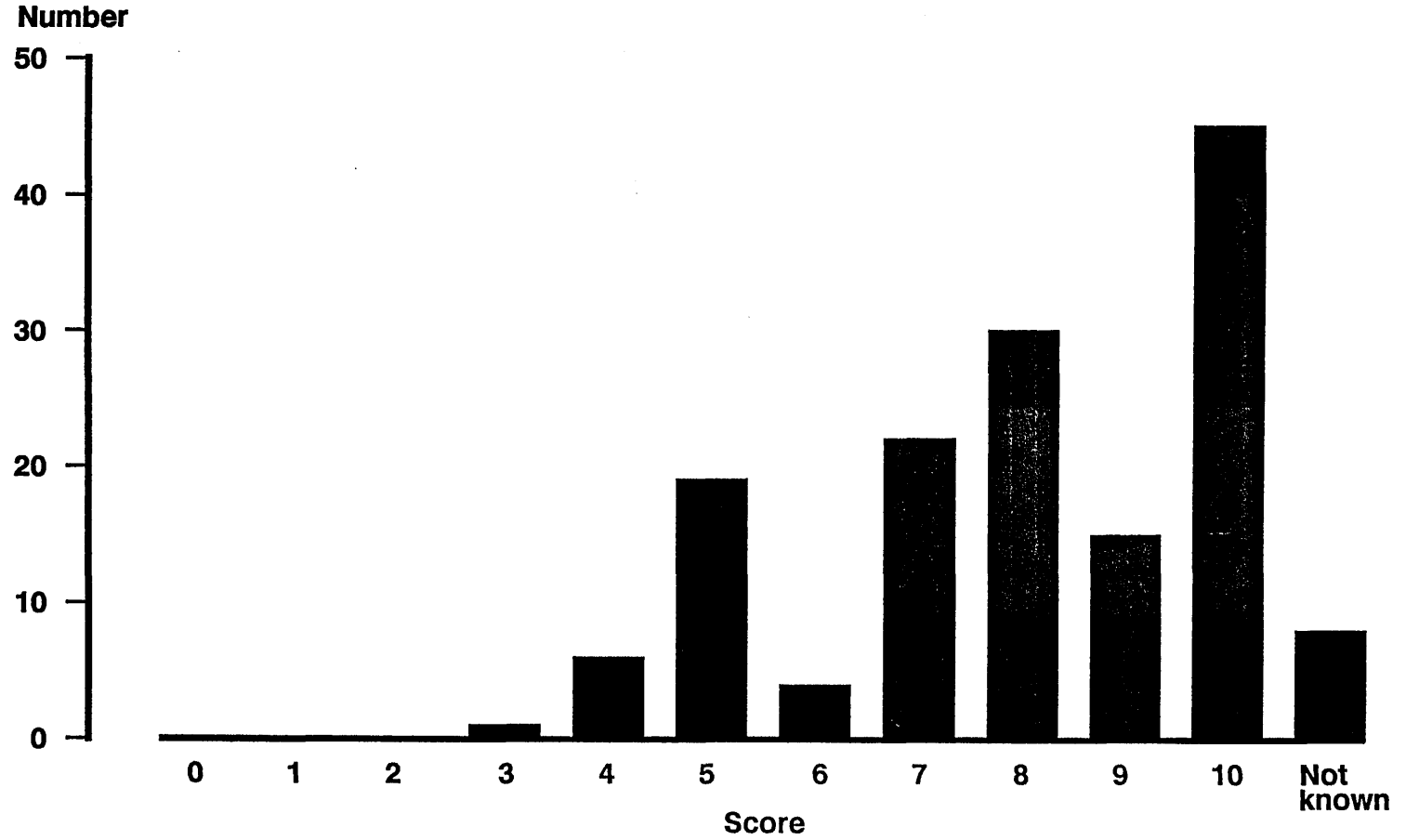


Figure 3: 8 Severity of symptoms on presentation

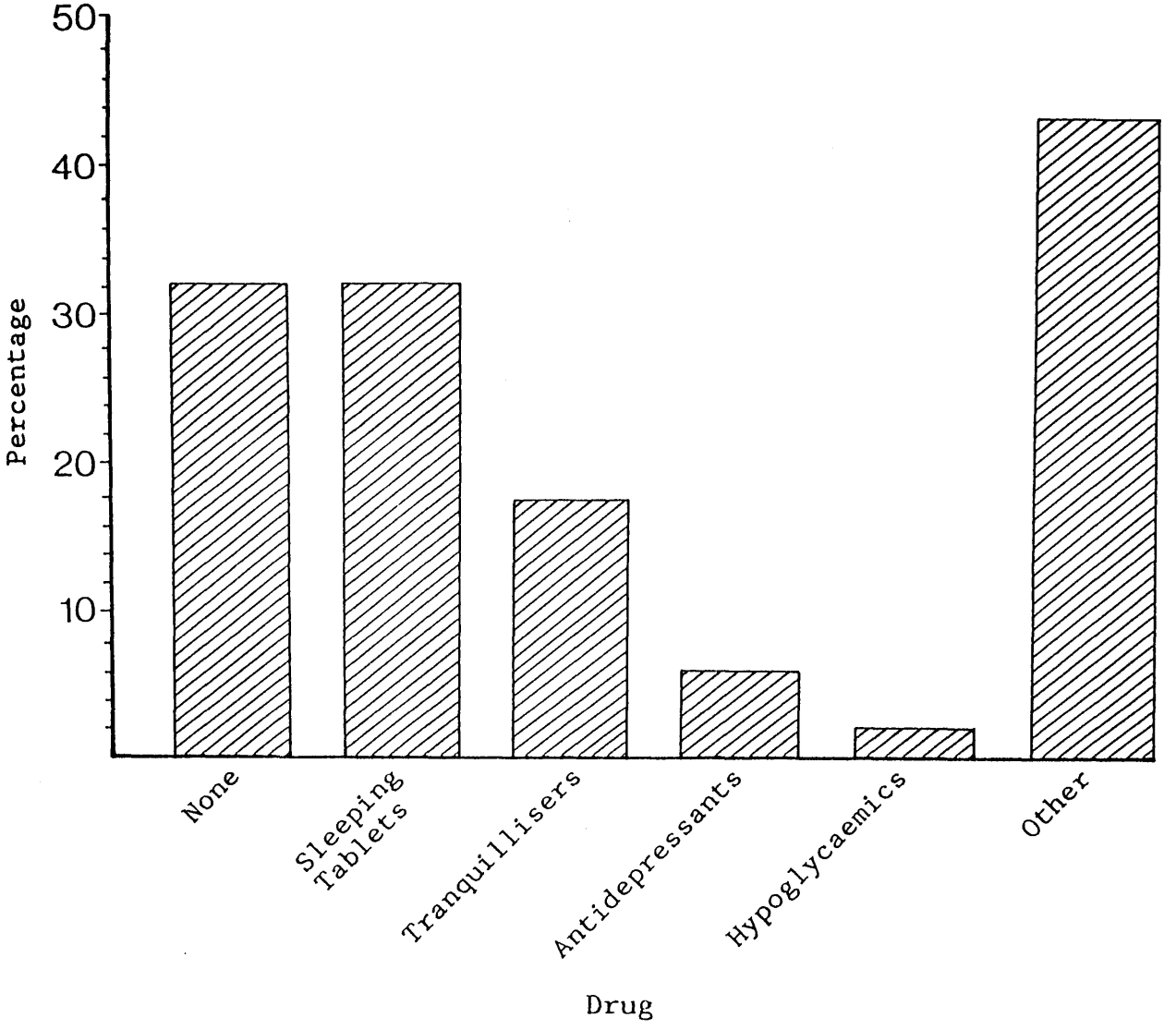
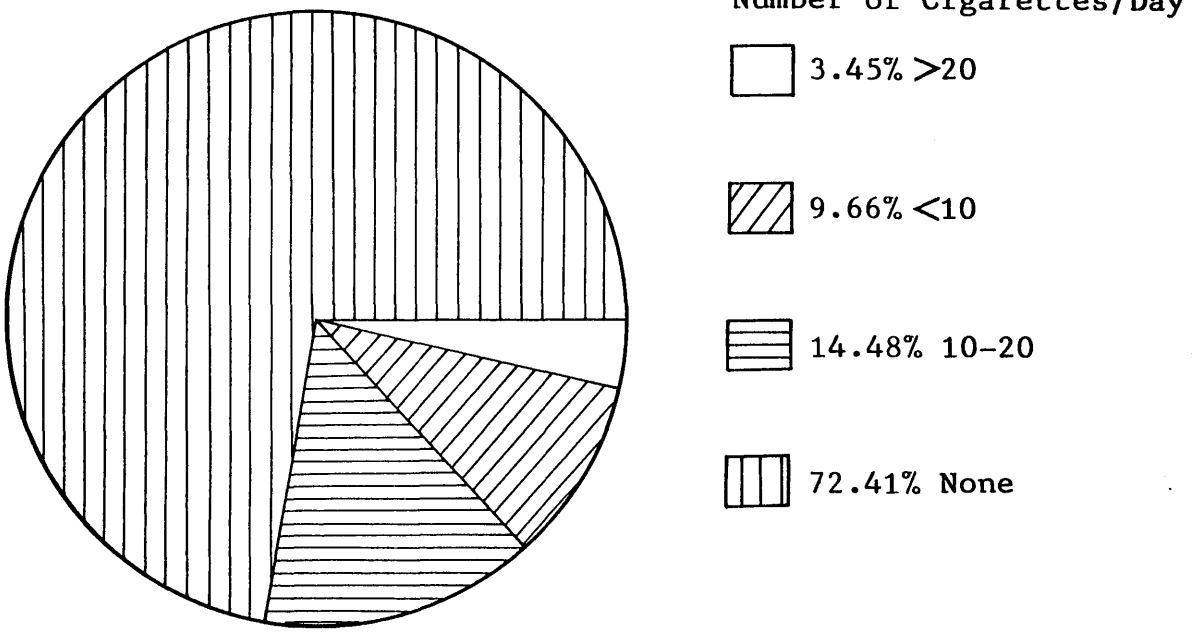


Figure 3:9 Medication at presentation



(n = 145)

Figure 3:10 Smoking habits

Normal	Difficulty getting to sleep	Early Morning wakening	Burning Causing wakening	Burning changed sleep pattern	Total
*					44
*	*				4
*		*			9
*	*	*			2
*	*		*	*	1
*			*		4
*			*	*	1
*				*	2

Table 3:6 Combination of sleep patterns in patients who describe their sleep as normal (n = 67)

Normal	Difficulty getting to sleep	Early Morning wakening	Burning Causing wakening	Burning changed sleep pattern	Total
	*				7
	*	*			20
	*			*	2
	*	*	*		3
	*		*	*	6
	*	*		*	3
	*	*	*	*	9
		*			5
		*		*	1
		*	*		1
		*	*	*	2

Table 3:7 Combination of sleep patterns in patients who described their sleep as abnormal (n = 59)

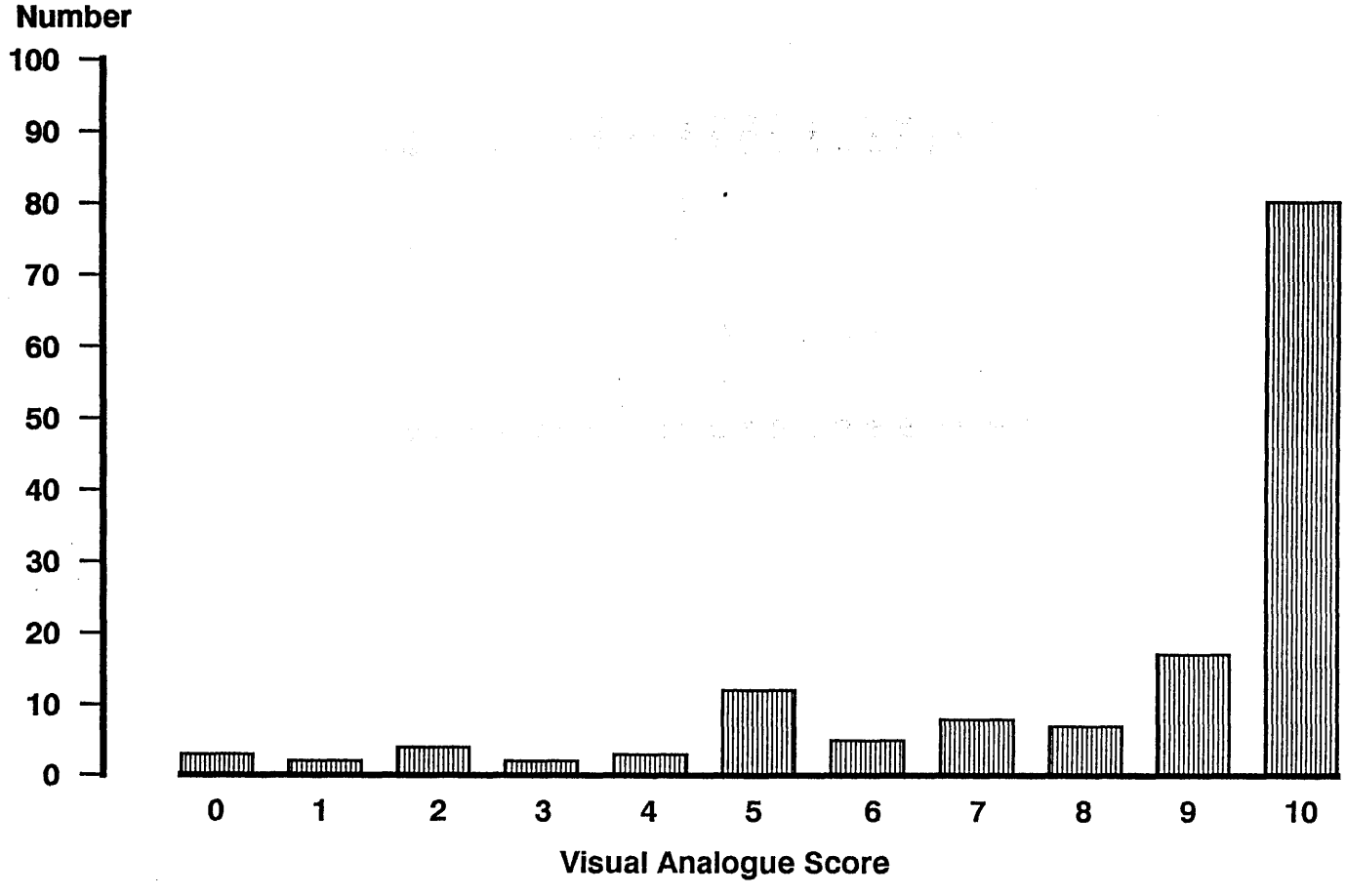


Figure 3: 11 Domestic and social circumstances

PATIENT REFERENCE NUMBER	SOCIAL AND DOMESTIC CIRCUMSTANCES ANALOGUE SCORE
003	02
008	01
011	06
014	05
017	07
020	05
022	05
023	04
027	07
031	07
034	07
046	06
047	05
052	07
053	05
055	05
063	05
064	05
065	0
071	05
074	0
076	02
079	07
083	02
085	06
092	01
098	03
099	04
101	0
104	07
118	05
123	06
124	03
126	02
132	05
138	04
139	07
145	06
150	05

Table 3:8 Analogue score for those patients with adverse social and domestic circumstances

CHAPTER 4

HAEMATOLOGICAL INVESTIGATIONS - RESULTS

4.1 FULL BLOOD COUNT

Estimation of full blood count was undertaken for 146 patients. (Appendix 1).

4.1.1 Mean corpuscular haemoglobin (Hgb)

One female patient (ref no 032) was found to have a mean corpuscular haemoglobin below that of the normal range, being 10.2g/dL. Her film was reported as, "Red cells show anisopoikilocytosis with features of iron deficiency. There is an increase in polychromasia which, in the absence of iron therapy would suggest blood loss". Serum ferritin was found to be 3ng/ml: all other haematological findings were normal and the patient was referred to a consultant physician for investigation of the possible source of blood loss, and the resulting anaemia.

4.1.2 Mean Cell Volume (M.C.V.)

Five patients had a mildly elevated M.C.V.(see Table 4:1 and Figure 4:1) being just outwith the normal range (78-99 fl). The films of two of these patients (ref nos 064 and 083) were reported as showing mild macrocytosis, and the reports of their films raised the question of alcohol abuse. Both of the patients, however, indicated that they were abstainers. All 5 patients had normal values for corrected whole blood folate and serum vitamin B12.

4.2 FILM

Of the 146 patients in whom the film was reported

136 patients had entirely normal films. Table 4:2 shows the findings for the remaining 10 patients.

The patient (ref no 011) with neutropenia had a repeat film undertaken which confirmed the neutropenia and she was referred to a consultant haematologist for investigation. no cause could be found and the patient is under regular review at the haematology clinic.

One patient (ref no 012) with mild thrombocytopenia had been on regular reviews for this condition by her general medical practitioner and the condition had developed following radiotherapy following a mastectomy two and a half years previously. All other haematological parameters were normal.

Patient (ref no 032) has been described in section 4.1.1. Patient (ref no 064) has been described in section 4.1.2 in addition to the macrocytosis her GGT level was 186U/L (normal range 5-50 U/L) further raising the question of alcohol excess.

Patient (ref no 068) was referred to a consultant haematologist who confirmed the elevated platelet count and the presence of a primary myeloproliferative disorder involoving especially megakaryocytes.

The increased platelet count in patient (ref no 071) was confirmed by subsequent films, advice from a consultant haematologist was that there was no need to investigate further.

Patient (ref no 083) had similar findings to patient (ref no 064) in that in addition to an increased MCV reported on the film there was a mildly elevated GGT

level of 72U/L (normal range 5-50 U/L) with other haematological parameters being normal.

In addition to the mild anaemia reported in the film for patient (ref nor 124) he had a borderline vitamin B12. normal MCV and haemoglobin.

4.3 SERUM FERRITIN

Serum ferritin was measured for 139 patients. Eight patients (5.75%) were deficient in iron having Serum ferritin levels below the normal range (12-300 ng/ml) see Table 4:3. It can be seen that 4 of the patients were aged less than 45 years representing 31% of patients in the study group below this age.

All patients were referred to a consultant haematologist to investigate the causes of the deficiency.

4.4 SERUM VITAMIN B12

Serum Vitamin B12 was estimated for 147 patients. Twelve patients (Table 4:4) had results below the normal range (175-800 pg/ml) representing 8% of those tested. None of these patients had an abnormal MCV and on the advice of a consultant haematologist they were treated with a six week course of weekly vitamin B12 injections (1000 µg of cyanocobalamin). Serum vitamin B12 estimations were repeated three months later and were within the normal range.

4.5 CORRECTED WHOLE BLOOD FOLATE

Corrected whole blood folate was investigated for 145 patients none of whom were found to be deficient in folic acid.

4.6 CONCLUSIONS

In this study haematological deficiencies were present in a small but important proportion of patients. Comparison with other studies is particularly complicated in this area as most authors have included the discussion of burning symptoms with patients who have visible oral changes. However, since only 6% of the patients were iron deficient this represents a much lower incidence than reported by Brooke and Seganski (1977) who claimed 53% of such patients were iron deficient, with 3 of their 55 patients being anaemic. The patients in the study of Brooke and Seganski were of a similar age distribution and sex ratio to the present study and no oral changes were reportedly present. Also in the latter study vitamin B12 and folate levels were only measured if initial haematological studies indicated a possible deficiency and none of their patients were vitamin B12 deficient while 4% were folate deficient. In the present study 8% of patients were vitamin B12 deficient and none of those had an abnormal MCV. This finding supports the opinion that serum vitamin B12 should be routinely estimated and assessment of haemoglobin and MCV alone is insufficent. None of the patients in the present study were deficient in folic acid.

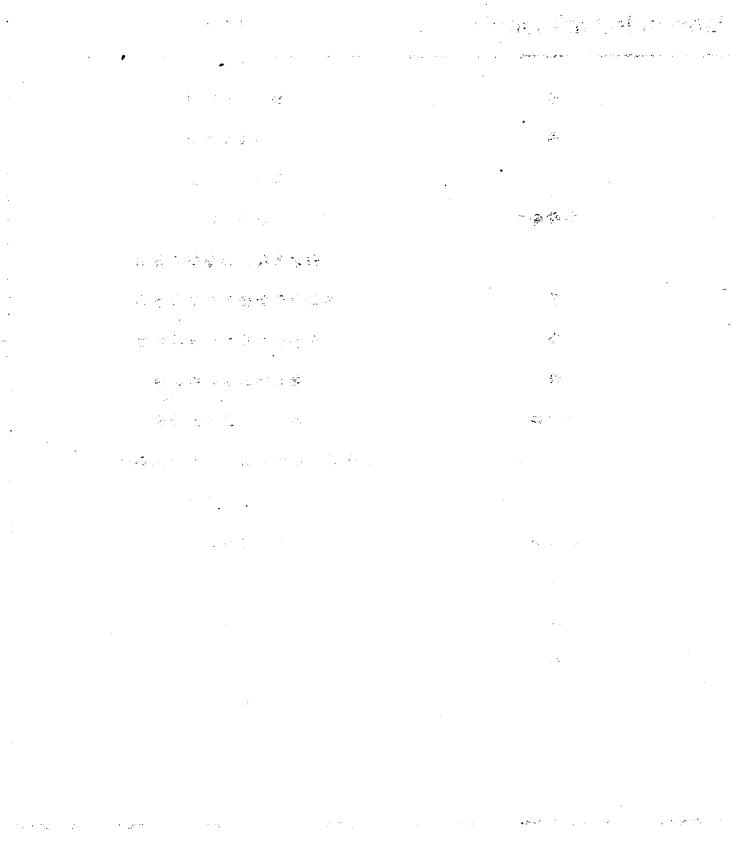
Comparison with the studies of Basker et al (1978), Main and Basker (1983), and Zegarelli (1984) in which haematological deficiencies were reported in 33%, 40% and 0.5% of patients respectively is not possible since mucosal changes were reported in patients included

in those papers.

However, since mucosal discomfort in patients with mucosal change is well known (Hjörting-Hansen and Bertram 1968, Hardisty and Weatherall 1974, and Dolby 1975), it is possible that BMS represents an early stage in the development of such deficiencies.

Comparison of the haematological findings of this study with what might be expected in the general population is difficult. There have been few studies undertaken on an epidemiological basis in the United Kingdom, the last being that of McFarlane et al(1967) in Glasgow. In that study total iron binding capacity and percentage saturation was employed as an indicator of iron depletion and of iron status. McFarlane et al (1967) reported that 22.2% of female patients attending a general medical practice were iron deficient compared to 16.3% of controls. Both figures are considerably higher than the 6% found in the present study. As for iron deficiency anaemia, 9.7% of their female patients and 2% of controls had positive findings compared to 0.6% in the present BMS group. It is likely that in the intervening years medical care and diet has improved which might account for a lowering of the incidence of iron deficiency in the general population. In addition, the use of serum ferritin as a more accurate indicator of iron status may also have played a role. The 1967 study of McFarlane et al showed no differences between the incidence of deficiency in pre and postmenopausal women. Vitamin B12 deficiency was found in only 1% of the patients in the

1967 survey compared to 8.16% in the BMS group. The significance of these haematological deficiencies are discussed in Chapter 11.



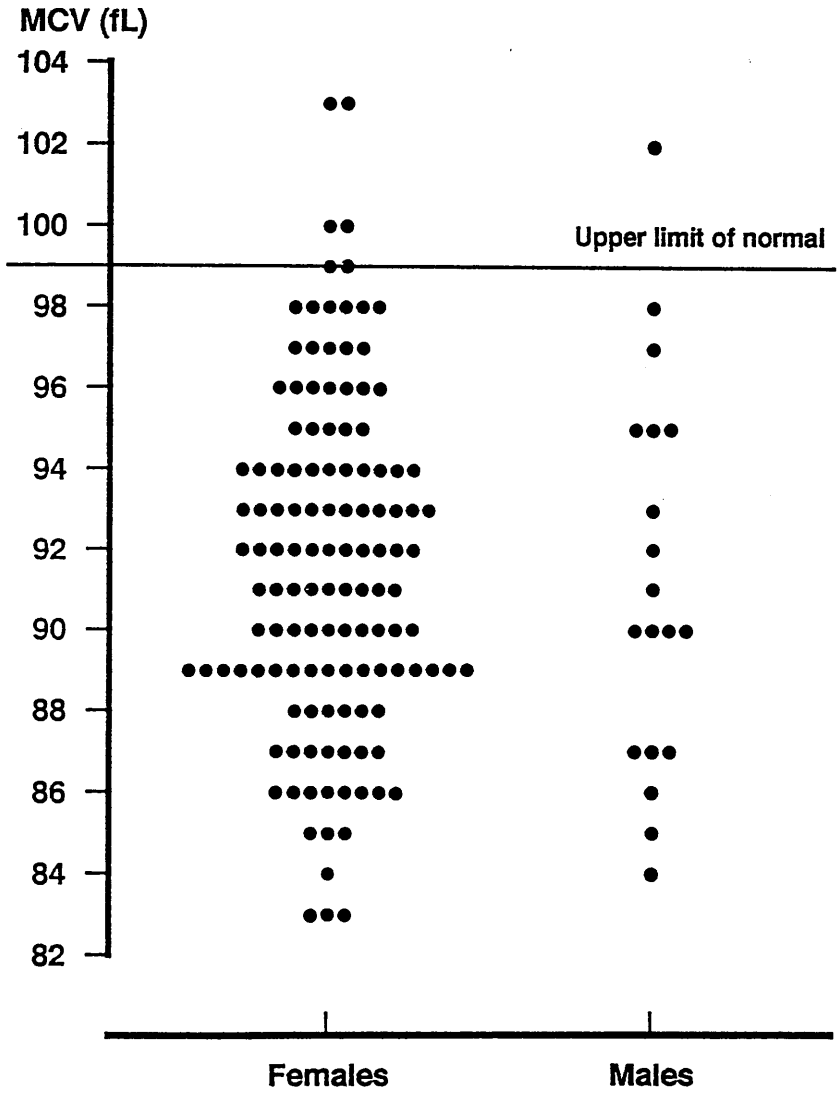


Figure 4: 1 Number of patients / MCV

Patient reference number	Sex	Age	MCV	Menopausal status
050	F	59	100	Post
064	F	47	103	Post
083	F	70	103	Post
105	F	46	100	Menopausal
126	M	60	102	--

Table 4:1 Sex, age and hormonal status of those patients with an elevated MCV

PATIENT REFERENCE NUMBER	FILM REPORT
011	Red cells normochromic and normocytic. Total white cell count is reduced with the following differential: Neutrophils 50%, Lymphocytes 45% and Monocytes 5%.
012	Platelets reduced (mild thrombocytopenia) 100,000. Red cells normochromic and normocytic. White cell series is normal.
032	Red cells show anisopoikilocytosis with features of iron deficiency. There is an increase in polychromasia which in the absence of iron therapy would suggest blood loss.
064	Raised MCV with generalised macrocytosis as seen in the hepatic dysfunction of alcohol excess. White cell series is normal.
068	Red cells show anisocytosis with variable haemoglobinisation. Fragmented cells and elongated cells are also seen. White blood cells appear essentially normal. Platelets are markedly increased and anisothrombia is noted. Appearances suggest a myelo-proliferative disorder and further investigation is indicated.
071	Platelets increased in an otherwise normal film.
083	Red cells show mild round macrocytosis. In the absence of hepato-biliary dysfunction B12 and folate assays would be of value. White cells and platelets are normal.
100	Total white cells count is reduced with differential: Neutrophils 80%, Lymphocytes 14% Monocytes 3%, Eosinophils 3%. Platelets are normal.
120	Red cells normochromic and normocytic. White cell series is normal. Platelet count is reduced and there is anisothrombia.
124	Mild anaemia. Red cells are normochromic and normocytic.

Table 4:2 Film reports for those patients with abnormal films (n = 10)

Patient reference number	Sex	Age	Serum ferritin ng/ml	Menopausal status
032	F	35	3	Pre
047	F	78	5	Post
080	F	40	6	Menopausal
092	F	64	11	Post
107	F	43	3	Menopausal
113	M	58	12	--
131	F	68	10	Post
147	F	41	4	Pre

Table 4:3 Sex age and hormonal status of those patients with reduced serum ferritin levels.

Patient reference number	Sex	Age	Serum Vitamin B12 pg/ml	Menopausal status
020	F	78	105	Post
037	F	62	156	Post
056	F	49	162	Menopausal
057	F	55	124	Post
071	F	54	155	Post
077	F	64	170	Post
082	F	69	133	Post
124	M	66	136	--
131	F	68	71	Post
133	F	80	158	Post
146	F	67	129	Post
149	F	68	*	Post

* >2000pg/ml. This patient had been treated for low vitamin B12 prior to referral.

Table 4:4 Age, sex and hormonal status of those patients with low serum vitamin B12

CHAPTER 5

BIOCHEMICAL INVESTIGATIONS - RESULTS

5.1 PLASMA GLUCOSE ESTIMATION

Plasma glucose was estimated for 128 patients (appendix 1). Eleven patients (8.6%) had raised fasting blood glucose concentrations (>6.5 mmol/L). The glucose tolerance test was abnormal in 4 patients, normal in another 4 and the other 3 patients had known maturity onset diabetes with random blood glucose concentrations of 7.7 mmol/L, 14.2 mmol/L and 14.7 mmol/L (Table 5:1). One further patient (ref no 006) with a normal fasting plasma glucose estimation on presentation (4.9 mmol/L) developed overt maturity onset diabetes while under review (fasting plasma glucose estimation 9.0 mmol/L), and one known diabetic (ref no 110) had good glycaemic control (fasting plasma glucose estimation 5.6 mmol/L).

5.2 VITAMIN ASSAYS

Vitamin assays were undertaken for only 67 patients (45%) as indicated in Chapter 2.5.

5.2.1 Vitamin B1, B2 and B6

Table 5:2 shows the results of the assays with those results indicating deficiency underlined.

Forty two patients (62%) had normal vitamin B complex assays, vitamin B1 deficiency was found in 11 patients (18%), vitamin B2 in 8 (12%) and vitamin B6 in 14 (19%). Seven of the patients (10%) were deficient in more than one vitamin of the B complex group.

5.2.2 Vitamin A, C, D and E

Sixty-six patients had assays undertaken for these vitamins. Table 5:3 shows the results of the assays with those results indicating deficiency underlined.

Twenty nine patients (41%) were deficient in vitamin A. 7 (11%) in vitamin C. 10 (15%) in vitamin D. and 2 (3%) in vitamin E.

5.3 CONCLUSIONS

In this study 4 patients. 3% of those tested had previously undiagnosed maturity onset diabetes confirmed. This incidence is much lower than that reported by Chinn et al (1965) and Brody et al (1971) at 27% and 39% respectively. It is likely that once again these figures are distorted because of the inclusion of patients with conditions other than BMS. Basker et al (1978) would appear to confirm a lower incidence. In their survey of oral complaints amongst patients with maturity onset diabetes mellitus attending a diabetic clinic only 10% of these patients had complained of burning mouth or strange tastes. Of the 21 patients presenting with burning mouth (not necessarily BMS) only 1 had unrecognised diabetes mellitus (4.7%). Main and Basker (1983). reporting on 37 patients. found 1 patient (2.7%) with undiagnosed diabetes mellitus.

Basker et al (1978) and Main and Basker (1983) therefore report an incidence of undiagnosed diabetes mellitus similar to that found in the present study. It is likely that this is a small but important finding as the predicted level of undiagnosed diabetes mellitus in the general population is 0.5% (Baird 1987).

Lamey et al (1986) showed that 51% of patients with BMS were deficient in vitamin B1. B2 or B6 or a combination of these vitamins. however some patients had been excluded from the total study group because they did not have either normal haematological parameters or normal stimulated parotid flow rates or had significant isolates of candidal species. In the present study 38% of patients had abnormal assays for vitamin B1. B2 or B6 or a combination of these vitamins.

No other reports appear in the literature in which assays of the vitamin B complex have been completed in patients with BMS. Similarly there are no reports of assays for vitamin A. C. D. and E having been undertaken for patients with BMS and their clinical significance is unproven.

PATIENTS REFERENCE NUMBER	FASTING PLASMA GLUCOSE CONCENTRATIONS (mmol/L)	PREVIOUSLY DIAGNOSED AS MATURITY ONSET DIABETIC	ABNORMAL GLUCOSE TOLERANCE TEST
015	6.7	NO	NO
028	14.2	YES	-
031	7.3	NO	YES
037	6.5	NO	NO
066	6.5	NO	NO
081	17.7	NO	YES
089	11.1	NO	YES
091	11.8	NO	YES
119	7.1	NO	NO
140	7.7	YES	-
142	14.7	YES	-

Table 5:1 Fasting plasma glucose concentrations for those patients in which the finding was 6.5 mmol/L or greater.

RED CELL ACTIVATION EFFECT

PATIENTS REFERENCE NUMBER	VITAMIN B1 (NORMAL <25%)	VITAMIN B2 (NORMAL <23%)	VITAMIN B6 (NORMAL <20%)
003	5	2	8
005	4	16	13
009	2	11	14
013	11	6	11
014	17	3	7
017	3	13	5
018	60	7	21
019	29	5	20
020	4	7	3
021	11	18	13
023	17	4	4
024	9	30	3
026	10	5	5
029	21	14	13
030	20	7	11
031	27	17	3
036	12	24	11
040	15	2	25
041	12	9	6
045	22	39	6
046	3	13	7
048	11	9	10
049	7	8	17
052	22	103	35
053	15	13	47
054	21	12	20
055	27	6	23
061	6	3	22
063	71	3	7
066	18	9	7
067	40	65	7
068	22	21	4
069	29	3	11
070	9	3	7
072	14	1	9
073	19	2	22
077	9	4	11
081	11	4	6
082	4	3	7

Table 5: 2 Vitamin B1. B2 and B6 assays.

RED CELL ACTIVATION EFFECT

PATIENTS REFERENCE NUMBER	VITAMIN B1 (NORMAL <25%)	VITAMIN B2 (NORMAL <23%)	VITAMIN B6 (NORMAL <20%)
085	4	7	22
088	6	14	126
092	9	13	4
093	17	6	5
094	9	23	11
097	20	6	29
098	8	29	8
100	8	10	7
101	54	8	2
102	10	7	15
104	11	2	83
107	50	32	8
113	11	3	6
115	9	3	6
116	25	4	10
117	19	14	16
118	11	2	10
119	7	2	14
124	80	2	43
125	9	10	16
127	6	10	12
128	3	7	19
132	20	8	75
133	11	14	11
135	12	5	10
137	13	50	29
138	52	5	14
139	5	18	5

Table 5:2 Vitamin B1 B2 and B6 assays.

PLASMA VITAMIN

PATIENT REFERENCE NUMBER	A (NORMAL RANGE 1-2.8 µmols/L)	C (NORMAL >12 µmols/L)	D (NORMAL RANGE 10-50 ng/ml)	E (NORMAL RANGE 14-39 µmols/L)
003	3.0	24.5	10.8	22.9
005	2.1	41.0	17.8	17.9
009	2.6	17.0	19.6	17.8
013	2.9	15.9	44	20.7
014	3.9	15.9	17	19.4
017	2.9	9.0	13.1	24.4
018	2.0	17.0	14	23.1
019	3.9	13.6	29.0	28.4
020	2.6	7.9	18	21.0
021	3.8	23.9	24.0	20.7
023	1.7	-	36	23.5
024	2.4	15.9	8.6	13.3
026	1.8	32.9	61	25.9
029	2.0	20.4	33	19.5
030	2.1	25.6	52	22.2
031	3.8	28.0	16	26.7
036	3.2	15.9	12	16.9
040	2.8	13.6	27	13.4
041	4.1	15.8	27	20.5
045	2.2	30.7	40	21.0
046	3.8	27.3	20	19.6
048	4.1	19.3	23	18.0
049	2.4	13.6	20	18.4
052	3.2	18.2	12.8	25.3
053	2.7	18.2	25	25.6
054	3.4	43.2	35	19
055	3.2	34.1	21.5	29.5
061	2.4	11.4	45	19.7
063	3.1	18.2	24.2	25.4
066	2.6	29.5	16	25.3
067	1.8	27.3	55	21.2
068	3.5	45.4	48	22.7
069	2.9	22.7	INS	22.9
070	2.6	17.0	21	25.9
072	3.2	40.1	24.5	26.1
073	2.3	21.6	63	22.7
077	4.2	22.7	110	25.4
081	3.4	13.6	23	22.9
082	2.2	27.3	33	17.6

Table 5:3 Vitamin A, C, D and E assays.

PLASMA VITAMIN

PATIENT REFERENCE NUMBER	A (NORMAL RANGE 1-2.8 µmols/L)	C (NORMAL >12 µmols/L)	D (NORMAL RANGE 10-50 ng/ml)	E (NORMAL RANGE 14-39 µmols/L)
085	3.4	15.9	41	24.9
088	4.7	4.5	14.7	33.0
092	3.5	59.4	19.1	17.9
093	2.4	21.6	44	27.6
094	3.1	21.6	11.4	21.0
097	2.5	18.2	31	20.5
098	-	-	-	-
100	2.1	15.9	17	20.0
101	3.6	13.7	21.3	27.3
102	2.2	17.0	88	19.0
104	3.2	18.2	8	22.9
107	2.2	15.9	46	24.4
113	2.1	12.5	36	24.1
115	2.4	23.9	25	27.6
116	1.9	25.0	28	23.4
117	2.5	11.4	16.3	14.0
118	1.9	34.1	20	29.9
119	3.5	20.2	16.1	28.0
124	1.6	17.0	16	20.4
125	1.6	15.9	17	22.8
127	4.4	11.4	38	21.0
128	2.8	15.9		23.4
132	1.8	20.4	10	22.8
133	2.5	11.4	66	25.8
135	2.5	25	7	22.4
137	3.4	15.9	20	28.3
138	2.3	15.9	25	21.5
139	2.0	18.2	16.3	19.7

Table 5:3 Vitamin A, C, D and E assays.

CHAPTER 6

MICROBIOLOGICAL INVESTIGATIONS - RESULTS

6.1 ORAL RINSE

This investigation was undertaken for 128 patients. 112 females and 16 males (F:M. 7:1). The results were compared with those of a control group of 90 females and 13 males (F:M. 6.92:1) who were matched for age and denture status attending for routine denture replacement.

None of the test or control patients were receiving antibiotic or steroid therapy.

6.1.1 *Candida* Species

Table 6:1 shows the oral carriage of *Candida* species in both the BMS patients and the controls.

The oral carriage rate of *Candida* species in the BMS patients was 32% (41 of 128) as compared with 21% (22 of 103) of the control population. In only 70% of BMS patients however was the candidal count greater than 100 colony forming units per ml of the oral rinse. a level which is thought to be clinically significant (Mason and Willoughby. 1986). The predominant *Candida* species isolated from both groups was *Candida albicans* while *C. glabrata* and *C. tropicalis* were isolated less frequently. A variety of other *Candida* species were isolated (Table 6:1).

6.1.2 Coliform Species

The identity and frequency of coliforms isolated from the BMS patients and the control group are shown in Table 6:2. The most common coliforms isolated from either group were *Enterobacter* species followed by *Klebsiella*

species. Twenty-one per cent of BMS patients (27 of 128) harboured coliforms intra-orally as opposed to 12% (12/103) of the healthy controls.

On further analysis it was noted that 63% (17 of 27) of the test samples which yielded coliforms had greater than 40 colony forming units (cfu's) of coliforms/ml of oral rinse as compared with 42% (5 of 12) of control samples. The results indicated that a greater proportion of BMS patients harbour larger numbers of coliforms intra-orally than the control population.

Seven of the 16 BMS males (44%) carried coliforms compared with 2 of the 13 control males (15%). Twenty of the 112 BMS females (18%) carried coliforms compared with 10 of the 90 control females (11%). The only finding in this part of the study that was statistically significant was that a higher proportion of male patients with BMS carried coliforms compared with female BMS patients ($p < 0.05$, chi-squared test).

6.2 CONCLUSIONS

A greater proportion of BMS patients were shown to harbour Candida species and coliforms intra-orally than did a healthy control population (not statistically significant, chi-squared test).

The candidal carriage rate of 21.35% among the control population agrees well with the value of 17.7% quoted by Odds (1988) for similar populations. It may be that the increased oral prevalance of Candida in 32% of BMS patients is a noteworthy finding, however only 23% of them had counts greater than 100 colony forming units.

Haematological factors and xerostomia have been

shown to be related to increased oral carriage of Candida (Parvinen and Larmas, 1981 and Challacombe, 1986). In this study there was no significant association of candidal carriage with those patients who had haematological deficiencies or reduced salivary gland function (Table 6:3). (chi-squared test).

Comparison of these findings with other candidal studies in BMS and in the normal population is difficult since common sampling methods have not been employed. For this reason a control group was included for this part of the study. Also other studies have reported a lower percentage carriage rate than the 17.7% quoted by Odds (1988) for normal populations. Brooke and Seganski (1977) reported a value of candidal carriage in patients with burning symptoms of 13%, Zegarelli (1983) 3.5% and Alteras and Cojocaru (1969) 10.5%. Once again it has to be emphasised that these studies were not restricted to patients with true BMS.

The significance of the oral carriage of coliforms in BMS particularly in male patients is uncertain, it is possible that there was a link between cancerphobia and oral coliform carriage. These patients might repeatedly examine their mouths and translocate coliforms from nailbeds and fingers into the mouth, however only 8 of the 28 patients who expressed a fear that burning symptoms represented cancer carried oral coliforms. As with carriage of candidal species there was no significant association between coliform carriage and haematological deficiencies or reduced salivary gland function (Table

6: 3) . Enterobacter species and Klebsiella species were the most frequent coliforms isolated concurring well with the results of other investigations for the normal population (Rosenthal and Tager, 1975; and Whalin and Holm. 1988).

It has been shown that Klebsiella species promote candidal colonisation of the epithelia (Makrides and MacFarlane. 1982; and Centeno et al 1983), however in these present BMS patients there was no correlation between candidal carriage and coliform carriage, (chi-squared test).

	Number of Occasions Isolated	
Candida spp.	Test	Controls
C. albicans	33	18
C. glabrata	4	3
C. tropicalis	4	2
C. pseudotopicalis	3	-
C. parapsilosis	3	-
C. guillermondii	1	1
C. krusei	-	1
Saccharomyces cerevisiae	2	-

Table 6:1 The identity and frequency of isolation of Candida species from the BMS patients and the controls.

Coliform spp.	Number of Occasions Isolated	
	Patients	Controls
Enterobacter cloacae	7	5
Enterobacter agglomerans	4	4
Enterobacter sakazaki	1	-
Klebsiella pneumoniae	1	4
Klebsiella oxytoca	3	-
Klebsiella ozonae	1	-
Acinetobacter calcoaceticus	2	1
Proteus mirabilis	2	-
Escherichia coli	3	1
Serratia liquefacines	2	-
Serratia maracesens	1	-
Citrobacter freundii	-	1
Pseudomonas aeruginosa	1	-
Pseudomonas paucimobilis	1	-
Pseudomonas maltophila	2	-
Aeromonas hydrophila	1	-

Table 6:2 The identity and frequency of isolation of coliforms from the BMS patients and the controls.

PATIENT REFERENCE NUMBER	CANDIDAL SPECIES cfu/ml	COLIFORMS cfu/ml	RIGHT PAROTID FLOW RATE ml/min	LEFT PAROTID FLOW RATE ml/min	SERUM FERRITIN ng/ml	SERUM VITAMIN B12 pg/ml
002	N	100	-	-	18	438
004	540	N	0.4	0.5	14	285
005	200	N	0.6	0.5	48	194
012	10	N	1.2	1.0	60	382
013	N	80	0.9	1.3	102	621
014	20	N	0.9	1.0	66	542
015	N	1000	1.2	1.2	169	>1000
016	60	N	>0.7	>0.7	75	322
017	80	40	0.2	0.7	153	326
018	100	N	>0.7	>0.7	22	326
020	N	80	>0.7	>0.7	193	105
030	80	N	0.5	0.5	91	574
033	1000	140	-	-	47	261
035	260	N	0.9	0.8	72	459
036	1000	200	>2.0	>2.0	68	254
045	40	N	1.4	0.8	126	268
046	N	60	1.2	1.2	109	312
049	1000	N	0.5	0.4	28	340
051	80	N	>2.0	>2.0	34	390
053	200	N	-	-	58	254

N = NONE ISOLATED

Table 6:3 Flow Rates and Haematological Indicies for those patients carrying Candida species and/or Coliforms.

PATIENT REFERENCE NUMBER	CANDIDAL SPECIES cfu/ml	COLIFORMS cfu/ml	RIGHT PAROTID FLOW RATE ml/min	LEFT PAROTID FLOW RATE ml/min	SERUM FERRITIN ng/ml	SERUM VITAMIN B12 pg/ml
056	600	N	1.4	1.5	92	162
063	320	N	1.8	1.6	-	1624
064	460	N	1.25	1.25	190	474
065	N	40	>0.7	>0.7	-	344
075	1100	N	-	-	21	223
079	160	N	1.6	1.4	28	460
080	760	N	>2.0	>2.0	6	480
081	100	40	0.7	0.9	251	475
083	330	N	-	-	37	369
085	120	N	1.2	0.9	120	336
086	N	680	0.5	0.5	261	267
087	N	20	<0.2	<0.2	273	374
089	1000	N	>2.0	>2.0	467	393
095	1000	N	1.2	0.8	62	371
097	N	260	-	-	26	>2000
098	20	N	1.0	1.0	-	237
100	N	74	0.5	2.0	80	210
101	20	N	-	-	14	327
102	80	N	0.4	0	37	297
107	3000	340	0.9	1.2	3	273

N = NONE ISOLATED

Table 6:3 Flow Rates and Haematological Indicies for those patients carrying Candida species and/or Coliforms.

PATIENT REFERENCE NUMBER	CANDIDAL SPECIES cfu/ml	COLIFORMS cfu/ml	RIGHT PAROTID FLOW RATE ml/min	LEFT PAROTID FLOW RATE ml/min	SERUM FERRITIN ng/ml	SERUM VITAMIN B12 pg/ml
108	N	40	0.8	0.8	234	391
110	N	20	1.3	1.2	163	503
111	200	N	1.0	1.0	38	294
112	60	N	0.9	1.0	85	569
114	N	60	-	-	48	288
116	N	40	-	-	108	432
117	130	N	1.6	1.1	-	455
122	60	N	0.8	1.0	92	365
124	200	180	3.0	2.5	52	136
131	140	N	0.5	0.3	10	71
134	N	60	-	-	101	273
138	140	N	>2.0	>2.0	39.5	594
139	N	20	1.5	1.8	93	290
140	N	740	1.1	1.1	157	409
141	140	60	0.5	0.9	118	270
148	180	160	1.4	1.4	144	287
149	150	40	0.8	0.8	130	>2000*
150	220	40	0.8	1.0	489	479

N = NONE ISOLATED

Table 6:3 Flow Rates and Haematological Indicies for those patients carrying Candida species and/or Coliforms.

CHAPTER 7

STIMULATED PAROTID SALIVARY FLOW RATES

7.1 RESULTS

Stimulated parotid flow rates were measured for 114 patients (appendix I). 19 (17%) were classified as having reduced salivary gland function in that they had stimulated parotid flow rates of 0.5 ml/min or less from both of their parotid glands.

Fifty four (36%) of the 150 patients felt on questioning that they had inadequate saliva and that their mouths were dry. Parotid flow rates were undertaken for 51 of these patients and 12 (24%) had flow rates of 0.5 ml/min or less. Therefore, 12 (63%) of the 19 patients who had parotid flow rates of 0.5 ml/min or less felt that their mouths were dry.

7.2 CONCLUSIONS

In this study there was no correlation between parotid salivary gland function and haematological deficiencies. Of the 19 patients with reduced stimulated parotid gland function only 1 patient (ref no 131) had haematologial deficiencies, this being the one patient who was deficient in both ferritin and vitamin B12. None of the 19 patients had abnormal plasma glucose estimations. No conclusions can be made on the relation between stimulated parotid salivary flow rates and vitamin status since only 8 of the 19 patients had their vitamin status estimated.

There was no relationship between reduced stimulated parotid salivary gland function and the oral

carriage of coliforms and candidal species (see section 6.2).

Eighteen of the 19 patients were female, 14 were postmenopausal, 3 had menopausal symptoms and 1 was premenopausal.

The relationship between medication at the time of presentation and parotid gland activity tend to agree with the conclusions of Sreebny and Schwartz (1986) on the influence of medication and dryness of the mouth which was that anti-cholinergic antidepressants were more likely to cause dryness than tranquillisers or sleeping tablets.

Of the 9 patients who were taking antidepressants at presentation stimulated parotid flow rates were measured for 6 of them. 4 showed reduced stimulated parotid flow rates. Parotid flow rates were measured for 22 of the 26 patients who were taking tranquillisers at presentation. only 4 had reduced activity and 1 of these patients was also taking antidepressants.

Stimulated parotid flow rates were measured for 38 of the 48 patients known to be taking sleeping tablets at presentation. Seven patients had reduced function. however 4 of these patients were taking antidepressants concurrently.

A relationship that was noteworthy was the one between stimulated parotid gland function and BMS type. BMS type had been determined for 18 of the 19 patients with reduced activity 15 (83%) were Type 2, 2(11%) were Type I and 1 (6%) was Type 3. BMS type was known for 93 of the 95 patients for whom stimulated parotid flow rates

were within the normal range 47 (50%) were Type 2, 34 (37%) were Type I and 12 (13%) were Type 3. This was a statistically significant difference (chi-squared test) $p < 0.05$ between the BMS types, it was more likely that Type 2 patients would have reduced salivary gland function compared to Type 1 and 3 patients.

It is possible that a tendency towards dryness of the oral cavity is going to be more pronounced during sleep and may account for the burning that is present on waking which allows the Type 2 classification to be applied. It was apparent also during the course of the study that a few patients had difficulty in differentiating between the burning symptoms and those of xerostomia.

CHAPTER 8

PROSTHODONTIC AND DENTAL INVESTIGATIONS - RESULTS

8.1 DENTAL STATUS

Ninety five patients (63%) were edentulous. Of the remaining 55 patients with some natural teeth, 34 wore dentures. Figure 8:1 illustrates the denture wearing status of the edentulous patients on presentation. Eighty seven patients wore complete upper and complete lower dentures, two wore a complete upper only, one a complete lower only for at least part of the day, whilst 5 patients wore no dentures at all. Figure 8:2 illustrates the denture wearing status of those patients with some remaining natural teeth. Sixteen patients wore a partial upper denture and 5 had partial upper and lower dentures. Six patients wore a complete upper denture opposing a lower dentition without a partial denture and one patient a complete lower denture opposing natural upper teeth only. Four patients had a complete upper and partial lower denture and 2 patients a partial upper opposing a complete lower denture.

8.2 EDENTULOUS PATIENTS

The complete data summarised in this section is presented in Appendix 1. Figure 8:3 illustrates the time lapse since total dental clearance for 85 of the 95 edentulous patients. Eighty seven per cent of patients had been edentulous for over 5 years. Figure 8:4 shows the age of the patients' dentures for 88 of the 95 edentulous patients. In 68% of patients their dentures were 5 years old or less. The patient whose dentures were

greater than 30 years old rarely wore them.

Table 8:1 illustrates the denture wearing pattern for the 84 of the 95 edentulous patients. For most patients (77%) the pattern for wearing their upper and their lower denture was consistent.

Removal of dentures partially alleviated symptoms in 54% of patients. totally alleviated symptoms in 3%. made symptoms worse for 5% of patients and made no difference to 38%.

The onset of burning symptoms was related to the provision of dentures or the modification to dentures in 19 patients (20%).

Relevant design faults were judged to be present in 57% of edentulous patients. Factors contributing to tissue overloading were found in 54% and tongue restriction noted in 19%.

8.3 PARTIALLY DENTATE PATIENTS - DENTURES

The raw data summarised in this section is presented in Appendix 1.

Figure 8:5 shows the age of the present dentures of the 23 of the 34 partially dentate patients. In 83% of cases. dentures were less than 5 years old.

The denture wearing pattern for each patient is presented in the appendix and is not summarised due to the variability of the type and combination of denture.

Removal of dentures partly alleviated symptoms in 48% of patients. totally alleviated symptoms in only one patient. made symptoms worse for 2 patients and had no effect on 41%.

The onset of symptoms was related to the provision of dentures or their modification in 9 patients (26%), and in one patient it was associated with endodontic treatment of an upper premolar.

Design faults were considered relevant overall in 26% of patients with a subdivision into, factors contributing to tissue overloading in 18% and to tongue restriction in 9%.

8.4 PARAFUNCTIONAL ACTIVITY

Table 8:2 lists the patients for whom parafunctional activity was noted. In total 31 patients (21%) had parafunctional habits including 2 dentate patients. Clenching and grinding was present in 21 patients of whom, 9 patients tongue thrusted and 1 both clenched and tongue thrusted.

The removal of dentures improved symptoms for 14 of the 29 dentures wearers (48%) suggesting a causal relationship.

8.5 CONCLUSIONS

Despite the finding that 50% of patients with burning mouth had denture faults as the single most tangible cause of their complaint (Main and Basker, 1983), no other report appears in the literature in which an attempt has been made to identify and quantify the relationship between denture design and BMS.

The population of the present study was remarkably similar to that of Main and Basker, (1983) with regard to dental status with 63% being edentulous compared with 62% reported by the Leeds group. Similarly overall 52% of

denture wearers, 57% of edentulous patients and 26% of partially dentate patients, were felt to have faults in denture design contributing to the patients' complaint in the present study compared to 50% reported by Main and Basker (1983) despite design assessment being largely subjective. It is, however encouraging that similar design objectives are aimed for nationally and that the other findings of the present study may also pertain in a British population.

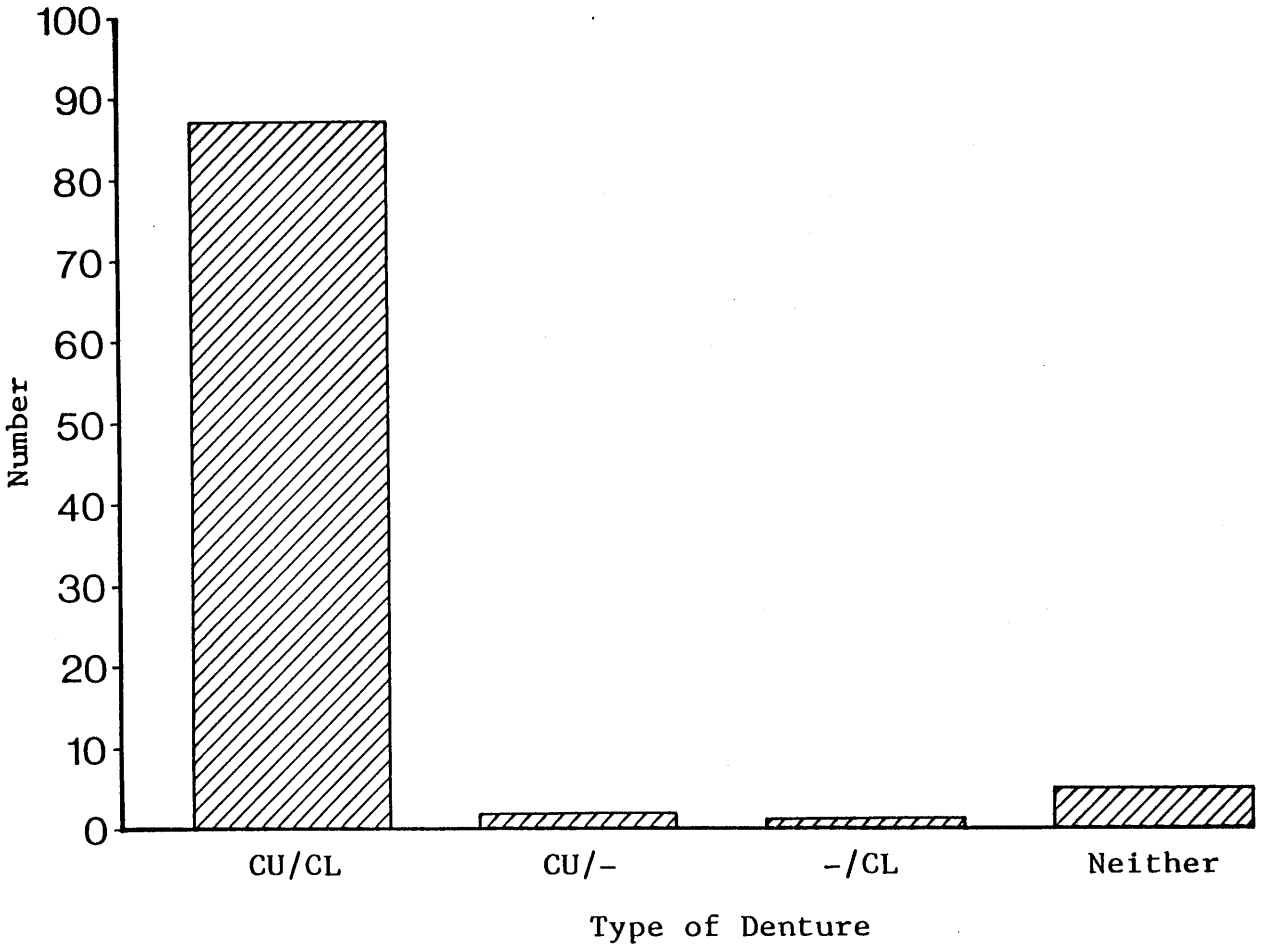


Figure 8: 1 Denture wearing status of the edentulous patients on presentation

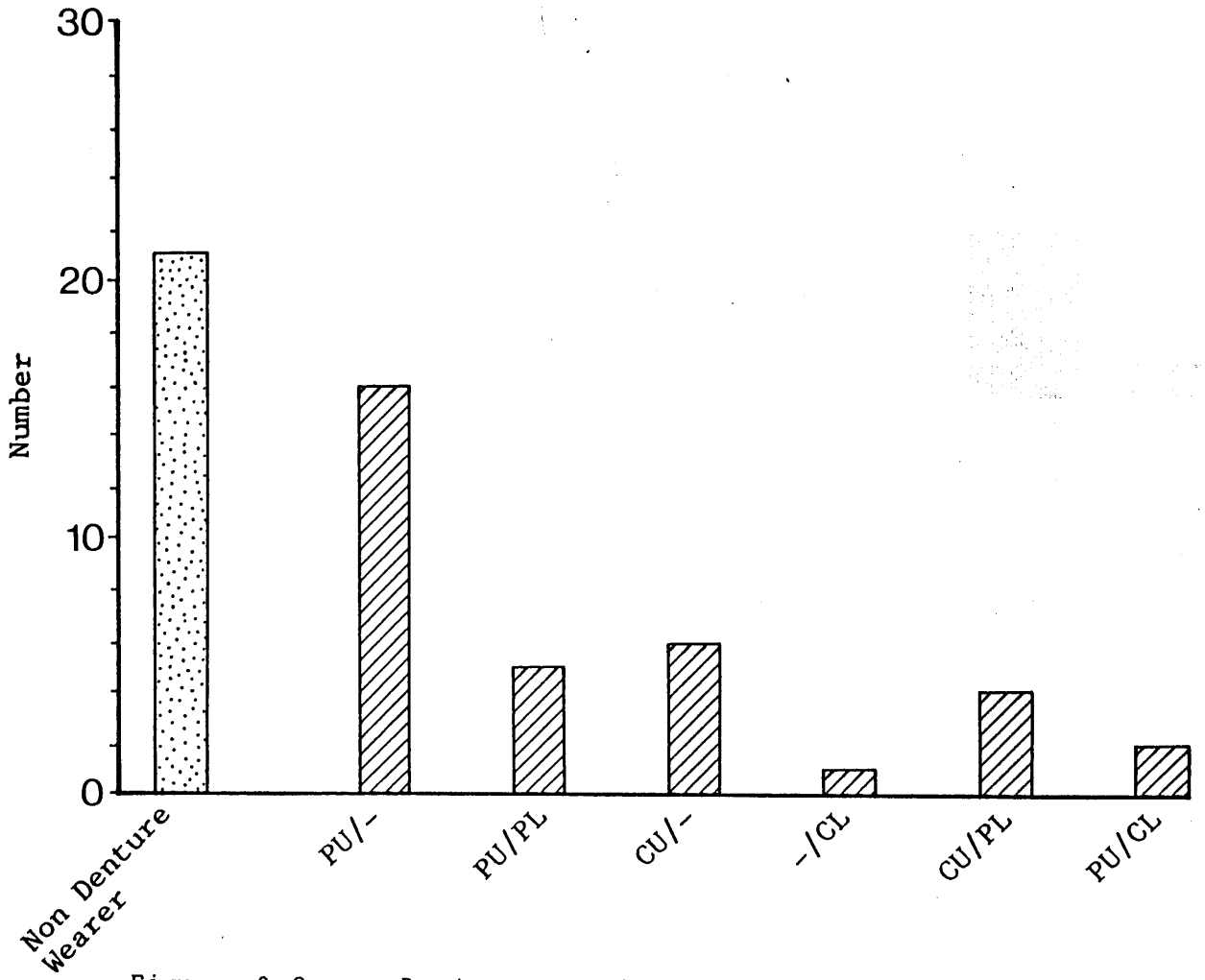


Figure 8: 2 Denture wearing status of the dentate patients on presentation

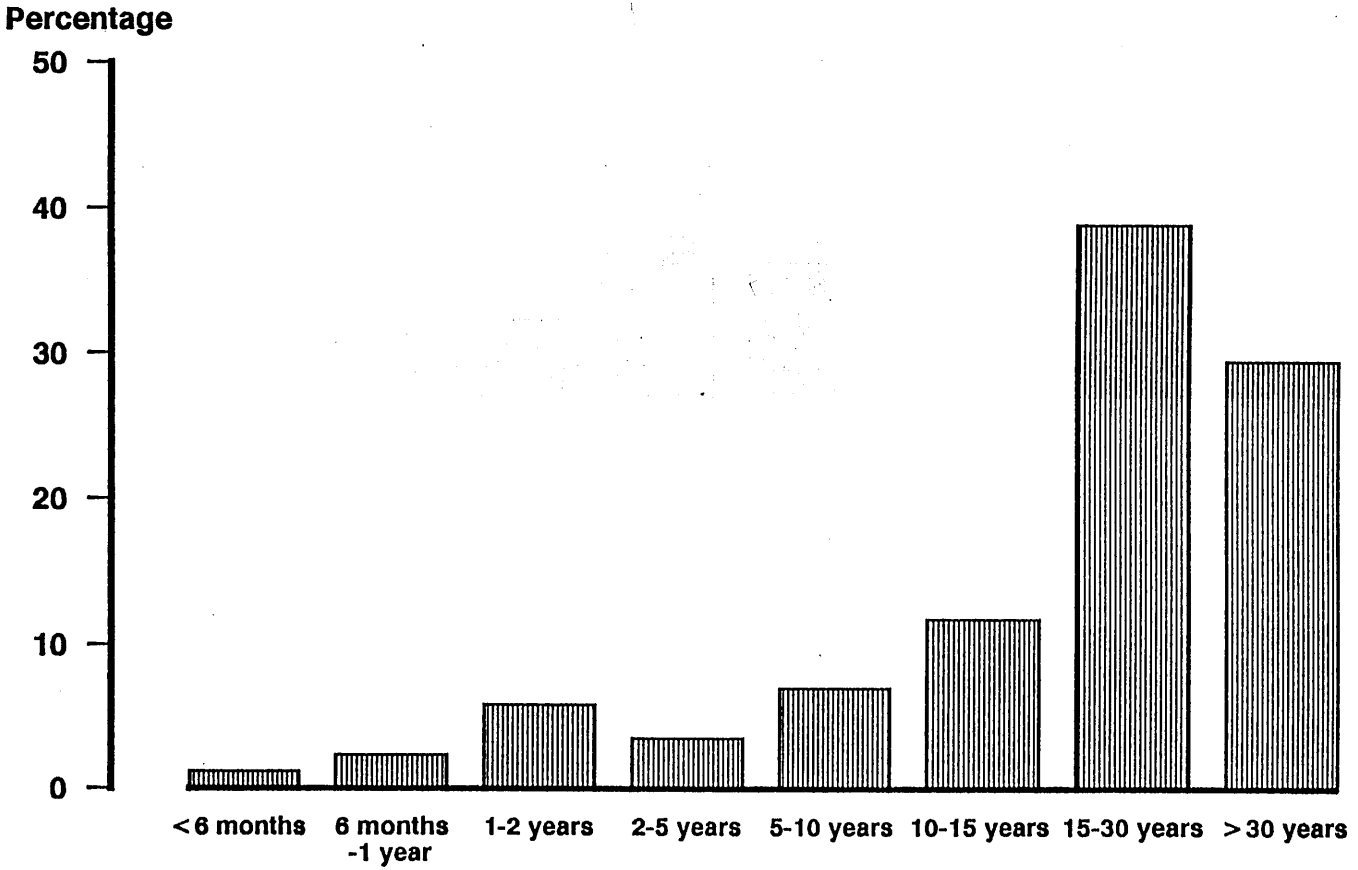


Figure 8: 3 Time since dental clearance (n = 85)

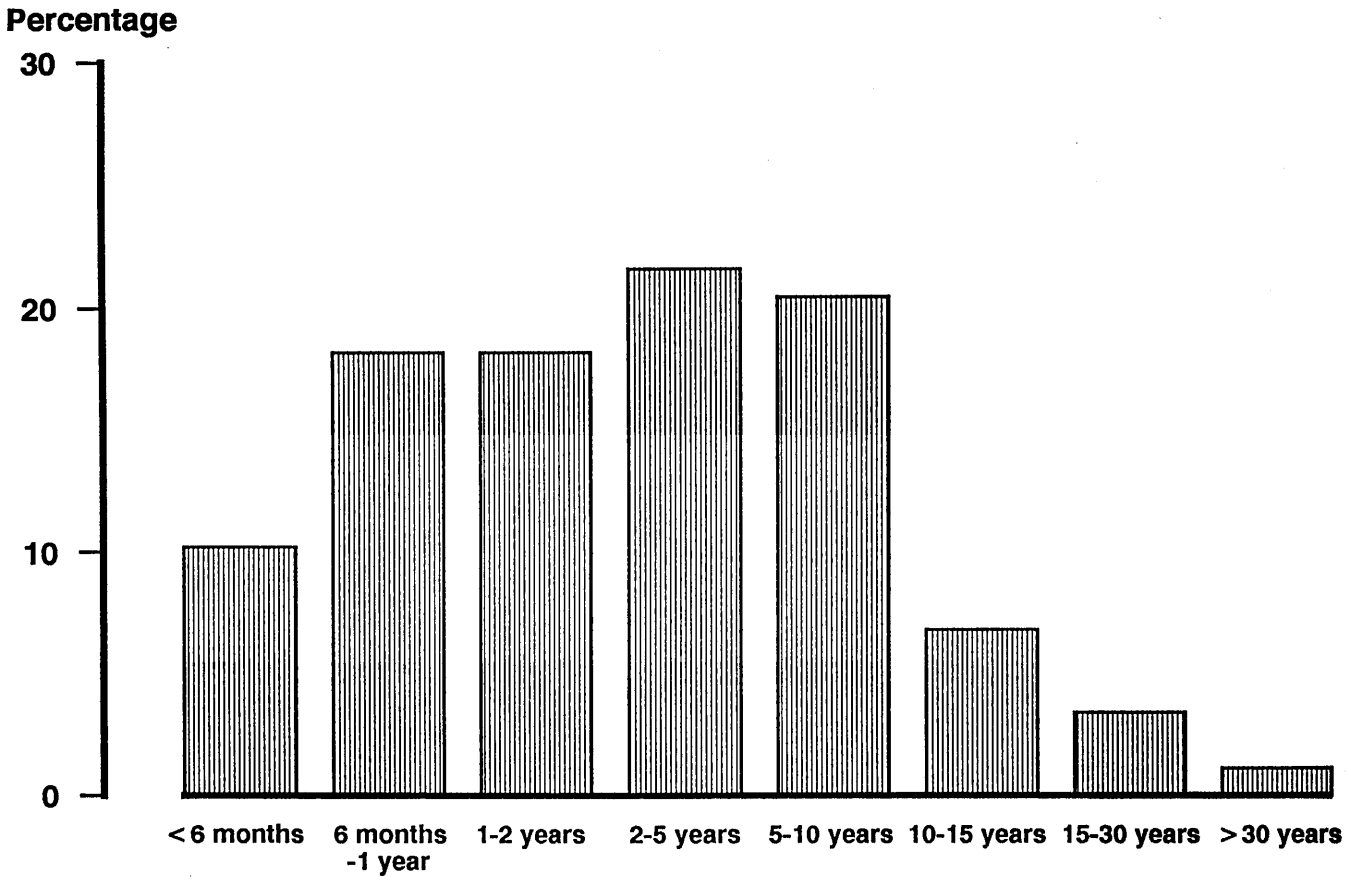


Figure 8: 4 Age of present dentures - edentulous patients (n = 88)

		UPPER DENTURE					
	DAY AND NIGHT	DAY ONLY	ONLY MEALS	EXCEPT MEALS	SOCIALLY	OCCASIONALLY	NEVER
DAY AND NIGHT	22.19	-	-	-	-	-	-
DAY ONLY	7.14	36.90	-	-	-	-	1.19
ONLY MEALS	2.38	-	1.19	-	-	-	-
EXCEPT MEALS	-	-	-	-	-	-	-
SOCIALLY	1.19	3.57	-	-	7.14	-	-
OCCASIONALLY	-	-	-	-	-	3.57	-
NEVER	1.19	-	-	-	-	1.19	5.95

LOWER DENTURE

TABLE 8:1 Denture wearing pattern of the edentulous patients expressed as a percentage (n = 84).

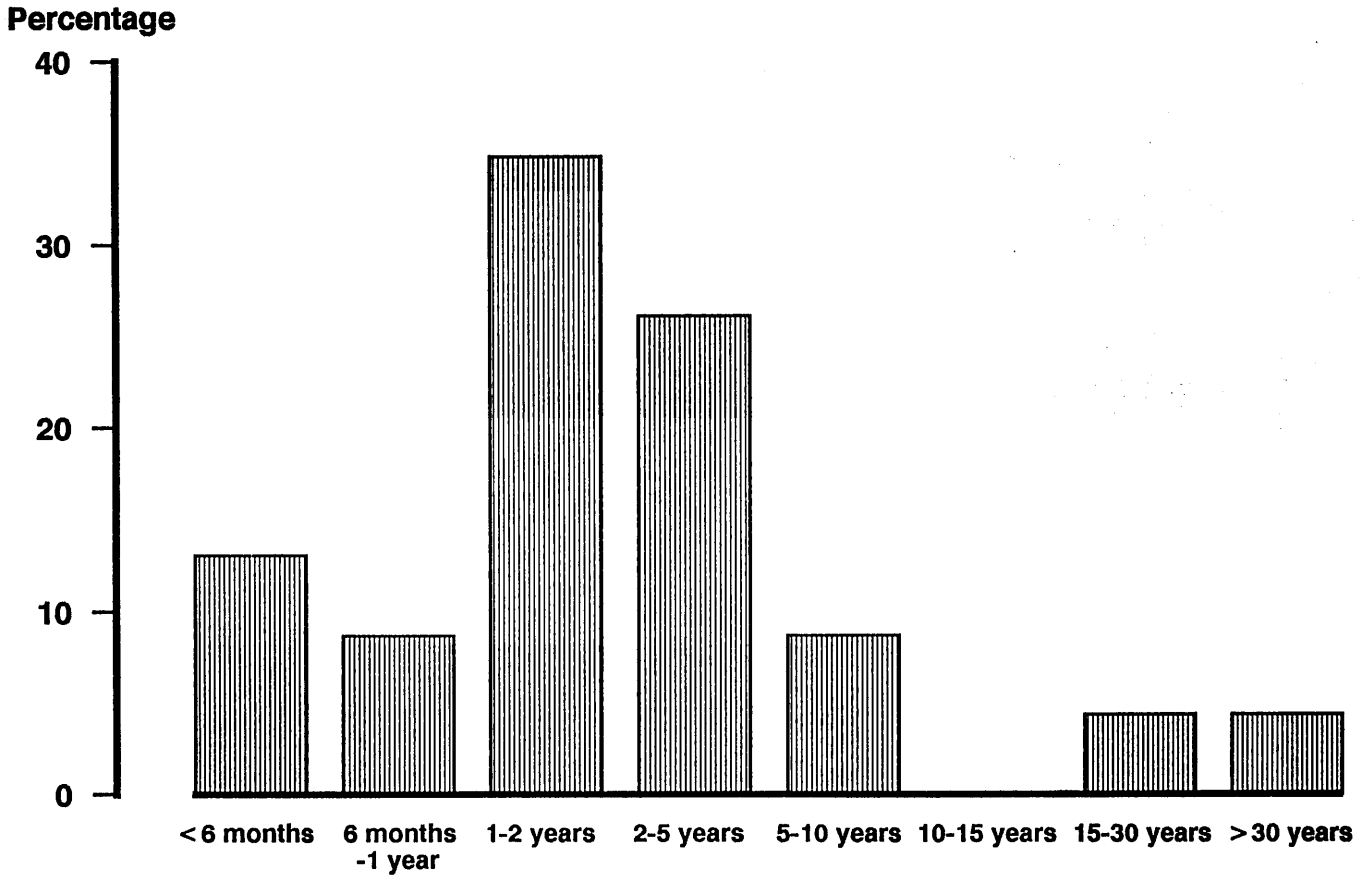


Figure 8:5 Age of present dentures - partially dentate patients (n = 23)

PATIENT REFERENCE NUMBER	DENTURES	PARAFUNCTIONAL HABIT	EFFECT OF DENTURE REMOVAL
001	PU	Tongue Thrusting	-
005	CU/CL	Clenching	Improvement
007	CU/CL	Clenching	Improvement
014	CU/CL	Clenching	Improvement
016	CU/CL	Clenching	No Change
017	CU/CL	Tongue Thrusting	No Change
023	CU/CL	Clenching	Improvement
028	CU/CL	Clenching	Improvement
037	-	Tongue Thrusting	-
048	CU/CL	Clenching	No Change
050	PU/-	Tongue Thrusting	Worse
055	CU/CL	Clenching	No Change
069	CU/CL	Clenching	No Change
071	CU/CL	Clenching and Tongue Thrusting	U - Improvement L - No Change
074	CU/CL	Clenching	No Change
076	CU/PL	Clenching	No Change
077	CU/PL	Clenching	Improvement
078	CU/CL	Tongue Thrusting	No Change
102	PU/PL	Clenching	No Change
106	CU/CL	Clenching	Improvement
114	CU/CL	Clenching	Improvement
117	-/CL	Clenching	-
126	CU/CL	Clenching	No Change
128	CU/CL	Clenching	Worse
132	CU/CL	Tongue Thrusting	U - No Change L - Improvement
134	CU/CL	Tongue Thrusting	Improvement
137	CU/CL	Clenching	No Change
138	-	Tongue Thrusting	-
140	CU/CL	Tongue Thrusting	Improvement
141	CU/CL	Clenching	Improvement
143	CU/-	Clenching	Improvement

Table 8:2 Effect of denture removal in patients with a parafunctional activity.

CHAPTER 9

PSYCHOLOGICAL INVESTIGATIONS - RESULTS

9.1 CANCERPHOBIA

Twenty eight patients (18.6%) were concerned that the burning sensation in their mouth represented cancer or the future development of cancer (Table 9:1).

9.2 CATTELL'S 16PF FORM C QUESTIONNAIRE

This questionnaire was presented to every third patient in the study group, three patients failed to complete the form. The mean response profile for the remaining 47 patients (40 female and 7 male) was determined (Figure 9:1).

This mean profile showed significant deviations on eight factors when compared to that of the general adult population of the same age range, using the chi-squared test.

Certain clusters of factors, with particular degrees of deviation from the normal range have been shown to correlate with instability, anxiety and depression (Cattell et al 1970: Krug, Scheier and Cattell, 1976). When profiles for each of the 47 individuals were studied, 28 patients (60%) revealed signs of these psychological abnormalities (Table 9:2), these are reflected in the total sample profile (Figure 9:1).

The mean profile for those patients without instability, anxiety or depression (n=19) differs significantly on four factors from those patients (n=28) with one or more of these psychological dysfunctions (Figure 9:2). These psychologically abnormal patients

were significantly less stable (C-), more apprehensive (O+), less socially-precise (i.e. more casual) (Q3-) and more tense (Q4+), using one tailed t tests.

The degree of introversion and extroversion has been shown in other studies to correlate with both positive and negative areas of behaviour (Eysenck, 1971; Reeve and Watson, 1985). In this sample 23 (49%) emerged as introverts, 11 (23%) as extroverts and 13 (28%) as ambiverts (Table 9:2).

The significance of these findings and the relevance to outcome of treatment is described fully in Chapter 11.8.

There was no correlation between the psychological status of the patients with any of the following, the severity of symptoms at presentation (section 3.7), their age or sex, whether dentures had been provided or not (thirty three (70%) of the group sampled had either complete or partial dentures), or their indicated satisfaction with their social and domestic circumstances (section 3.9). Since the proportion dissatisfied in the sample psychologically assessed was 27.05% compared favourably with the sample as a whole (25.87%) this is likely to be true of BMS patients in general. In addition there was no correlation between the psychological findings and an expressed fear of cancer (section 9.1).

9.3 CONCLUSIONS

It is possible that cancerphobia can develop in this group of patients as a result of a number of

factors. These include the patient's own irrational fear particularly if they are not reassured following attendance at clinics of the benign nature of their condition and also family history of cancer.

The study using the Cattell's 16PF Form C Questionnaire showed that there were psychological factors operating in more than half of the patients studied. When symptom pattern (section 3.6) was compared to psychological abnormality there was a higher incidence in Type 2 compared with Type I (Table 9:3). Since the distribution of BMS type in the 47 patients tested with the questionnaire was TYPE 1, 34%, TYPE 2 53%, and TYPE 3 13%, and since this compares favourably with the distribution in the 150 patients of the present study, (Section 3.6) it is likely that the group sampled with the questionnaire is representative of the study group as a whole.

Thirty six per cent of the sample were severely psychologically disabled in that they demonstrated instability, anxiety and depression.

There was no correlation between either altered sleep patterns that have been described as relating to psychological dysfunction (Walton, 1987) (section 3.10) or with the patients' subjective assessment of social and domestic circumstances and the psychological assessment data gained from the Cattell's 16PF Form C. Therefore it is important that a valid objective psychological assessment, using a standard psychological proforma, is undertaken early in management so that those patients

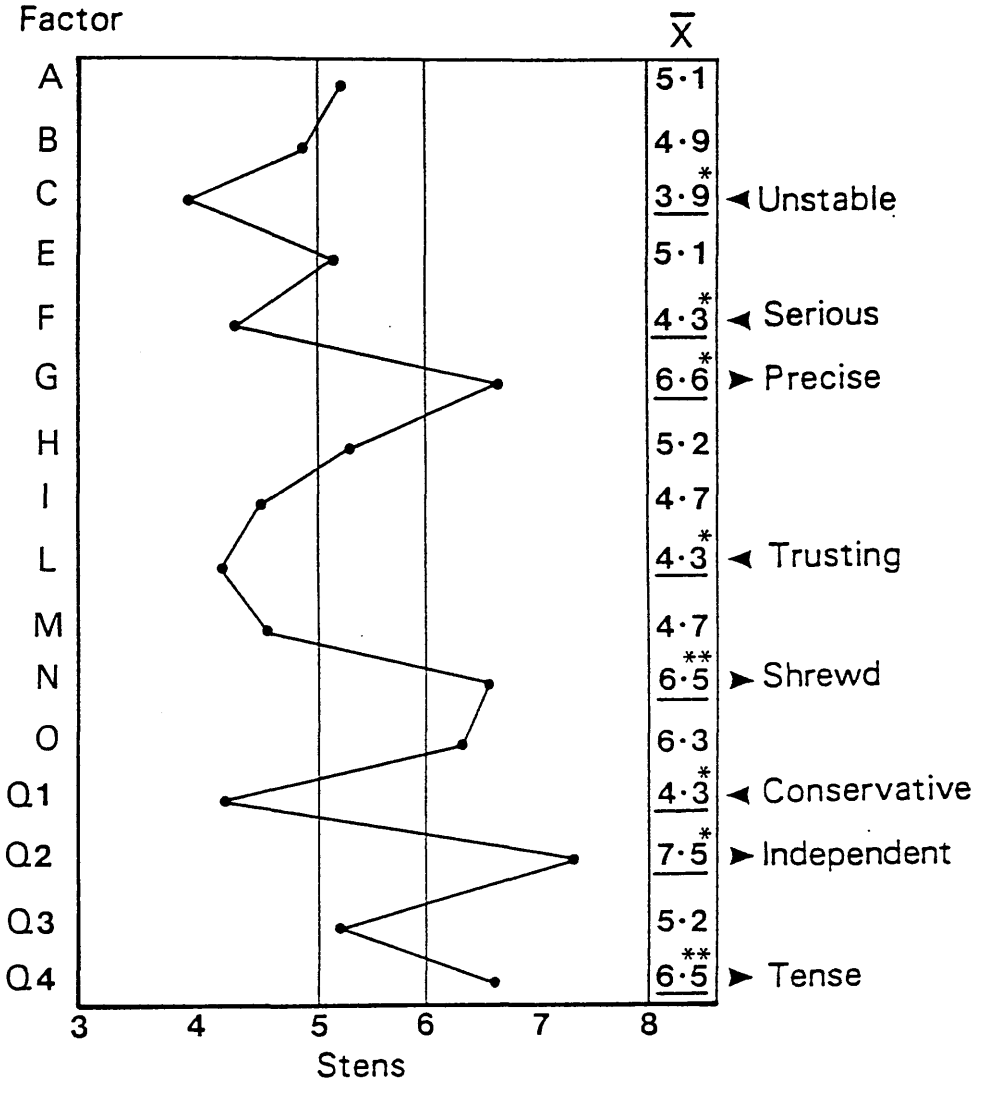
requiring positive reassurance and support are correctly identified. Subsequently the management of each individual patient can be shaped to fit their psychological as well as their medical and dental needs.

The relationship of the psychological assessment and outcome of treatment is discussed fully in section 11.9.

In the absence of a psychological assessment for an individual patient the mean profile (Figure 9:1) should be borne in mind. this suggests that BMS patients are not decisive, yet prefer their own decisions, they are averse to change but have an eye for what may pay-off. Their propriety and sense of guilt, linked to timidity, make them reluctant to complain but amenable to firm advice and professional reassurance.

PATIENT REFERENCE NUMBER
010
011
013
014
017
023
033
034
038
046
064
065
067
071
082
085
090
105
116
120
128
129
136
138
140
142
145
149

Table 9:1 Patients who expressed a marked fear of cancer in relation to their BMS Symptoms.



——— Differs significantly from general adult population

* $p<\cdot001$

** $p<\cdot01$

Figure 9:1 Mean profile recorded by Cattell's 16 PFQ Form C for BMS patients (n = 47) compared with an age matched general population.

PATIENT REFERENCE NUMBER	SEX	AGE	BMS TYPE	INSTABILITY	ANXIETY	DEPRESSION	INTROVERSION /EXTROVERSION
002	F	36	1	*	*	*	I
004	F	58	2	*	*	*	A
008	F	63	1	*	*	*	I
014	F	78	1	*	*	*	I
016	F	65	1				A
025	F	52	2		*		E
026	F	59	2	*	*	*	E
031	F	74	3	*			I
034	F	63	2	*		*	I
035	F	65	1		*		E
037	F	62	2			*	I
038	M	54	1				I
049	F	51	2	*		*	I
055	F	66	2	*		*	I
056	F	49	2		*		E
057	F	55	1				A
060	F	43	3				I
061	F	57	3				I
076	F	76	1				I
080	F	40	1				A

* - Presence of dysfunction
E - Extroversion
I - Introversion
A - Ambiversion

Table 9:2 Presence of instability anxiety and depression.

PATIENT REFERENCE NUMBER	SEX	AGE	BMS TYPE	INSTABILITY	ANXIETY	DEPRESSION	INTROVERSION /EXTROVERSION
081	M	71	2				A
082	F	69	1				I
086	M	63	2	*	*	*	I
089	F	62	2	*		*	I
092	F	64	2	*	*	*	I
094	F	47	2		*	*	A
096	F	39	1				E
098	F	53	2	*	*	*	A
099	F	71	2				A
100	F	64	1				A
101	F	56	2	*	*	*	I
102	F	50	2	*	*	*	I
104	F	76	2	*	*	*	I
107	F	43	3				I
108	M	63	2				E
111	F	54	1	*	*	*	E
112	M	47	2				E
113	M	58	2	*		*	I
114	F	53	1				E
118	F	48	2	*	*	*	E

* - Presence of dysfunction
E - Extroversion
I - Introversion
A - Ambiversion

Table 9:2 Presence of instability anxiety and depression.

PATIENT REFERENCE NUMBER	SEX	AGE	BMS TYPE	INSTABILITY	ANXIETY	DEPRESSION	INTROVERSION /EXTROVERSION
124	M	66	3				A
125	F	65	2	*	*	*	I
129	F	64	1	*	*	*	I
130	F	63	1				E
132	F	64	2	*	*	*	A
136	F	51	2	*	*	*	A
137	F	69	3				A

* - Presence of dysfunction
E - Extroversion
I - Introversion
A - Ambiversion

Table 9:2 Presence of instability anxiety and depression.

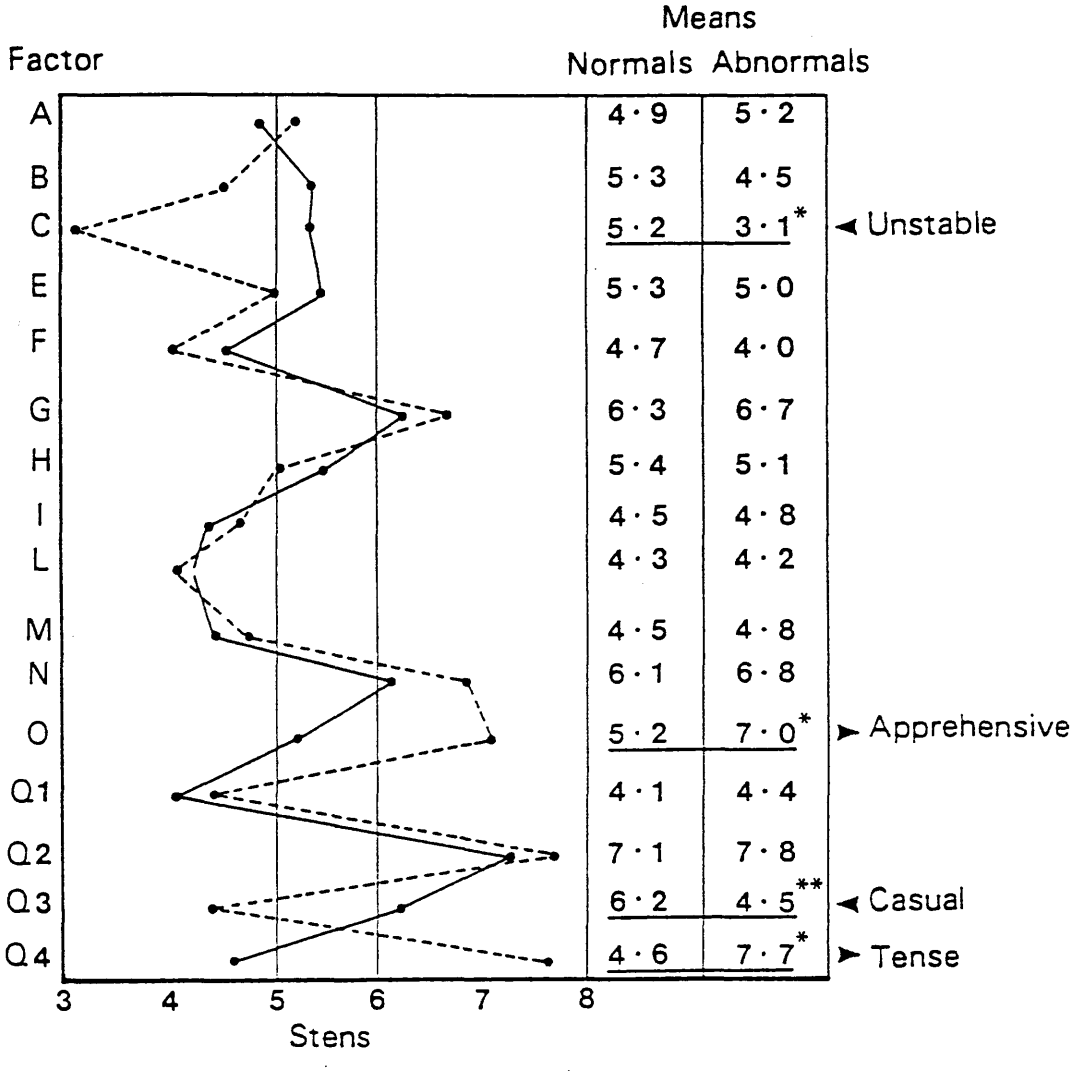


Figure 9: 2 Mean profile recorded by Cattell's 16 PFQ Form C for BMS patients with (n = 28) and without (n = 19) a psychological dysfunction

	BMS TYPE		
PSYCHOLOGICAL ABNORMALITY	1	2	3
INSTABILITY	31	68	16
ANXIETY	37	60	0
DEPRESSION	31	76	0
OVERALL	37	84	16

Table 9:3 Percentage frequency of psychological dysfunction present in patients with Type 1, 2 or 3 BMS.

CHAPTER 10

INVESTIGATION OF ALLERGY - RESULTS

Fourteen patients were patch tested. Table 10:1 indicates the substances to which patients were found to be allergic. In six of these patients the allergens identified were thought to be relevent to the patients' complaint and in one additional patient the significance was doubtful.

10:1 ALLERGY TO FOODSTUFFS

Three patients (reference numbers 029, 037, and 101) were found to be allergic to the food related products indicated in Table 10:1, the significance of the patch testing for patient 029 who reacted to benzoic acid was doubtful.

10:2 ALLERGY TO DENTAL MATERIALS

Five patients were found to be allergic to dental materials. Patient 058 was shown to be allergic to dental filling materials however since she was edentulous this was thought irrelevant. Patient 004 was allergic to nickel and was wearing partial upper and partial lower dentures with cobalt chromium alloy bases. Her BMS symptoms coincided with provision of these dentures and were eased by denture removal and there were no obvious design faults. Patients 009, 044 and 127 were shown to have positive skin reactions to components of the acrylates group. Patient 009 related onset of symptoms to the provision of new complete dentures constructed in polymethylmethacrylate (PMMA). Burning symptoms were

eased by denture removal and there was no obvious denture fault. Patient 044 wore a partial upper denture constructed in PMMA and although the onset of symptoms was related to provision of this denture and symptoms were totally alleviated following denture removal there was no obvious design fault. Patient 127 was wearing complete upper and lower denture again symptoms developed at the time of provision of these dentures and were improved by denture removal in this case however there were relevant design faults present.

PATIENT REFERENCE NUMBER	POSITIVE ALLERGENS	DIAGNOSIS	RELEVANCE TO BMS
004	NICKEL	ACD - NICKEL	RELEVANT
006	NEOMYCIN	ACD - NEOMYCIN	NOT RELEVANT
009	ACRYLATES URETHANE DIMETHACRYLATES 2%	ACD - ACRYLATES	RELEVANT
029	BENZOIC ACID	UNCERTAIN	UNCERTAIN
037	PROPYLENE GLYCOL SORBIC ACID	CU - FOOD ADDITIVES	RELEVANT
044	NICKEL. ACRYLATES. ACRYLIC MONOMER	ACD- NICKEL. ACRYLATES. ACRYLIC MONOMER	RELEVANT
058	RUBBERS. MERCURIALS. THIOSALICYLIC ACID	ACD - DENTAL FILLING MATERIALS	NOT RELEVANT
065	RUBBERS	ACD - RUBBERS	NOT RELEVANT
090	NIL	NIL	NOT RELEVANT
100	COLOPHONY	ACD - COLOPHONY	NOT RELEVANT
101	CINNAMALDEHYDE	CU CINNAMALDEHYDE	RELEVANT
114	NIL	NIL	NOT RELEVANT
127	ACRYLATES - ACRYLIC MONOMER	ACD ACRYLATES	RELEVANT
141	NIL	NIL	NOT RELEVANT

ACD = Allergic Contact Dermatitis (Type IV delayed hypersensitivity)

CU = Contact urticaria

Table 10:1 Patients who were patch tested, allergens and relevance to BMS.

CHAPTER 11

RESULTS OF TREATMENT

11.1 INTRODUCTION

With the use of the linear analogue scale outlined in section 2.3 it was possible to quantify patients' pre- and post-treatment burning severity and thus whether treatment had any effect on the patients' symptoms. Those patients who scored 0 following treatment were regarded as cured (symptomfree). With the exception of one patient (number 120) all patients who scored 1, 2 or 3 post-treatment said that they were improved and could now live with their symptoms. Allowing for individual variation in description of pain severity and in view of the strict criteria adopted in this study (being the first to quantify pain in BMS) then without the use of an analogue scale these patients would otherwise also have been considered as cured since they neither sought nor required further treatment. On this basis the post-treatment score of 0-3 could be interpreted as indicating a cure. Table 11.1 indicates the pre-treatment and post-treatment scores for 142 patients as to whether they were cured, improved, unchanged or worse. Although clearly scores were evaluated in a longitudinal manner, it is important to emphasise that these post-treatment scores represent the scores given by patients at least 18 months after completion of active treatment. There is a placebo element in any study of orofacial pain and therefore this minimum period of 18 months was necessary to both

eliminate any placebo effect and provide a sustained answer to the efficacy of the treatment provided.

Post-treatment scores are demonstrated in Figure 11:1 and combining these with Figure 3:8 for the scores on initial presentation Figure 11:2 shows the reduction in severity scores reported by the 142 patients as a group. On an individual basis 51 patients (36%) were symptomfree, 34 (24%) had minimal symptoms, 16 patients (11%) were improved, no change was reported by 37 patients (26%) and 4 patients (3%) were worse. For ease of comparison with the various treatment regimes described in this Chapter patients are categorised as in Table 11:1 ie cured, improved, unchanged or worse.

Table 11:2 compares the cure rate for males and females with the overall cure rate irrespective of gender. There was no difference in treatment outcome for males and females, (chi - squared test).

The effect of age on outcome of treatment is shown in Table 11:3. Again there was no significant relationship between age and treatment success (chi - squared test). Similarly for female patients there was no significant association between menopausal status and outcome of treatment (chi - squared test) (Table 11:4).

Analysis of outcome of treatment and oral site affected by the burning sensation is complex due to the many combinations of single or multiple oral sites that are possible. However analysis of response to treatment when one or more of the major sites was affected is illustrated in Table 11:5. Once more there was no

significant difference in outcome of treatment irrespective of site affected (chi-squared test).

Table 11:6 shows outcome of treatment based on categorising patients into Type 1, 2 or 3. Although numbers are small in Type 3, the treatment regime developed seem most satisfactory since these patients generally have a satisfactory outcome. However Type 1 patients are not significantly (chi-squared test) easier to treat successfully compared to Type 2, but the trend would support the hypothesis that BMS patients are not a homogeneous group and lends credence to the proposed sub-division (see also section 11.9.2).

Rather surprisingly there was no significant difference between cure rate and patients' quantification of their social/domestic circumstances (chi-squared test). Sixty four per cent of those patients reporting adverse circumstances were cured (Table 11:7) compared with 58% of those who did not. This finding is difficult to interpret but may indicate that the setting of a Burning Mouth clinic has a psychological effect on patients' wellbeing by providing a forum for open discussion of adverse home circumstances, an avenue perhaps not readily available to such patients.

11.2 HAEMATOLOGICAL DEFICIENCY

No patient declined haematological investigation or had a phobia to needles.

The one patient found to be anaemic (ref no 032) was referred to a consultant physician with an interest in haematology but following extensive investigation no

cause for the anaemia could be found. Nevertheless following iron replacement therapy she was symptom free, her mean corpusular haemoglobin rose to 14.4 g/dL (previously 10.2g/dL) and her blood film was reported as normal and serum ferritin had risen to 35 ng/ml (previously 3 ng/ml). This patient still had regular but somewhat heavy periods and on balance it was thought that this factor was probably contributing to her anaemia. She remains under regular 6 monthly reviews to ensure that she does not become anaemic again.

For none of the patients for whom an abnormal MCV or film was reported was there found to be a link between treatment directed towards the abnormality and alleviation of symptoms.

In addition to the one patient who was suffering from iron deficiency anaemia, 7 patients were sideropenic. Following referral to a consultant physician for investigations these patients were given ferrous sulphate 200 mg three times daily for at least 3 months to restore serum ferritin levels to normal, (confirmed by repeated estimation). Of the initial patients, three (ref numbers 080, 107 and 113) were rendered asymptomatic. One patient (ref number 131), who was also vitamin B12 deficient, reported partial relief in symptoms when these haematological indices were restored to normal. Improvement was judged, as in all other cases, by a reduction of the patients' reported score on the linear analogue scale.

Of the 12 patients with low serum vitamin B12

estimations. 3 patients (ref numbers 057, 082 and 149), were symptom-free following referral to a consultant physician who instituted treatment with 6 weekly intramuscular injections of 1,000 μg cyanocobalamin. A further 5 patients similarly referred (ref numbers 020, 124, 131, 133 and 146) noted an improvement in symptoms.

Overall, haematological deficiencies of ferritin or vitamin B12 were found in only 19 patients (13.2% of those tested), and 7 of these patients (37%) had total alleviation of symptoms whereas 6 (31%) had partial alleviation of symptoms following treatment for these deficiencies. In this study therefore 68% of patients with a haematological deficiency reported a sustained improvement in symptoms following correction of the deficiency.

11.3 DIABETES MELLITUS

The findings of the present study were consistent with previous reports that undiagnosed maturity onset diabetes mellitus was a relatively infrequent finding in BMS. However in terms of management early detection of underlying diabetes in this group is important as diabetes mellitus has a high morbidity and mortality. Following referral to a diabetic clinic each of the 4 patients in this category was symptom-free once adequate glycaemic control was instituted, although one patient initially had reported partial resolution of symptoms with empirical vitamin B1 and B6 supplements, and another with topical antifungal therapy to eliminate oral candidiasis. For the three known maturity onset diabetic patients who had poor

glycaemic control, no improvement in symptoms was achieved by treatment including provision of new complete dentures and avoidance of clenching for patient reference number 028, supplements of vitamins B1 and B6 however cured symptoms for one patient (ref number 140) and no treatment was successful for one patient (ref number 142).

11.4 VITAMIN DEFICIENCY

Of the 68 patients tested 38% (26) were found to be deficient in vitamin B1, B2 or B6 or combinations of these vitamins. Of these 26 patients, 4 patients were cured and 5 patients were partly cured with replacement by the appropriate vitamin (Table 11:8). Vitamin replacement therapy consisted of vitamin B1 300 mg, vitamin B2 20 mg or vitamin B6 150 mg daily in divided doses, and this treatment was responsible for improving symptoms in 35% of patients shown to be deficient.

The remaining 82 patients for whom assays were not undertaken had vitamin B1 and B6 therapy as described above. Improvement in symptoms was reported by 17 patients (21%) (Table 11:9). Only 2 (2.4%) of these patients were totally cured with vitamin B1 and B6 therapy alone, compared to 5.8% of those cured in the 68 patients known by testing to be vitamin B complex deficient. Fourteen patients not tested for deficiency who were eventually cured in the study had partial resolution of symptoms with Vitamin B1 and B6 therapy compared to only 5 patients in the group known to be deficient. Since none of the 42 patients demonstrated to have had no deficiency were given vitamin B1 and B6 it is possible that some of

these patients also would have reported some improvement as a temporary placebo effect. It is likely that this is the reason for the improvement in the 17 patients who were not tested and since they did not report a total cure on vitamin therapy, further treatment regimes were employed. Although these 17 patients ultimately had sustained relief from symptoms it is difficult to assess what proportion of this was due to vitamin B complex therapy alone.

11.5 ORAL INFECTIONS

Of the 41 patients in whom candidal species were isolated, 7 (17%) reported improvement in burning symptoms following denture hygiene and antifungal treatment (patient's reference numbers 033, 087, 089, 112, 124, 138, 148). All patients who had acrylic dentures were instructed to soak their dentures overnight in a hypochlorite solution and in addition all received topical antifungal treatment with Amphotericin B lozenges four times a day for 28 days. Following therapy, repeat oral rinse cultures confirmed the absence of candidal species. Despite this, for only 1 patient could total alleviation of BMS be attributed to antifungal therapy alone. (Reference number 112.)

11.6 REDUCED SALIVARY FUNCTION

All patients in whom stimulated parotid flow rates were reduced (<0.5 ml/min) and also all patients who on questioning felt they had a dry mouth were prescribed artifical saliva substitute (Orthana spray).

Of the 12 patients who felt they had dry mouths (and also had low measured parotid flow rates) only 2

(16%) were helped by this treatment. Similarly of the remaining 36 patients who felt they had dry mouths but had normal stimulated parotid flow rates 7 patients (19%) noted an improvement in symptoms following treatment with Orthana spray (Table 11:10). Two of the remaining seven patients who had a quantitative reduction in salivary gland function, but no complaint of dry mouth, noted an improvement with Orthana spray (29%) (Table 11:11).

It would appear that artifical saliva replacement helped approximately 20% of patients who had either a complaint of xerostomia or who had reduced parotid salivary gland function. These findings would tend to support the view of Daniels in Talal, Moutsopoulos and Kassan (1987) that standardised parotid flow rate assessment provides a useful quantitative assessment of an individuals salivary gland function, but is too variable and non-specific to serve as a diagnostic criterion.

Only 2 patients in the study, neither of whom had reduced parotid flow rates, were cured with artifical saliva replacement therapy alone (reference numbers 062 and 079). (see Table 11:10) and only a further 2 patients noted improvement without any other effective treatment regime.

11.7 INADEQUATE DENTURES

11.7.1 Edentulous Patients

Replacement dentures were provided for 47 of the 95 edentulous patients (49%) (Appendix 1). Dentures were constructed when either a relevant design fault was present and denture removal reduced the severity of the

symptoms or if the onset of BMS symptoms was related to provision of dentures (or their modification). Ten other patients in these categories did not have dentures provided, 6 because other treatment regimes outlined in this Chapter had resulted in cures, 2 (ref numbers 008 and 043) because they declined dentures, one (ref number 093) who was psychologically unfit and one (ref number 147) who had markedly improved on other treatment regimes. Dentures were constructed to recognised standards (British Society for the study of Prosthetic Dentistry, 1981) particular care was taken to ensure maximum load distribution by utilising all of the available denture bearing mucosa, and avoidance of an excessive occlusal face height. Also correct bucco-lingual tooth position and correct positioning of the lower occlusal plane to enhance stability and avoid tongue restriction was employed. These dentures were provided either by the author or his staff and the author personally ensured standards had been met.

Of the 47 patients who had replacement dentures constructed 23 (49%) reported improvement in BMS symptoms. However for 3 patients this was not sustained. Overall therefore 21% of edentulous patients had improvement in their symptoms by modification to denture design. Six of the 20 patients (30%) who were improved by new denture construction, were cured by dentures alone (patients reference numbers, 035, 036, 063, 073, 106, 130).

11.7.2 Denture wearing partially dentate patients

As for edentulous patients new dentures were

generally constructed only when indicated, and treatment was aimed at avoidance of tongue restriction and overloading of soft tissues.

Of the 34 patients in this group who wore or required dentures 16 patients (47%) had new dentures constructed (Appendix 1). Of these 16 patients 10 reported sustained improvement in BMS symptoms (62%). Therefore 29% of patients with some natural teeth who wore or required dentures were improved by the provision of new dentures. Four of the 10 patients improved by new denture construction were improved by new dentures alone, 3 being cured. (Patients reference numbers, 110, 115, 118).

11.8 PARAFUNCTIONAL ACTIVITY

When parafunctional activity was noted, treatment was aimed at stopping or reducing this activity by the following methods. Reinforcement of the need to consciously avoid the habit, modification of dentures particularly reduction of excessive occlusal face height, better load distribution by maximising base extension and maximising tongue space, muscle relaxation by providing dothiepin (Prothiaden, Boots Drug Company Ltd) 50 mg-150 mg nocte or by hypnotherapy.

Of the 31 patients with parafunctional activity 4 patients had improvement in symptoms and a further 16 patients were cured by the management regime to be described in Chapter 12. However reduction in parafunctional activity alone by the methods listed in this section resulted in a cure for 10 patients - 32% of the 31 patients with parafunctional activity.

11.9 PSYCHOLOGICAL DISTURBANCES

11.9.1 Cancerphobia

For the twenty percent or so of patients who were cancerphobic the presence of unremitting symptoms frequently lead them to think that they already had cancer or that they were about to develop it. Repeated reassurance and direct questioning of their fears appeared important in helping patients overcome this concern. Dothiepin (50-150 mg nocte) was useful in alleviating fears for some patients.

Table 11.12 shows treatment response for those patients who were cancerphobic. Twenty one patients (64%) were cured and a further 2 (10%) were improved by the treatment regime described in the next Chapter. Of the 21 patients 18 responded favourably to reassurance and or Dothiepin therapy. Of these 18. 8 (44%) responded with the involvement of no other treatment regime. Dothiepin helped 12 (52%) of the 23 patients who were either cured or improved.

11.9.2 Psychological profile

Twenty-eight patients (60%) of those sampled with the Cattell's 16 PF Form C Questionnaire were cured. Table 11:13 shows their psychological status, initial severity score, post-treatment score, and the relevant treatment methods responsible for the cure. Psychological status was determined by the absence of instability, anxiety and depression (normal), the presence of one or two of these dysfunctions (abnormal), and the presence of all three as (severely abnormal). (see Table 9:1). On the

basis of this division between psychologically normal and psychologically disabled the cure rate for both groups was 68% and 54% respectively. Whilst not statistically significant. there was a clear difference between these two groups yet more than half of the psychologically disabled obtained relief from BMS symptoms with treatment (Table 11.13). More detailed analysis showed that 17 of the 28 patients with abnormal profiles were severely abnormal. Table 11:14 shows the outcome of treatment. It was apparent. therefore. that those patients with a severe psychological dysfunction were more resistant to successful treatment. Table 11:15 shows the degree of success in treating patients with BMS who demonstrated instability, anxiety and depression. Anxiety being the most recalcitrant psychological obstacle to cure.

The mean profiles of the cured patients (n = 28) and not cured (n = 19) were compared. The mean scores differed significantly on four factors using a one-tailed t-test. The not cured patients were more unstable (C -). more intelligent (B+). more apprehensive (O+) and more tense (Q4+). the levels of significance being P < 0.025. P < 0.05. P < 0.05 and P < 0.10. respectively.

A comparison of the mean score for patients with psychological dysfunctions (n = 28) for those who were cured (n = 15) and for those who were not (n = 13) produced statistically significant values on three key factors using one-tailed t-tests for small samples. Those cured were more unstable (C-) (P < 0.01), more insecure (O+) (P < 0.025) and more tense (Q4+) (P < 0.05).

When Type I, Type 2 and Type 3 BMS patients were compared the cure rates were 69%, 52%, and 66% repectively. Table 11:16 illustrates the cure rate frequency in Type 1 and Type 2 BMS patients in relation to specific psychological abnormality. It can be seen that the presence of instability, anxiety and depression in Type I BMS reduced cure rate by a small amount whereas in Type 2 their presence particularly when anxiety was present greatly reduced the likelihood of a successful outcome.

Of the 47 patients assessed 23 (49%) emerged as introverts, 11 (23%) as extroverts and 13 (28%) as ambiverts, and the cure rates were 65%, 73% and 38% respectively. Although no clear pattern emerged in this sample, severely abnormal introverts and ambiverts were more difficult to cure than abnormal extroverts.

11.10 TREATMENT WITH DOTHIEPEN

Dothiepen is a tricyclic antidepressant with anxiolytic effects. The mechanism of action of tricyclic antidepressants means that a therapeutic effect does not occur until treatment has been continued for 10-14 days. In psychological disorders generally it is likely that an antidepressant would be required for 3 months to 1 year before remission of the depressive illness is likely to occur (Storey, 1986).

Dothiepen has also been shown to have properties independant of its antidepressant effect namely an analgesic effect (Feinmann Harris and Cawley, 1984; Feinmann and Harris, 1984) and possible muscle relaxant

properties.

In this study Dothiepin was prescribed as Prothiaden 50 mg - 150 mg nocte for patients in whom there was marked cancerphobia, adverse social circumstances or where parafunctional activity was present and other therapeutic measures had failed to alter symptoms or had only partially improved symptoms.

Prothiaden could often be withdrawn after patients had accepted that BMS did not represent cancer or its development, adverse social circumstances had been removed or time had passed since an adverse life event, or parafunctional activity had been reduced possibly following construction of replacement dentures if appropriate.

Twenty three patients noted an improvement with Prothiaden therapy (Table 11.17). Of the 28 patients in the study group that were cancerphobic 11 had Prothiaden prescribed. 3 were cured by Prothiaden therapy alone, 5 were cured with Prothiaden and other treatment regimes, 2 were improved with Prothiaden therapy and only 1 failed to respond. Of the 31 patients with parafunctional activity 10 patients had Prothiaden therapy: 1 was cured by Prothiaden alone, 3 were partly cured with Prothiaden, 3 were partly improved and 3 did not respond to Prothiaden treatment.

Of the 37 patients who were thought to have significant adverse social or domestic circumstances 9 patients were prescribed Prothiaden: 3 were cured by this treatment alone, 3 were cured with Prothiaden and other

treatment. 2 were improved by Prothiaden and for 1 there was no change.

Overall, 35 patients were prescribed Prothiaden: 6 patients (17%) were cured with this medication alone. 10 patients (29%) were cured with Prothiaden and other treatment methods. 7 (20%) were also improved by Prothiaden and for 12 (34%) this medication had no effects. Twenty three patients (15%) in this study derived benefit from Prothiaden although as already stated this treatment was reserved for certain categories of patient.

11.11 PROVEN ALLERGIES

Although the significance of patch testing for patient 029 was uncertain, maintainence of a benzoic acid free diet resulted in the patient being largely symptom-free (cured). Patient 037 was reported by Lamey, Lamb and Forsyth (1987) and was symptom-free after adherance to a diet avoiding sorbic acid and propylene glycol. Patient 101 reported some improvement following vitamin B1 and B6 replacement therapy but was not rendered asymptomatic until avoidance of cinnamon in the diet.

An allergy to nickel was detected in patient 004 who subsequently had partial upper and partial lower dentures provided with bases constructed in a nickel free cobalt chromium alloy but there was no improvement in symptoms. Patient 009 had replacement dentures constructed using nylon bases and porcelain teeth but no improvement in symptoms was noted. Patient 044 had her partial denture replaced by precious metal and porcelain

fixed bridgework and was rendered symptom-free. Patient 127 had replacement dentures constructed in polycarbonate with porcelain teeth correcting denture design faults there was no improvement in symptoms.

11.12 CONCLUSIONS

Psychological aspects of BMS have tended to dominate the literature. This study has not revealed whether psychological dysfunction occurs as a result of BMS or BMS as a result of psychological dysfunction. Of the 47 patients tested by the Cattell's 16 PF form C 60% had psychological abnormalities. and 54% of these patients could be treated successfully.

In practice three main psychological factors emerged as being important in BMS. These were anxiety. depression and cancerphobia. If anxiety is present it is likely that treatment for BMS will be less successful particularly if the symptom pattern is that of Type 2.

Some patients volunteered that they thought anxiety and depression were factors in their condition. There were various reasons reported such as concern over mortgage repayments and difficult home circumstances. Some 28 patients had disabled or handicapped relations, causing social isolation and causing anxiety and tension within the home. Three patients had extremely adverse social circumstances and two of these lost their BMS symptoms once they were rehoused. In some cases depression appeared reactive, often owing to a recent family bereavement. In others there was no apparent reason, the depression could therefore be described as

endogenous. In Chapter 3.8 it was shown that at presentation 43 patients (29%) of the 146 patients asked said they had been or were being treated for depression although only 5 of these patients were on antidepressant medication and only 9 in all were on these types of drugs. Sleep disturbance has been shown to be indicative of depression (Walton, in Macleod, Edwards and Bouchier 1987). Seventy five of the 126 (60%) patients questioned about their sleep pattern admitted to difficulty getting to sleep or early morning wakening, irrespective of whether they thought that this represented a normal or abnormal sleep pattern for them. The Cattell's 16 PF Form C questionnaire revealed a 51% incidence of depression in those sampled.

An interesting feature was demonstrated when the outcome of treatment for smokers and non smokers was compared. Of the 37 smokers for whom outcome of treatment was known 19 (51%) were cured 1(3%) was improved, 14 (38%) reported no change and 3 patients said they were worse. For the 105 non smokers the figures were 66 (63%), 15 (14%), 23 (21%) and 1 (1%) respectively. This data shows that statistically significantly fewer smokers were cured or improved compared to non smokers ($p < 0.05$, chi-squared test).

Comparison of the relative importance of each of the aetiological factors discussed in this chapter is difficult since the structured treatment regime leads to successful outcome at various stages but in terms of numbers involved replacement dentures, management of

parafunctional activity, vitamin B1 and vitamin B6 replacement therapy and the use of the tricyclic antidepressant. Dothiepen (Prothiaden) were shown to be the most useful aspects of the management regime.

This study has shown that it is possible to cure 60% of patients with BMS and improve symptoms for a further 11% however only 47% of patients who were cured were cured as a result of treatment of one factor alone. This clearly shows the need to consider all possible aetiological factors as well as the psychological background against which the patients' symptoms are set.

PATIENT REFERENCE NUMBER	PRE-TREATMENT SEVERITY SCORE	POST-TREATMENT SEVERITY SCORE	TREATMENT OUTCOME
001	7	7	NO CHANGE
002	8	0	CURED
003	9	0	CURED
004	8	8	NO CHANGE
005	10	5	IMPROVED
006	10	10	NO CHANGE
007	10	4	IMPROVED
008	10	10	NO CHANGE
009	10	10	NO CHANGE
010	10	3	CURED
011	8	0	CURED
012	9	5	IMPROVED
013	6	1	CURED
014	7	0	CURED
015	8	4	IMPROVED
016	10	2	CURED
017	10	5	IMPROVED
018	5	0	CURED
019	8	1	CURED
020	10	0	CURED
021	8	0	CURED
022	8	0	CURED
023	10	0	CURED
024	9	9	NO CHANGE
025	10	10	NO CHANGE
026	7	3	CURED
027	10	0	CURED
028	9	9	NO CHANGE
029	10	1	CURED
030	5	5	NO CHANGE
031	8	0	CURED
032	8	2	CURED
033	9	0	CURED
034	7	0	CURED
035	9	0	CURED
036	9	3	CURED
037	7	0	CURED
038	10	10	NO CHANGE
039	5	5	NO CHANGE
040	4	4	NO CHANGE
041		NOT KNOWN	
042	8	1	CURED
043	8	8	NO CHANGE
044	10	0	CURED
045	7	0	CURED
046	8	2	CURED
047	8	8	NO CHANGE
048	7	7	NO CHANGE
049	8	0	CURED
050	5	0	CURED

Table 11:1 Severity score before and after treatment and treatment outcome.

PATIENT REFERENCE NUMBER	PRE-TREATMENT SEVERITY SCORE	POST-TREATMENT SEVERITY SCORE	TREATMENT OUTCOME
051		NOT KNOWN	
052	8	0	CURED
053	5	8	WORSE
054	9	4	IMPROVED
055	5	0	CURED
056	5	0	CURED
057	7	0	CURED
058	8	8	NO CHANGE
059	9	10	WORSE
060	4	4	NO CHANGE
061	9	0	CURED
062	10	0	CURED
063	10	0	CURED
064	10	10	NO CHANGE
065	9	10	WORSE
066	5	5	NO CHANGE
067	4	4	NO CHANGE
068		NOT KNOWN	
069	9	2	CURED
070		NOT KNOWN	
071	10	0	CURED
072	10	3	CURED
073	9	0	CURED
074	10	3	CURED
075	7	7	NO CHANGE
076	6	0	CURED
077	10	2	CURED
078	5	5	NO CHANGE
079	10	2	CURED
080	7	0	CURED
081	6	0	CURED
082	10	0	CURED
083	10	10	NO CHANGE
084	8	0	CURED
085	7	2	CURED
086	5	4	IMPROVED
087	8	0	CURED
088	8	8	NO CHANGE
089	7	1	CURED
090	10	2	CURED
091	8	2	CURED
092	9	0	CURED
093	10	10	NO CHANGE
094	6	10	WORSE
095	8	8	NO CHANGE
096	5	0	CURED
097		NOT KNOWN	
098	7	7	NO CHANGE
099	10	5	IMPROVED
100	7	7	NO CHANGE

Table 11:1 Severity score before and after treatment and treatment outcome.

PATIENT REFERENCE NUMBER	PRE-TREATMENT SEVERITY SCORE	POST-TREATMENT SEVERITY SCORE	TREATMENT OUTCOME
101	5	0	CURED
102	7	7	NO CHANGE
103	10	0	CURED
104	10	10	NO CHANGE
105	7	0	CURED
106	5	0	CURED
107	4	0	CURED
108	10	3	CURED
109	10	1	CURED
110	5	0	CURED
111	10	6	IMPROVED
112	7	0	CURED
113	4	0	CURED
114	8	2	CURED
115	7	2	CURED
116	7	0	CURED
117	10	3	CURED
118	8	0	CURED
119	10	4	IMPROVED
120	3	3	NO CHANGE
121	8	5	IMPROVED
122	10	10	NO CHANGE
123	10	0	CURED
124	5	3	CURED
125	7	5	IMPROVED
126	10	10	NO CHANGE
127	10	10	NO CHANGE
128	10	0	CURED
129	8	3	CURED
130	4	0	CURED
131		NOT KNOWN	
132	5	5	NO CHANGE
133	7	0	CURED
134	5	1	CURED
135		NOT KNOWN	
136	5	4	IMPROVED
137	10	1	CURED
138	5	0	CURED
139	9	5	IMPROVED
140	8	2	CURED
141	8	8	NO CHANGE
142	8	2	CURED
143	7	7	NO CHANGE
144		NOT KNOWN	
145	10	3	CURED
146	10	1	CURED
147	8	4	IMPROVED
148	10	2	CURED
149	10	1	CURED
150	8	5	IMPROVED

Table 11:1 Severity score before and after treatment and treatment outcome.

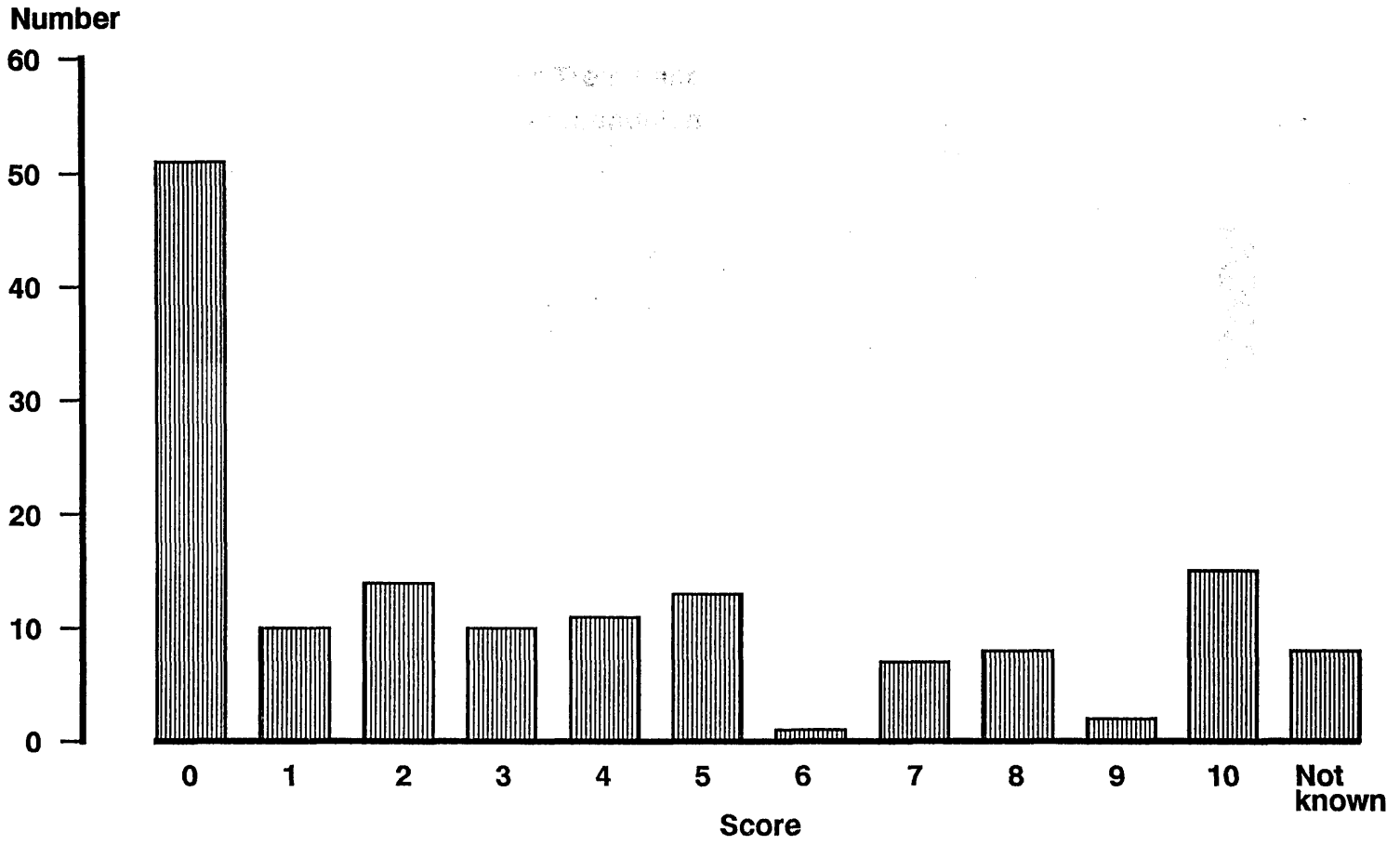


Figure 11:1 Severity of symptoms after treatment with at least an 18 month follow-up period

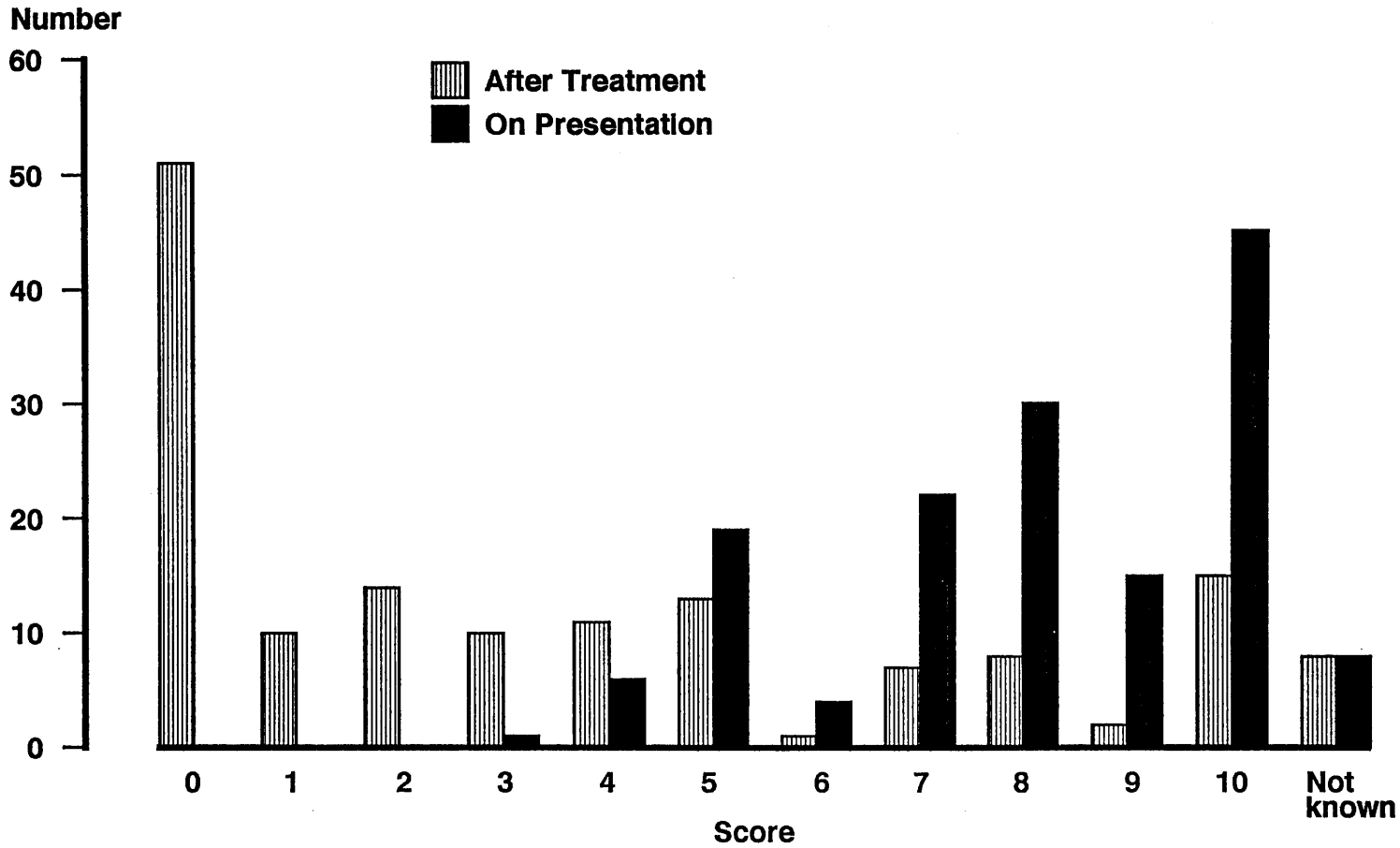


Figure 11: 2 Severity of symptoms before and after treatment with at least an 18 month follow-up period

	CURED	IMPROVED	NO CHANGE	WORSE
MALES	65%	12%	23%	-
FEMALES	58%	11%	27%	3%
OVERALL	59%	11%	27%	3%

Table 11.2 Result of treatment for 142 BMS patients with minimum of 18 months follow-up period.

AGE	NUMBER	CURED	IMPROVED	NO CHANGE	WORSE
31-40	8	63%	12%	25%	-
41-50	17	65%	12%	17%	6%
51-60	35	63%	6%	28%	3%
61-70	56	52%	18%	27%	3%
>70	26	62%	8%	31%	-

Table 11:3 The relationship between age and outcome of treatment for 142 BMS patients.

	NUMBER	CURED	IMPROVED	NO CHANGE	WORSE
PREMENOPAUSAL	8	75%	12.5%	12.5%	-
MENOPAUSAL	19	53%	11%	31%	5%
POSTMENOPAUSAL	98	60%	10%	28%	3%

Table 11:4 The relationship between menopausal status and outcome of treatment for 125 female patients.

	CURED	IMPROVED	NO CHANGE	WORSE
OVERALL	60% (85)	11% (16)	26% (37)	3% (4)
TONGUE	57% (63)	15% (16)	25% (28)	3% (3)
UPPER ALVEOLUS AND PALATE	55% (35)	9% (6)	30% (19)	6% (4)
LOWER ALVEOLUS	59% (31)	11% (6)	28% (15)	2% (1)
LIPS	61% (34)	13% (7)	21% (12)	5% (3)

Figures in brackets are totals.

Table 11:5 Outcome of treatment and site affected.

	TYPE 1	TYPE 2	TYPE 3	OVERALL
n =	50	76	16	142
CURED	68% (34)	49% (37)	75% (12)	60% (85)
IMPROVED	8% (4)	16% (12)	6% (1)	11% (16)
NO CHANGE	22% (11)	31% (24)	19% (3)	26% (37)
WORSE	2% (1)	4% (3)	-	3% (4)

Figures in brackets are totals.

Table 11:6 Outcome of treatment and BMS type.

PATIENT REFERENCE NUMBER	SOCIAL/DOMESTIC CIRCUMSTANCES SCORE	SEVERITY SCORE ON PRESENTATION	SEVERITY SCORE AFTER TREATMENT	OUTCOME
003	02	9	0	CURED
008	01	10	10	NO CHANGE
011	06	8	0	CURED
014	05	7	0	CURED
017	07	10	5	IMPROVED
020	05	10	0	CURED
022	05	8	0	CURED
023	04	10	0	CURED
027	07	10	0	CURED
031	07	8	0	CURED
034	07	7	0	CURED
046	06	8	2	CURED
047	05	8	8	NO CHANGE
052	07	8	0	CURED
053	05	5	8	WORSE
055	05	5	0	CURED
063	05	10	0	CURED
064	05	10	10	NO CHANGE
065	0	9	10	WORSE
071	05	10	0	CURED
074	0	10	3	CURED
076	02	6	0	CURED
079	07	10	2	CURED
083	02	10	10	NO CHANGE
085	06	7	2	CURED
092	01	9	0	CURED
098	03	7	7	NO CHANGE
099	04	10	5	IMPROVED
101	0	5	0	CURED
104	07	10	10	NO CHANGE
118	05	8	0	CURED
123	06	10	0	CURED
124	03	5	3	CURED
126	02	10	10	NO CHANGE
132	05	5	5	NO CHANGE
138	04	5	0	CURED
139	07	9	5	IMPROVED
145	06	10	3	CURED
150	05	8	5	IMPROVED

Table 11:7 Outcome of treatment for those patients with adverse social/domestic circumstances.

PATIENT REFERENCE NUMBER	EFFECT OF VITAMIN B COMPLEX REPLACEMENT
018	CURE
019	CURE
031	PART OF CURE
045	CURE
055	PART OF CURE
085	PART OF CURE
101	PART OF CURE
124	PART OF CURE
137	CURE

Table 11:8 Effect of vitamin B complex therapy on patients known to be deficient who reported improvement with this therapy

PATIENT REFERENCE NUMBER	EFFECT OF EMPIRICAL VITAMIN B1 & B6
010	CURE
012	IMPROVEMENT
016	PART OF CURE
042	CURE
062	PART OF CURE
074	PART OF CURE
076	PART OF CURE
087	PART OF CURE
096	PART OF CURE
103	PART OF CURE
105	PART OF CURE
123	PART OF CURE
130	PART OF CURE
140	PART OF CURE
142	PART OF CURE
145	PART OF CURE
148	PART OF CURE

Table 11:9 Effect of empirical vitamin B1 & B6 therapy on those patients who reported improvement with this therapy

PATIENT REFERENCE NUMBER	REDUCED SALIVARY GLAND FUNCTION (<0.5 ml/min from each parotid gland)	EFFECT OF SALIVARY SUBSTITUTES ON BMS SYMPTOMS
001	Y	NO CHANGE
004	Y	NO CHANGE
007	Y	NO CHANGE
009	N	NO CHANGE
011	N	NO CHANGE
012	N	IMPROVED
017	N	IMPROVED
022	N	NO CHANGE
023	N	NO CHANGE
026	Y	IMPROVED
033	-	IMPROVED
035	N	NO CHANGE
036	N	NO CHANGE
043	N	NO CHANGE
050	Y	NO CHANGE
051	N	NO CHANGE
053	-	NO CHANGE
055	N	IMPROVED
059	Y	NO CHANGE
062	N	CURED
063	N	NO CHANGE

Table 11:10 Effect of salivary substitutes on BMS symptoms on those patients who had reduced parotid salivary gland activity or those who felt their mouths were dry.

PATIENT REFERENCE NUMBER	REDUCED SALIVARY GLAND FUNCTION (<0.5 ml/min from each parotid gland)	EFFECT OF SALIVARY SUBSTITUTES ON BMS SYMPTOMS
064	N	NO CHANGE
066	N	NO CHANGE
068	-	NO CHANGE
069	N	NO CHANGE
074	-	NO CHANGE
075	-	NO CHANGE
079	N	CURED
081	N	NO CHANGE
087	Y	IMPROVED
089	N	NO CHANGE
090	N	NO CHANGE
093	Y	NO CHANGE
094	N	NO CHANGE
096	N	NO CHANGE
098	N	NO CHANGE
099	N	NO CHANGE
102	Y	NO CHANGE
104	N	NO CHANGE
115	-	NO CHANGE
120	N	NO CHANGE

Table 11:10 Effect of salivary substitutes on BMS symptoms on those patients who had reduced parotid salivary gland activity or those who felt their mouths were dry.

PATIENT REFERENCE NUMBER	REDUCED SALIVARY GLAND FUNCTION (<0.5 ml/min from each parotid gland)	EFFECT OF SALIVARY SUBSTITUTES ON BMS SYMPTOMS
122	N	NO CHANGE
123	N	IMPROVED
124	N	NO CHANGE
128	Y	NO CHANGE
129	N	NO CHANGE
130	Y	NO CHANGE
131	Y	NO CHANGE
132	N	NO CHANGE
139	N	IMPROVED
140	N	NO CHANGE
144	N	NO CHANGE
145	N	NO CHANGE
147	N	NO CHANGE

Table 11:10 Effect of salivary substitutes on BMS symptoms on those patients who had reduced parotid salivary gland activity or those who felt their mouths were dry.

PATIENT REFERENCE NUMBER	EFFECT OF SALIVARY SUBSTITUTES ON BMS SYMPTOMS
019	NO CHANGE
027	NO CHANGE
030	NO CHANGE
049	NO CHANGE
073	IMPROVED
086	IMPROVED
121	NO CHANGE

Table 11:11 Effect of salivary substitutes on BMS symptoms of those patients who were not complaining of dry mouth but whose salivary gland function was measured as being reduced.

PATIENT REFERENCE NUMBER	OUTCOME OF TREATMENT	IMPROVEMENT OR CURE. RESULT OF REASSURANCE AND OR ANTIDEPRESSANTS	OTHER FACTORS INVOLVED IN CURE OR IMPROVEMENT
010	CURED	NO	YES
011	CURED	YES	NO
013	CURED	YES	NO
014	CURED	YES	NO
017	IMPROVED	YES	YES
023	CURED	YES	YES
033	CURED	YES	YES
034	CURED	YES	YES
038	NO CHANGE		
046	CURED	YES	NO
064	NO CHANGE		
065	WORSE		
067	NO CHANGE		
071	CURED	YES	NO
082	CURED	NO	YES
085	CURED	YES	YES
090	CURED	YES	YES
105	CURED	YES	YES
116	CURED	YES	NO
120	NO CHANGE		
128	CURED	YES	NO
129	CURED	YES	NO
136	IMPROVED	YES	NO
138	CURED	YES	YES
140	CURED	YES	YES
142	CURED	YES	YES
145	CURED	YES	YES
149	CURED	NO	YES

Table 11:12 Outcome of treatment of patients who expressed a fear of cancer.

PATIENT REFERENCE NUMBER	SEX	AGE	BMS TYPE	PSYCHOLOGICAL STATUS	INITIAL SEVERITY SCORE	POST TREATMENT SCORE	RELEVANT TREATMENT METHOD
002	F	36	1	SEVERELY ABNORMAL	8	0	NEW UPPER DENTURE
014	F	78	1	SEVERELY ABNORMAL	7	0	CANCERPHOBIA REASSURANCE PROTHIADEN THERAPY
016	F	65	1	NORMAL	10	2	PROTHIADEN THERAPY
026	F	59	2	SEVERELY ABNORMAL	7	3	SALIVA SUBSTITUTE PROTHIADEN THERAPY
031	F	74	3	ABNORMAL	8	0	VITAMIN B1 AND B6 NEW COMPLETE DENTURES
034	F	63	2	ABNORMAL	7	0	CANCERPHOBIA REASSURANCE NEW COMPLETE DENTURES
035	F	65	1	ABNONRMAL	9	0	NEW COMPLETE DENTURES

Table 11:13 Psychological status for those patients sampled with the Cattell's 16 PF Form C questionnaire who were subsequently cured and the treatment factors responsible for the cure.

PATIENT REFERENCE NUMBER	SEX	AGE	BMS TYPE	PSYCHOLOGICAL STATUS	INITIAL SEVERITY SCORE	POST TREATMENT SCORE	RELEVANT TREATMENT METHOD
037	F	62	2	ABNORMAL	7	0	FOOD ALLERGIES
049	F	51	2	ABNORMAL	8	0	VITAMIN B1 AND B6
055	F	66	2	ABNORMAL	5	0	VITAMIN B1 AND B6 SALIVA SUBSTITUTE
056	F	49	2	ABNORMAL	5	0	CLIMACTERIC
057	F	55	1	NORMAL	7	0	VITAMIN B12
061	F	57	3	NORMAL	9	0	PROTHIADEN THERAPY
080	F	40	1	NORMAL	7	0	IRON REPLACEMENT
081	M	71	2	NORMAL	6	0	DIABETIC THERAPY
082	F	69	1	NORMAL	10	0	VITAMIN B12 CANCERPHOBIA REASSURANCE
089	F	62	2	ABNORMAL	7	1	ANTIFUNGAL THERAPY NEW COMPLETE DENTURES
092	F	64	2	SEVERELY ABNORMAL	9	0	PROTHIADEN THERAPY
096	F	39	1	NORMAL	5	0	VITAMIN B1 AND B6

Table 11:13 Psychological status for those patients sampled with the Cattell's 16 PF Form C questionnaire who were subsequently cured and the treatment factors responsible for the cure.

PATIENT REFERENCE NUMBER	SEX	AGE	BMS TYPE	PSYCHOLOGICAL STATUS	INITIAL SEVERITY SCORE	POST TREATMENT SCORE	RELEVANT TREATMENT METHOD
101	F	56	2	SEVERELY ABNORMAL	5	0	VITAMIN B1 AND B6 NEW PARTIAL UPPER DENTURE
107	F	43	3	NORMAL	4	0	IRON REPLACEMENT
108	M	63	2	NORMAL	10	3	PROTHIADEN THERAPY NEW COMPLETE DENTURES
112	M	47	2	NORMAL	7	0	ANTIFUNGAL THERAPY
113	M	58	2	ABNORMAL	4	0	IRON REPLACEMENT
114	F	53	1	NORMAL	8	2	NEW COMPLETE DENTURES
129	F	64	1	SEVERELY ABNORMAL	8	3	CANCERPHOBIA REASSURANCE
130	F	63	1	NORMAL	4	0	NEW COMPLETE DENTURES
137	F	69	3	NORMAL	10	1	VITAMIN B2 AND B6

Table 11:13 Psychological status for those patients sampled with the Cattell's 16 PF Form C questionnaire who were subsequently cured and the treatment factors responsible for the cure.

	Number	Number cured	% cured
NORMAL	19	13	68
SLIGHTLY ABNORMAL	11	9	82
SEVERELY ABNORMAL	17	6	35

Table 11:14 Psychological status of BMS patients and the proportion of patients treated successfully.

	Number	% of total sample	Number cured	% cured
INSTABILITY	22	47	12	56
ANXIETY	21	45	8	38
DEPRESSION	24	51	12	50

Table 11:15 Proportion of BMS patients with specific psychological abnormalities and the number of these patients successfully treated.

PSYCHOLOGICAL ABNORMALITY	TYPE 1 % CURED	TYPE 2 % CURED	TYPE 3 % CURED
INSTABILITY	60 (n = 5)	44 (n = 16)	100 (n = 1)
ANXIETY	66 (n = 6)	27 (n = 15)	-
DEPRESSION	60 (n = 5)	44 (n = 18)	-
NORMAL	70 (n = 10)	75 (n = 4)	60 (n = 5)

Table 11.16 Cure rate frequency in Types 1, 2 and 3 BMS patients and relationship to specific psychological abnormality compared to cure rate of patients with no psychological abnormality.

PATIENTS REFERENCE NUMBER	CANCEROPHOBIA	PARAFUNCTIONAL ACTIVITY	ADVERSE SOCIAL CIRCUMSTANCES (SCORE)	EFFECT OF PROTHIADEN
005	NO	YES	NO	PART OF IMPROVEMENT
007	NO	YES	NO	PART OF IMPROVEMENT
011	YES	NO	YES	CURED
013	YES	NO	NO	CURED
014	YES	YES	YES	CURED
016	NO	YES	BORDERLINE	PART OF CURE
017	YES	YES	YES	PART OF IMPROVEMENT
020	NO	NO	YES	PART OF CURE
022	NO	NO	YES	CURED
023	YES	YES	YES	PART OF CURE
026	NO	NO	BORDERLINE	PART OF CURE
038	YES	NO	NO	NO CHANGE
043	NO	NO	NO	NO CHANGE
048	NO	YES	NO	NO CHANGE
059	NO	NO	NO	NO CHANGE
061	NO	NO	BORDERLINE	CURED
085	YES	NO	YES	PART OF CURE

Table 11:17 Effect of prothiaden treatment on patients' BMS symptoms.

PATIENTS REFERENCE NUMBER	CANCEROPHOBIA	PARAFUNCTIONAL ACTIVITY	ADVERSE SOCIAL CIRCUMSTANCES (SCORE)	EFFECT OF PROTHIADEN
088	NO	NO	NO	NO CHANGE
090	YES	NO	NO	PART OF CURE
094	NO	NO	NO	OVERALL BMS WORSE NO CHANGE WITH PROTHIADEN
100	NO	NO	NO	NO CHANGE
105	YES	NO	NO	PART OF CURE
108	NO	NO	NO	PART OF CURE
109	NO	NO	NO	CURED
111	NO	NO	NO	IMPROVEMENT
112	NO	NO	NO	OVERALL CURED NO CHANGE WITH PROTHIADEN
114	NO	YES	NO	OVERALL CURED NO CHANGE WITH PROTHIADEN
119	NO	NO	BORDERLINE	PART OF IMPROVEMENT
122	NO	NO	NO	NO CHANGE
127	NO	NO	NO	NO CHANGE
132	NO	YES	YES	NO CHANGE
136	YES	NO	NO	IMPROVEMENT
139	NO	NO	YES	IMPROVEMENT
140	YES	YES	NO	PART OF CURE
146	NO	NO	NO	PART OF CURE

Table 11:17 Effect of prothiaden treatment on patients' BMS symptoms.

CHAPTER 12

MANAGEMENT OF PATIENTS WITH BURNING MOUTH SYNDROME

DISCUSSION AND RECOMMENDATIONS

The treatment protocol devised in this study has developed as a result of careful consideration and application of knowledge of the literature. The stepwise quantification of reduction of BMS score argues in favour of a structured management regime. In the previous chapter it was shown that when treatment of the known aetiological factors in BMS is instituted some 60% of patients would consider themselves cured with a further 11% of patients reporting some sustained improvement.

Despite the few statistically significant findings in this study, the results have indicated trends which have allowed the treatment regime described later in this chapter to develop. Because of the multifactorial nature of the condition and despite the fact 150 patients were investigated, numbers of patients in various categories inevitably were small. This present study was the first to investigate BMS prospectively and is the largest to date where a broad view of the condition has been investigated. Previously only Main and Basker (1983) investigating only 37 patients had attempted to look at a range of aetiological factors. Comprehensive prospective data has been collected on a further 250 patients in Glasgow, however it is likely that a multicentre study using a standard protocol such as has been proposed in this study will be required to generate sufficient numbers

so that sophisticated multifactorial analysis can be undertaken.

It is essential that the initial consultation is unhurried so that in addition to determining full details of the patient's complaint of burning, tactful but direct enquiry can be made into home and social circumstances and cancerphobia. In this way problems with stress, anxiety or depression can be identified. Only in the minority of patients (21% of those questioned) does the burning sensation interfere with sleep although an accompanying psychological problem may produce sleep disturbance, particularly early morning wakening. In the study one fifth of patients were responsible for the care of handicapped relatives. Patients themselves often believe that the complaint signifies emotional instability a belief reinforced by attendance at many diverse clinics where nothing is found on clinical examination. A further feature of BMS is the personal isolation it can produce particularly since patients rarely have heard of the condition nor know anyone else with the complaint. In order to be objective when assessing a patient's psychological status it is recommended that some form of psychological proforma is employed giving information particularly about anxiety and depression, since this study has demonstrated that it might be wise not to consider BMS patients as a homogenous group, and that Type 2 BMS patients particularly if anxiety is present were less successfully treated. The Cattell's 16PF form C used in this study is lengthy and therefore the use of the HAD

scale (Lamey and Lamb. 1989) provides a rapid initial screening. In order that an assessment of the initial symptoms and the response to treatment can be made. it is helpful to ask the patient to quantify their burning sensation using the analogue scale. The same scale is useful in determining happiness or otherwise with home and social circumstances and following this present study has been used to measure the degree of cancerphobia.

Also at the first visit it is essential to measure haemoglobin. ferritin. corrected whole blood folate, and vitamin B12 levels. In addition a blood glucose estimation should be undertaken. If possible vitamin B1, B2 and B6 concentrations should be assayed. If this facility is not available then replacement regime of vitamin B1 300 mg and vitamin B6 150 mg per day in divided doses for 4 weeks may be effective in deficient patients and should be tried empirically. Vitamin B2 deficiency is less common and routine replacement is probably not justified. In addition there are practical considerations since the preparation is not available commercially in the necessary dose. It is advised that self medication with multivitamins is avoided as this can greatly complicate biochemical evaluation. Reduced salivary gland function can be quantified by stimulated parotid flow rates in the hospital situation but not in medical or dental practice. However, in the absence of quantification if the patient thinks that the burning sensation and oral dryness are the same thing, then saliva substitutes may be effective.

Since it is not always recognised that pathogenic

numbers of candidal species can be isolated from the oral cavity in the absence of clinical signs, an oral rinse (Samaranayake et al 1986) is preferred to quantify carriage of candidal species.

The results of the haematological investigations and the blood glucose estimation are usually available after four weeks, and it is useful to assess progress at that time following vitamin B1 and vitamin B6 therapy. Any other haematological deficiencies detected need specialist investigation and replacement therapy in conjunction with the patient's medical practitioner. Arrangements should be made to complete a glucose tolerance test to exclude hyperglycaemia if indicated and once detected, undiagnosed diabetes mellitus requires referral for glycaemic control. At this second visit it is also important to reassure once again if the patient is cancerphobic, and to continue enquiries about adverse home circumstances. If candidal species have been isolated topical antifungal therapy, such as Amphotericin B lozenges or Nystan pastilles four times daily for 4 weeks should be prescribed and denture hygiene instructions given if appropriate.

An initial assessment of dentures, if worn, is important. The patient should be observed for signs of parafunctional activity such as clenching or tongue thrusting, attrition facets on acrylic denture teeth or scalloping of the lateral margin of the tongue are useful indicators of parafunctional habits. Dentures should be examined for features that contribute to overloading of

the supporting tissues or tongue restriction, these include underextension of denture bases, excessive occlusal face height, faulty bucco-lingual tooth position and high level of the occlusal plane.

New dentures should be constructed as appropriate after other aetiological factors listed previously in this chapter have been dealt with. In addition it is wise not to construct new dentures until parafunctional activity has been reduced. If Prothiaden is to be prescribed this should be done also before dentures are constructed and maintained throughout the post delivery stage, until the dentures are accepted. In addition to reducing parafunctional activity Prothiaden was found useful in alleviating reactive depression to stress or in helping patients cope with adverse home and social circumstances and for helping those patients who were cancerphobic to overcome their fears. The lowest therapeutic dose should be employed starting initially with 50-75 mg nocte however for some patients up to 150 mg nocte was required.

Allergy testing should be reserved for those patients with a Type 3 pattern of BMS but may also have a role in those patients in whom no cure has been achieved. The most important allergens discovered in this study were food related products and denture materials. If a food allergen is identified professional dietary counselling should be employed. Constructing complete dentures with base materials other than polymethylmethacrylate (PMMA) is difficult. Alternative materials such as nylon and polycarbonate are not readily available and vulcanite is

becoming less easy to obtain. It is unwise to label a patient allergic to PMMA unless properly controlled skin patch testing has been undertaken, and even then its role with regard to the oral mucosa is uncertain.

In this study 19 of the 104 female patients were perimenopausal and 17 of these patients had additional symptoms of the climacteric such as facial flushing and night sweats. In only 6 patients was the climacteric thought to be playing a part in BMS and this would be expected to improve with time. Hormone replacement therapy has been shown by most investigators not to be effective in relieving the oral burning sensation (Chapter 1.5).

It is important that patients who have responded to treatment are followed up in the long term to assess sustained improvement or perhaps relapse. For those patients in whom treatment was ineffective long term follow up is also necessary since predeficiency states and occult undiagnosed maturity onset diabetes can manifest at a later stage.

Finally future strategies should focus on the 28 % of non responders often Type 2 to determine whether other treatment modalities such as group therapy or psychological counselling have a role. Nevertheless other factors of potential importance also require expansion such as the role of additional haematinic deficiencies, including the significance of deficiencies of vitamin A, C, D and E suggested in this study, sialochemistry and the physiological aspects of pain perception.

APPENDIX I

RAW DATA

Patient Reference Number	Age	Sex	Menopausal Status	Source of Referral	Duration of Symptoms*	BMS Type	Severity of symptoms at Presentation
001	69	F	POSTMENOPAUSAL	GDP	4	2	7
002	36	F	PREMENOPAUSAL	GDP	2	1	8
003	31	F	PREMENOPAUSAL	GMP	1	1	9
004	58	F	POSTMENOPAUSAL	GDP	2	2	8
005	68	F	POSTMENOPAUSAL	GDP	3	2	10
006	60	F	POSTMENOPAUSAL	GDP	5	1	10
007	75	F	POSTMENOPAUSAL	GDP	3	2	10
008	63	F	POSTMENOPAUSAL	SELF	3	1	10
009	69	F	POSTMENOPAUSAL	GDP	2	2	10
010	72	F	POSTMENOPAUSAL	CONSULT.	3	2	10
011	57	F	POSTMENOPAUSAL	SELF	2	1	8
012	64	F	POSTMENOPAUSAL	GDP	5	1	9
013	57	F	POSTMENOPAUSAL	GMP	4	3	6
014	78	F	POSTMENOPAUSAL	SELF	6	1	7
015	61	F	POSTMENOPAUSAL	SELF	3	2	8

*DURATION OF SYMPTOMS

<6 months	= 1	5 - 10 years	= 5
6 months to 1 year	= 2	10 - 15 years	= 6
1 - 2 years	= 3	15 - 30 years	= 7
2 - 5 years	= 4	>30 years	= 8

Appendix: 1 Age, sex, menopausal status, source of referral, duration of symptoms, BMS type and severity of symptoms at presentation.

Patient Reference Number	Age	Sex	Menopausal Status	Source of Referral	Duration of Symptoms*	BMS Type	Severity of symptoms at Presentation
016	65	F	POSTMENOPAUSAL	GMP	4	1	10
017	69	F	POSTMENOPAUSAL	GMP	1	3	10
018	58	M		SELF	1	1	5
019	51	F	MENOPAUSAL	GDP	2	2	8
020	78	F	POSTMENOPAUSAL	SELF	3	1	10
021	58	F	POSTMENOPAUSAL	SELF	4	1	8
022	66	F	POSTMENOPAUSAL	SELF	2	1	8
023	49	F	POSTMENOPAUSAL	GDP	1	2	10
024	79	F	POSTMENOPAUSAL	GMP	2	2	9
025	52	F	MENOPAUSAL	GDP	4	2	10
026	59	F	POSTMENOPAUSAL	CONSULT.	1	2	7
027	70	F	POSTMENOPAUSAL	SELF	1	2	10
028	65	F	POSTMENOPAUSAL	GDP	4	1	9
029	51	F	POSTMENOPAUSAL	CONSULT.	5	2	10
030	57	F	MENOPAUSAL	GDP	4	2	5

*DURATION OF SYMPTOMS

<6 months	= 1	5 - 10 years	= 5
6 months to 1 year	= 2	10 - 15 years	= 6
1 - 2 years	= 3	15 - 30 years	= 7
2 - 5 years	= 4	>30 years	= 8

Appendix: 1 Age, sex, menopausal status, source of referral, duration of symptoms, BMS type and severity of symptoms at presentation.

Patient Reference Number	Age	Sex	Menopausal Status	Source of Referral	Duration of Symptoms*	BMS Type	Severity of symptoms at Presentation
031	74	F	POSTMENOPAUSAL	GMP	3	3	8
032	35	F	PREMENOPAUSAL	GDP	4	3	8
033	59	F	POSTMENOPAUSAL	CONSULT.	3	3	9
034	63	F	MENOPAUSAL	SELF	5	2	7
035	65	F	POSTMENOPAUSAL	GDP	2	1	9
036	68	F	POSTMENOPAUSAL	GDP	1	2	9
037	62	F	POSTMENOPAUSAL	GMP	3	2	7
038	54	M		SELF	1	2	10
039	38	F	MENOPAUSAL	GDP	1	1	5
040	68	M		GMP	4	2	4
041	58	M		GDP	1	1	
042	55	F	POSTMENOPAUSAL	GDP	1	1	8
043	64	F	POSTMENOPAUSAL	GDP	1	2	8
044	42	F	MENOPAUSAL	GDP	2	1	10
045	57	F	POSTMENOPAUSAL	GDP	1	1	7

*DURATION OF SYMPTOMS

<6 months	= 1	5 - 10 years	= 5
6 months to 1 year	= 2	10 - 15 years	= 6
1 - 2 years	= 3	15 - 30 years	= 7
2 - 5 years	= 4	>30 years	= 8

Appendix: 1 . Age, sex, menopausal status, source of referral, duration of symptoms, BMS type and severity of symptoms at presentation.

Patient Reference Number	Age	Sex	Menopausal Status	Source of Referral	Duration of Symptoms*	BMS Type	Severity of symptoms at Presentation
046	68	M		GMP	1	2	8
047	78	F	POSTMENOPAUSAL	SELF	4	2	8
048	59	F	POSTMENOPAUSAL	GDP	4	2	7
049	51	F	PREMENOPAUSAL	GDP	2	2	8
050	59	F	POSTMENOPAUSAL	GDP	1	3	5
051	57	F	POSTMENOPAUSAL	GDP	4		
052	69	F	POSTMENOPAUSAL	GDP	1	1	8
053	56	F	POSTMENOPAUSAL	SELF	1	1	5
054	47	F	MENOPAUSAL	GMP	1	1	9
055	66	F	POSTMENOPAUSAL	GDP	2	2	5
056	49	F	MENOPAUSAL	SELF	1	2	5
057	55	F	POSTMENOPAUSAL	GDP	3	1	7
058	72	F	POSTMENOPAUSAL	GDP	3	1	8
059	62	F	POSTMENOPAUSAL	GMP	2	2	9
060	43	F	MENOPAUSAL	GDP	3	3	4

*DURATION OF SYMPTOMS

<6 months	= 1	5 - 10 years	= 5
6 months to 1 year	= 2	10 - 15 years	= 6
1 - 2 years	= 3	15 - 30 years	= 7
2 - 5 years	= 4	>30 years	= 8

Appendix: 1 Age, sex, menopausal status, source of referral, duration of symptoms, BMS type and severity of symptoms at presentation.

Patient Reference Number	Age	Sex	Menopausal Status	Source of Referral	Duration of Symptoms*	BMS Type	Severity of symptoms at Presentation
061	57	F	POSTMENOPAUSAL	GDP	3	3	9
062	85	F	POSTMENOPAUSAL	SELF	2	2	10
063	65	F	POSTMENOPAUSAL	CONSULT.	3	2	10
064	47	F	POSTMENOPAUSAL	GMP	1	2	10
065	62	F	POSTMENOPAUSAL	SELF	1	2	9
066	78	F	POSTMENOPAUSAL	GMP	1	3	5
067	39	F	PREMENOPAUSAL	GMP	2	2	4
068	72	F	POSTMENOPAUSAL	GDP	3		
069	67	M		GMP	3	1	9
070	63	M		SELF	1	2	
071	54	F	POSTMENOPAUSAL	GDP	2	1	10
072	76	F	POSTMENOPAUSAL	GMP	3	2	10
073	61	F	POSTMENOPAUSAL	SELF	2	2	9
074	76	F	POSTMENOPAUSAL	GMP	4	2	10
075	56	F	POSTMENOPAUSAL	GDP	1	3	7

*DURATION OF SYMPTOMS

<6 months	= 1	5 - 10 years	= 5
6 months to 1 year	= 2	10 - 15 years	= 6
1 - 2 years	= 3	15 - 30 years	= 7
2 - 5 years	= 4	>30 years	= 8

Appendix: 1 Age, sex, menopausal status, source of referral, duration of symptoms, BMS type and severity of symptoms at presentation.

Patient Reference Number	Age	Sex	Menopausal Status	Source of Referral	Duration of Symptoms*	BMS Type	Severity of symptoms at Presentation
076	76	F	POSTMENOPAUSAL	GDP	1	1	6
077	64	F	POSTMENOPAUSAL	SELF	2	1	10
078	61	F	POSTMENOPAUSAL	GDP	3	1	5
079	44	M		GDP	1	2	10
080	40	F	MENOPAUSAL	GDP	2	1	7
081	71			GMP	1	2	6
082	69	F	POSTMENOPAUSAL	GMP	1	1	10
083	70	F	POSTMENOPAUSAL	GDP	1	2	10
084	55	F	POSTMENOPAUSAL	GDP	1	2	8
085	61	M		SELF	1	3	7
086	63	M		GMP	3	2	5
087	66	F	POSTMENOPAUSAL	GDP	2	2	8
088	74	F	POSTMENOPAUSAL	GDP	4	2	8
089	62	F	POSTMENOPAUSAL	GMP	1	2	7
090	55	F	POSTMENOPAUSAL	GDP	3	2	10

*DURATION OF SYMPTOMS

<6 months	= 1	5 - 10 years	= 5
6 months to 1 year	= 2	10 - 15 years	= 6
1 - 2 years	= 3	15 - 30 years	= 7
2 - 5 years	= 4	>30 years	= 8

Appendix: 1 Age, sex, menopausal status, source of referral, duration of symptoms, BMS type and severity of symptoms at presentation.

Patient Reference Number	Age	Sex	Menopausal Status	Source of Referral	Duration of Symptoms*	BMS Type	Severity of symptoms at Presentation
091	55	F	POSTMENOPAUSAL	GDP	3	1	8
092	64	F	POSTMENOPAUSAL	GDP	5	2	9
093	75	F	POSTMENOPAUSAL	SELF	3	1	10
094	47	F	MENOPAUSAL	SELF	3	2	6
095	63	F	POSTMENOPAUSAL	GDP	4	2	8
096	39	F	MENOPAUSAL	CONSULT.	2	1	5
097	55	F	POSTMENOPAUSAL	GDP	1		
098	53	F	POSTMENOPAUSAL	GDP	1	2	7
099	71	F	POSTMENOPAUSAL	GDP	1	2	10
100	64	F	POSTMENOPAUSAL	SELF	3	1	7
101	56	F	MENOPAUSAL	GDP	3	2	5
102	50	F	MENOPAUSAL	CONSULT.	4	2	7
103	63	F	POSTMENOPAUSAL	GMP	1	2	10
104	76	F	POSTMENOPAUSAL	SELF	3	2	10
105	46	F	MENOPAUSAL	GDP	2	2	7

*DURATION OF SYMPTOMS

<6 months	= 1	5 - 10 years	= 5
6 months to 1 year	= 2	10 - 15 years	= 6
1 - 2 years	= 3	15 - 30 years	= 7
2 - 5 years	= 4	>30 years	= 8

Appendix: 1 Age, sex, menopausal status, source of referral, duration of symptoms, BMS type and severity of symptoms at presentation.

Patient Reference Number	Age	Sex	Menopausal Status	Source of Referral	Duration of Symptoms*	BMS Type	Severity of symptoms at Presentation
106	75	F	POSTMENOPAUSAL	GDP	3	1	5
107	43	F	MENOPAUSAL	SELF	3	3	4
108	63	M		GMP	1	2	10
109	46	F	MENOPAUSAL	GDP	3	2	10
110	76	M		GDP	1	3	5
111	54	F	POSTMENOPAUSAL	SELF	2	1	10
112	47	M		GDP	4	2	7
113	58	M		GDP	1	2	4
114	53	F	POSTMENOPAUSAL	SELF	4	1	8
115	74	F	POSTMENOPAUSAL	SELF	3	1	7
116	66	F	POSTMENOPAUSAL	GDP	1	3	7
117	37	F	PREMENOPAUSAL	GDP	3	2	10
118	48	F	POSTMENOPAUSAL	GDP	1	2	8
119	64	F	POSTMENOPAUSAL	GMP	3	2	10
120	75	M		GDP	2	2	3

*DURATION OF SYMPTOMS

<6 months	= 1	5 - 10 years	= 5
6 months to 1 year	= 2	10 - 15 years	= 6
1 - 2 years	= 3	15 - 30 years	= 7
2 - 5 years	= 4	>30 years	= 8

Appendix: 1 Age, sex, menopausal status, source of referral, duration of symptoms, BMS type and severity of symptoms at presentation.

Patient Reference Number	Age	Sex	Menopausal Status	Source of Referral	Duration of Symptoms*	BMS Type	Severity of symptoms at Presentation
121	64	F	POSTMENOPAUSAL	CONSULT.	3	2	8
122	58	F	MENOPAUSAL	GDP	2	1	10
123	75	F	POSTMENOPAUSAL	SELF	3	2	10
124	66	M		GDP	1	3	5
125	65	F	POSTMENOPAUSAL	GMP	1	2	7
126	60	M		SELF	2	1	10
127	70	F	POSTMENOPAUSAL	GDP	1	2	10
128	63	F	POSTMENOPAUSAL	GMP	2	2	10
129	64	F	POSTMENOPAUSAL	GMP	3	1	8
130	63	F	POSTMENOPAUSAL	SELF	5	1	4
131	68	F	POSTMENOPAUSAL	SELF	4		
132	64	F	POSTMENOPAUSAL	CONSULT.	1	2	5
133	80	F	POSTMENOPAUSAL	GMP	2	2	7
134	63	F	POSTMENOPAUSAL	GDP	2	1	5
135	66	F	POSTMENOPAUSAL	GMP	4	2	

*DURATION OF SYMPTOMS

<6 months	= 1	5 - 10 years	= 5
6 months to 1 year	= 2	10 - 15 years	= 6
1 - 2 years	= 3	15 - 30 years	= 7
2 - 5 years	= 4	>30 years	= 8

Appendix: 1 Age, sex, menopausal status, source of referral, duration of symptoms, BMS type and severity of symptoms at presentation.

Patient Reference Number	Age	Sex	Menopausal Status	Source of Referral	Duration of Symptoms*	BMS Type	Severity of symptoms at Presentation
136	51	F	MENOPAUSAL	GMP	1	2	5
137	69	F	POSTMENOPAUSAL	GMP	3	3	10
138	49	F	POSTMENOPAUSAL	GDP	2	1	5
139	59	F	POSTMENOPAUSAL	GDP	1	2	9
140	70	F	POSTMENOPAUSAL	GDP	1	1	8
141	65	F	POSTMENOPAUSAL	GDP	4	1	8
142	73	F	POSTMENOPAUSAL	GDP	3	1	8
143	62	F	POSTMENOPAUSAL	GDP	2	2	7
144	63	F	POSTMENOPAUSAL	GDP	2		
145	43	F	PREMENOPAUSAL	GDP	2	2	10
146	67	F	POSTMENOPAUSAL	GDP	7	1	10
147	41	F	PREMENOPAUSAL	GDP	2	2	8
148	78	F	POSTMENOPAUSAL	GMP	6	1	10
149	68	F	POSTMENOPAUSAL	SELF	3	2	10
150	66	M		GDP	3	1	8

*DURATION OF SYMPTOMS

<6 months	= 1	5 - 10 years	= 5
6 months to 1 year	= 2	10 - 15 years	= 6
1 - 2 years	= 3	15 - 30 years	= 7
2 - 5 years	= 4	>30 years	= 8

Appendix: 1 Age, sex, menopausal status, source of referral, duration of symptoms, BMS type and severity of symptoms at presentation.

PATIENT REFERENCE NUMBER	TIP OF TONGUE	DORSUM OF TONGUE	R LATERAL BORDER OF TONGUE	L LATERAL BORDER OF TONGUE	PALATE	UPPER RIDGE	LOWER RIDGE	UPPER LIP	LOWER LIP	WHOLE MOUTH	THROAT	BUCCAL MUCOSA	FLOOR OF MOUTH
001	*	*	*	*	*			*	*				
002					*								
003	*	*	*	*	*								
004	*	*	*	*				*	*				
005	*		*	*	*	*	*	*	*				
006					*	*	*						
007					*	*	*						
008							*						
009	*		*	*	*	*	*						
010	*	*											
011	*	*	*	*	*			*	*				
012			*	*									
013								*	*				
014					*	*	*						
015				*				*				*	
016	*						*		*				
017	*	*	*	*				*	*	*			
018	*	*	*	*			*						
019	*	*	*	*	*								
020	*	*	*	*		*	*						
021	*		*										
022							*						
023	*				*	*	*	*	*				
024	*												
025								*	*				
026	*	*			*						*	*	
027								*					
028					*	*	*						
029								*	*			*	
030	*	*	*	*									
031	*	*	*	*	*	*	*	*	*	*			
032					*		*						
033								*	*				
034	*	*											
035	*		*	*		*	*						

* = Site affected

APPENDIX: 1 SITE OF BURNING SYMPTOMS

PATIENT REFERENCE NUMBER	TIP OF TONGUE	DORSUM OF TONGUE	R LATERAL BORDER OF TONGUE	L LATERAL BORDER OF TONGUE	PALATE	UPPER RIDGE	LOWER RIDGE	UPPER LIP	LOWER LIP	WHOLE MOUTH	THROAT	BUCCAL MUCOSA	FLOOR OF MOUTH
036	*	*	*	*			*						
037	*	*			*						*		
038	*	*	*	*									
039			*	*									
040	*	*	*	*	*			*	*				
041					*			*					
042					*	*							
043	*	*	*	*	*	*	*	*	*				
044					*			*	*				
045	*	*	*	*									
046		*									*		
047	*	*	*	*								*	
048	*	*	*	*	*	*	*						
049									*				
050	*	*	*	*	*	*	*	*	*	*			
051	*	*	*	*	*			*	*				
052	*								*				
053	*	*	*	*	*			*	*				
054	*	*	*	*									
055	*		*	*									
056				*									
057	*	*	*	*									
058	*	*	*	*	*	*	*	*	*	*			
059	*		*	*	*	*	*	*	*				
060	*	*											
061	*	*		*			*		*				
062	*	*	*	*	*	*	*	*	*				
063	*	*	*	*									
064	*	*	*	*	*	*	*						
065					*								
066							*				*		
067				*	*								
068	*	*	*	*									
069	*											*	
070							*						

* = Site affected

APPENDIX: 1 SITE OF BURNING SYMPTOMS

PATIENT REFERENCE NUMBER	TIP OF TONGUE	DORSUM OF TONGUE	R LATERAL BORDER OF TONGUE	L LATERAL BORDER OF TONGUE	PALATE	UPPER RIDGE	LOWER RIDGE	UPPER LIP	LOWER LIP	WHOLE MOUTH	THROAT	BUCCAL MUCOSA	FLOOR OF MOUTH
071		*						*	*		*		
072	*	*	*	*		*	*						
073	*	*	*	*			*						
074	*	*	*	*				*	*				
075						*							
076	*	*			*			*	*				
077		*						*	*				
078	*												
079	*	*	*	*	*	*							
080		*											
081	*	*			*	*							
082	*	*	*	*				*	*				
083					*	*	*	*	*				
084	*	*	*	*								*	
085	*		*	*				*	*				
086	*	*	*	*	*								
087		*							*				
088	*	*	*	*	*	*	*	*	*	*			
089	*	*	*	*	*	*	*						
090	*	*	*	*	*	*	*						
091						*	*						
092	*			*			*		*				
093	*	*	*	*	*	*	*	*	*	*			
094	*	*			*	*		*	*				
095			*										
096	*	*	*	*	*	*	*			*			
097	*				*	*	*						
098	*	*	*	*				*	*				
099				*	*						*		
100	*	*			*								
101	*				*	*							
102	*	*	*	*	*	*	*	*	*	*			
103	*	*	*	*									
104	*	*	*	*									
105					*								

* = Site affected

APPENDIX: 1 SITE OF BURNING SYMPTOMS

PATIENT REFERENCE NUMBER	TIP OF TONGUE	DORSUM OF TONGUE	R. LATERAL BORDER OF TONGUE	L. LATERAL BORDER OF TONGUE	PALATE	UPPER RIDGE	LOWER RIDGE	UPPER LIP	LOWER LIP	WHOLE MOUTH	THROAT	BUCCAL MUCOSA	FLOOR OF MOUTH
106							*						
107	*		*										
108	*		*	*									
109			*										
110						*	*						
111	*		*	*									
112	*	*	*	*									
113									*				
114	*		*	*	*	*	*	*	*				
115						*			*			*	*
116							*						
117	*		*	*			*						
118	*	*	*	*			*						
119	*						*		*				
120	*												
121	*								*	*			
122				*									
123	*	*	*	*	*	*	*	*	*	*			
124	*	*	*	*				*	*				
125	*		*	*									
126	*	*	*	*									
127	*	*	*	*	*	*	*	*	*				
128	*	*	*	*	*	*	*	*	*				
129								*					
130	*				*	*	*	*	*				
131	*	*	*	*							*		
132	*												
133					*	*	*		*				
134	*	*	*	*									
135	*	*	*	*									
136	*		*	*							*		
137	*												
138	*		*	*				*	*				
139	*	*	*	*	*	*	*	*	*	*	*		
140	*		*	*									

* = Site affected

APPENDIX: 1 SITE OF BURNING SYMPTOMS

PATIENT REFERENCE NUMBER	TIP OF TONGUE	DORSUM OF TONGUE	R LATERAL BORDER OF TONGUE	L LATERAL BORDER OF TONGUE	PALATE	UPPER RIDGE	LOWER RIDGE	UPPER LIP	LOWER LIP	WHOLE MOUTH	THROAT	BUCCAL MUCOSA	FLOOR OF MOUTH
141					*	*	*						
142	*	*	*	*	*	*	*	*	*				
143					*	*							
144	*	*	*	*									
145	*				*	*							
146							*	*	*		*	*	*
147	*	*	*	*	*	*	*	*	*	*			
148	*	*	*	*									
149	*	*	*	*							*		
150	*	*											

* = Site affected

APPENDIX: 1 SITE OF BURNING SYMPTOMS

Patients reference number	Burning on wakening	Worse as day progresses	Maximum discomfort at night	Relation to eating and drinking	Normal sleep pattern	Difficulty getting to sleep	Early morning wakening	Burning causes wakening	Burning changed sleep pattern
001	1	1	1	0	0	1	1	0	0
002	0	1	1	0					
003	0	1	1	0	0	1	0	0	0
004	1	1	0	0	1	0	1	0	0
005	1	1	1	W	0	1	1	0	0
006	0	1	1	0	1	0	1	0	0
007	0	1	1	0	0	0	1	1	1
008	0	1	1	W	0	0	1	0	0
009	1	0	0	B/W	0	1	1	0	0
010	1	1	1	W	0	0	1	1	1
011	0	1	1	B/W	0	0	1	0	0
012	0	1	1	W	1	0	0	0	0
013	0	1	1	0	1	0	0	0	0
014	0	1	1	0	0	1	0	0	0
015	1	1	0	0	0	1	1	0	0
016	0	1	0	0	0	0	1	0	0
017	0	0	0	B/W	0	0	1	0	0
018	0	1	1	0					
019	1	0	0	B	1	0	0	0	0
020	0	1	1	W	0	1	1	0	0

1 = Symptom present
0 = Symptom absent
B = Burning relieved by eating/drinking
W = Burning made worse by eating/drinking
B/W = Burning relieved by some foods/drink
Made worse by others

APPENDIX: 1 SYMPTOM AND SLEEP PATTERNS

Patients reference number	Burning on wakening	Worse as day progresses	Maximum discomfort at night	Relation to eating and drinking	Normal sleep pattern	Difficulty getting to sleep	Early morning wakening	Burning causes wakening	Burning changed sleep pattern
021	0	1	1	W	1	0	0	0	0
022	0	1	1	W	0	1	1	0	0
023	1	1	1	B	0	1	1	1	1
024	0	1	1	0	0	1	0	0	1
025	1	0	0	0	1	1	1	0	0
026	1	0	0	B	0	1	0	1	1
027	1	0	0	W	0	1	1	1	1
028	0	1	1	B	1	0	0	0	0
029	1	1	0	W	1	0	0	0	0
030	1	0	0	0	0	1	1	0	0
031	0	0	0	B	0	1	0	0	0
032	0	0	0	W					
033	0	0	0	W	1	0	0	0	0
034	1	0	1	W	0	1	0	1	1
035	0	1	1	0	1	0	0	0	0
036	1	1	0	W	0	1	0	0	0
037	1	0	0	W	1	0	0	0	0
038	1	0	0	0	0	1	1		
039	0	1	0	0	0	0	0	0	
040	1	0	0	0					

1 = Symptom present
0 = Symptom absent
B = Burning relieved by eating/drinking
W = Burning made worse by eating/drinking
B/W = Burning relieved by some foods/drink
Made worse by others

APPENDIX: 1 SYMPTOM AND SLEEP PATTERNS

Patients reference number	Burning on wakening	Worse as day progresses	Maximum discomfort at night	Relation to eating and drinking	Normal sleep pattern	Difficulty getting to sleep	Early morning wakening	Burning causes wakening	Burning changed sleep pattern
041	0	1		W				0	
042	0	1	1	0	1	0	0	0	0
043	1	1		0	0	1		0	
044					1	0	0	0	0
045	0	1	1	0	1	0	1	0	0
046	1	0	0	B					
047	1	0	0	W	1	0	0	0	0
048	1	1	1	W	0	1	1	0	0
049	1	0	0	0	1	0	0	0	0
050	0	1	1	0	0	1	1	0	0
051									
052	0	1	1	0	0	1	1	0	
053	0	1	1	0	0	1	0	0	0
054	0	1	1	W	1	0	0	0	0
055	1	0	0	W	0	0	1	1	0
056	1	0	0	0	1	0	0	0	0
057	0	0	1	W	1	0	0	0	0
058	0	1	0	W	1	1	0	0	0
059	1	1	1	0	0	1	1	0	0
060	1	0	1	W	1	0	1	0	0

1 = Symptom present
0 = Symptom absent
B = Burning relieved by eating/drinking
W = Burning made worse by eating/drinking
B/W = Burning relieved by some foods/drink
Made worse by others

APPENDIX: 1 SYMPTOM AND SLEEP PATTERNS

Patients reference number	Burning on wakening	Worse as day progresses	Maximum discomfort at night	Relation to eating and drinking	Normal sleep pattern	Difficulty getting to sleep	Early morning wakening	Burning causes wakening	Burning changed sleep pattern
061	0	0	0	W	1	0	1	0	0
062	0	1	0	W	0	1	0	1	1
063	0	1	1	B	0	1	1	1	1
064	1	1	1	W	0	1	1	0	0
065	1	1	0	0	0	1	1	0	0
066	0	1	0	0	1	0	0	0	0
067	1	0	0	0					
068									
069	0	1	1	B	1	0	0	0	0
070	0	1	1	0				1	1
071	0	1	1	0	1	0	1	0	0
072	1	1	1	W	0	1	1	1	1
073	1	1	1	W	0	1	1	1	1
074	1	0	0	0	0	1	1	1	1
075	0	1	1	0					
076	0	1	0	W	0	1	1	0	0
077	0	1	0	W	1	0	0	0	0
078	0	1	1	0	0	1	0	0	
079	1	0	0	B/W	1	0	0	0	0
080	0	1	1	W	1	0	0	0	0

1 = Symptom present
0 = Symptom absent
B = Burning relieved by eating/drinking
W = Burning made worse by eating/drinking
B/W = Burning relieved by some foods/drink
Made worse by others

APPENDIX: 1 SYMPTOM AND SLEEP PATTERNS

Patients reference number	Burning on wakening	Worse as day progresses	Maximum discomfort at night	Relation to eating and drinking	Normal sleep pattern	Difficulty getting to sleep	Early morning wakening	Burning causes wakening	Burning changed sleep pattern
081	1	0	0	W	1	0	0	1	0
082	0	1	1	0	1	0	0	0	0
083	1	0	0	B					
084	0	1	1	0	0	1	1	1	1
085	0	1	1	B	1	0	0	0	0
086	1	1	0	W	1	0	0	0	0
087	1	0	0	0	0	0			
088	1	1	1	W	0	1	1	1	0
089	1	0	0	0	1	0	0	1	0
090	1	1	1	W	1	0	0	0	1
091	0	1	0	0	1	0	0	0	0
092	1	0	0	W	0	1	1	1	0
093	0	1	1	0	1	0	0	0	0
094	1	1	0	B	0	0	1	0	1
095	1	0	0	W	1	0	0	0	1
096	0	1	1	0	1	0	0	0	0
097	0	1	1	B					
098	1	0	0	0	0	1	1	0	0
099	1	1	0	W	1	0	1	0	0
100	0	1	0	0	0	1	1	0	0

1 = Symptom present
0 = Symptom absent
B = Burning relieved by eating/drinking
W = Burning made worse by eating/drinking
B/W = Burning relieved by some foods/drink
Made worse by others

APPENDIX: 1 SYMPTOM AND SLEEP PATTERNS

Patients reference number	Burning on wakening	Worse as day progresses	Maximum discomfort at night	Relation to eating and drinking	Normal sleep pattern	Difficulty getting to sleep	Early morning wakening	Burning causes wakening	Burning changed sleep pattern
101	1	0	0	0	1	0	0	0	0
102	1	1	1	0	0	1	1	1	1
103	0	1	1	0	1	0	0	1	0
104	0	1	1	W	0	1	1	0	1
105	1	1	0	0	0	1	1	0	0
106	0	1	1	0	1	0	0	0	0
107	0	0	0	W	1	1	0	0	0
108	1	1	1	0	1	0	0	0	0
109	1	1	0	B					
110	0	0	1	0	1	0	0	0	0
111	0	1	1	0	1	0	0	0	0
112	1	0	0	0	1	0	0	0	0
113	1	0	0	0	1	1	0	0	0
114	0	1	0	0	1	1	0	0	0
115	0	1	1	0	1	0	1	0	0
116	0	0	0	W	0	1	0	1	1
117	1	1	1	W	1	0	0	0	0
118	0	1	1	0	0	1		1	1
119	1	1	1	0	1	1	0	1	1
120	1	0	0	0	1	0	0	1	1

1 = Symptom present
0 = Symptom absent
B = Burning relieved by eating/drinking
W = Burning made worse by eating/drinking
B/W = Burning relieved by some foods/drink
Made worse by others

APPENDIX: 1 SYMPTOM AND SLEEP PATTERNS

Patients reference number	Burning on wakening	Worse as day progresses	Maximum discomfort at night	Relation to eating and drinking	Normal sleep pattern	Difficulty getting to sleep	Early morning wakening	Burning causes wakening	Burning changed sleep pattern
121	1	1	0	0	0	1	1	1	0
122	0	1	1	B/W	0	1	1	0	0
123	1	1	1	B	0	0	1	0	0
124	0	0	0	0	1	0	0	0	0
125	1	1	0	0	1	0	0	0	0
126	0	1	1	B	1	0	0	0	0
127	1	1	1	0	0	1	0	0	0
128	0	1	1	B	0	1		1	1
129	0	1	0	0	1	1	1	0	0
130	0	1	1	0	1	0	0	0	0
131	0	1	1	B/W					
132	0	1	1	W	0	1	0	0	1
133	0	1	1	0	0	1	1	1	
134	0	1	1	B	1	0	0	0	0
135	1	0	0	0					
136	0	1	1	B/W	1	0	0	1	0
137	0	0	0	B	1	1	0	0	0
138	0	1	1	W	0	1	0	0	0
139	1	0	0	0	0	1	0	1	1
140	0	1	1	B	0	1	1	0	0

1 = Symptom present
0 = Symptom absent
B = Burning relieved by eating/drinking
W = Burning made worse by eating/drinking
B/W = Burning relieved by some foods/drink
Made worse by others

APPENDIX: 1 SYMPTOM AND SLEEP PATTERNS

Patients reference number	Burning on wakening	Worse as day progresses	Maximum discomfort at night	Relation to eating and drinking	Normal sleep pattern	Difficulty getting to sleep	Early morning wakening	Burning causes wakening	Burning changed sleep pattern
141	0	1	1	0	1	0	0	0	0
142	0	1	1	W	1	0	1	0	0
143	0	1	1	0	0	1	1	0	1
144	0	1	1	B					
145	0	1	1	B/W	0	1	1	0	1
146	0	1	1	W	0	1	1	0	0
147	1	1	1	B	0	1	0	1	1
148	0	1	1	W	1	0	0	0	0
149	1	1	1	W	0	1	1	1	1
150	0	1	0	0	1	0	0	0	0

1 = Symptom present
0 = Symptom absent
B = Burning relieved by eating/drinking
W = Burning made worse by eating/drinking
B/W = Burning relieved by some foods/drink
Made worse by others

Patient Reference Number	Hgb	MCV	Serum Ferritin	Serum Vitamin B12	Corrected Whole Blood Folate
(Normal ranges)	male 13-18 female 11.5-16.5 g/dl	78-99 fl	12-300 ng/ml	175-800pg/ml	75-400mg/ml
001	14.4	94	129	425	272
002	12.5	90	18	438	149
003	12.2	93	39	195	167
004	14.9	88	14	285	150
005	13.2	90	48	194	303
006	15.1	94	142	389	351
007	12.6	96	30	253	210
008	15.5	94	46	187	122
009	11.8	94	18	232	151
010	13.9	94	105	615	252
011	14.5	84	63	343	249
012	14.5	86	60	382	105
013	12.8	92	102	621	260
014	11.9	92	66	542	140
015	14.5	89	169	>1000	243
016	13.2	97	75	322	200
017	14.6	92	153	326	149
018	15.5	93	22	326	206
019	14.3	83		298	234
020	11.8	94	193	105	127

Appendix: 1 Haematological data

Patient Reference Number	Hgb	MCV	Serum Ferritin	Serum Vitamin B12	Corrected Whole Blood Folate
(Normal ranges)	male 13-18 female 11.5-16.5 g/dl	78-99 fl	12-300 ng/ml	175-800pg/ml	75-400mg/ml
021	13.5	89	75	290	320
022	14.1	92	155	259	342
023	12.8	89	30	182	271
024	17.0	93		319	134
025	13.9	98	122	234	
026	13.7	95	153	223	195
027	14.5	94	65	328	133
028	12.8	91	304	673	241
029	14.7	95	259	184	91
030	14.5	89	91	574	167
031	12.6	96	46	529	135
032	10.2	85	03	216	130
033	14.7	90	47	261	166
034	14.4	89	44		
035	15.5	90	72	459	223
036	13.4	98	68	254	84
037	14.2	86	115	156	210
038	16.7	98	84	282	447
039	13.7	83	123	346	263
040	14.9	85	48	221	577

Appendix: 1 Haematological data

Patient Reference Number	Hgb	MCV	Serum Ferritin	Serum Vitamin B12	Corrected Whole Blood Folate
(Normal ranges)	male 13-18 female 11.5-16.5 g/dl	78-99 fl	12-300 ng/ml	175-800pg/ml	75-400mg/ml
041	15.9	86	69	360	121
042	13.8	93	153	331	368
043	13.9	89	347	346	152
044					
045	13.4	90	126	268	289
046	14.8	90	109	312	>600
047	13.9	86	5	474	184
048	14.7	97	114	218	215
049	13.8	88	28	340	165
050	12.3	100	162	188	92
051	14.9	93	34	390	195
052	14.3	91	52	430	153
053	12.9	87	58	254	194
054	12.9	89	36	420	263
055	15.0	87	58	213	191
056	15.1	97	92	162	256
057	13.7	92	159	124	287
058	12.4	92	193	548	271
059	13.7	91	165	471	297
060	12.8	87	47	273	193

Appendix: 1 Haematological data

Patient Reference Number	Hgb	MCV	Serum Ferritin	Serum Vitamin B12	Corrected Whole Blood Folate
(Normal ranges)	male 13-18 female 11.5-16.5 g/dl	78-99 fl	12-300 ng/ml	175-800pg/ml	75-400mg/ml
061	14.6	90	164	372	249
062	12.1	92		221	143
063	13.7	94		1624	282
064	16.1	103	190	474	171
065	13.9	91		344	273
066	13.2	96	143	430	200
067	13.2	95	37	269	334
068	13.1	83	37	350	353
069	15.3	84	408	352	205
070	14.6	92		441	139
071	14.0	86	44	155	155
072	12.7	89	69	461	179
073	13.1	93	166	330	92
074	15.2	89	285	201	140
075	13.4	87	21	223	294
076	13.6	89	19	249	168
077	13.8	89	40	170	151
078	12.8	91	76	266	275
079	16.8	90	28	460	398
080	12.8	89	6	480	128

Appendix: 1 Haematological data

Patient Reference Number	Hgb	MCV	Serum Ferritin	Serum Vitamin B12	Corrected Whole Blood Folate
(Normal ranges)	male 13-18 female 11.5-16.5 g/dl	78-99 fl	12-300 ng/ml	175-800pg/ml	75-400mg/ml
081	16.5	95	251	475	315
082	12.1	89	29	133	111
083	12.9	103	37	369	370
084	12.9	92	30	233	173
085	15.2	91	120	336	165
086	14.1	97	261	267	351
087	14.0	91	273	374	170
088	13.8	92	325	252	
089	15.7	98	467	393	288
090	13.3	91		406	128
091	15.0	98	378	317	233
092	13.3	85	11	91	193
093	14.4	93	130	294	288
094	14.3	86	45	385	117
095	12.9	97	62	371	315
096			14	359	152
097	13.6	99	26	>2000	210
098	13.7	96		237	177
099	12.5	87	38	177	127
100	13.0	96	80	210	231

Appendix: 1 Haematological data

Patient Reference Number	Hgb	MCV	Serum Ferritin	Serum Vitamin B12	Corrected Whole Blood Folate
(Normal ranges)	male 13-18 female 11.5-16.5 g/dl	78-99 fl	12-300 ng/ml	175-800pg/ml	75-400mg/ml
101	12.5	89	14	327	233
102	13.9	93	37	297	204
103	13.9	88	122	215	100
104	14.5	97	117	538	141
105	14.9	100	94	280	168
106	13.8	90	44	367	167
107	14.0	93	3	273	203
108	15.1	95	234	391	159
109	13.9	94	15	211	270
110	15.6	87	163	503	370
111	13.6	95	38	294	140
112	15.5	90	85	569	193
113	16.2	87	12	332	223
114	14.6	99	48	288	144
115	13.3	90		384	278
116	13.9	91	108	432	112
117	14.8	90		455	101
118	13.4	93	134	324	155
119	14.1	85	75	297	373
120	16.4	87	188	599	163

Appendix: 1 Haematological data

Patient Reference Number	Hgb	MCV	Serum Ferritin	Serum Vitamin B12	Corrected Whole Blood Folate
(Normal ranges)	male 13-18 female 11.5-16.5 g/dl	78-99 fl	12-300 ng/ml	175-800pg/ml	75-400mg/ml
121	13.2	88	100	714	205
122	15.1	89	92	365	200
123	11.5	92	20	338	165
124	13.7	90	52	136	122
125	13.4	88	49	1865	367
126	13.4	102	96	230	189
127					
128	15.6	89	146	282	135
129	15.8	96	123	197	114
130	16.0	93	100	>2000	168
131	13.2	86	10	71	167
132	14.1	96	23	301	91
133	13.4	87	29	158	145
134	13.2	94	101	273	159
135					
136	12.8	98	120	384	651
137	14.2	95	82	413	191
138	14.9	88	39.5	594	161
139	13.1	86	93	290	174
140	13.9	90	157	409	148

Appendix: 1 Haematological data

Patient Reference Number	Hgb	MCV	Serum Ferritin	Serum Vitamin B12	Corrected Whole Blood Folate
(Normal ranges)	male 13-18 female 11.5-16.5 g/dl	78-99 fl	12-300 ng/ml	175-800pg/ml	75-400mg/ml
141	13.9	89	118	270	224
142	14.2	93	254	404	305
143	13.8	87	72	497	171
144	15.1	94	149	335	247
145	11.6	93	153	196	193
146	13.4	92	68	129	252
147	13.1	86	4	242	133
148	13.5	91	144	287	184
149	13.3	98	130	>2000*	278
150	16.0	95	489	479	264

Appendix: 1 Haematological data

PATIENT REFERENCE NUMBER	FASTING PLASMA GLUCOSE CONCENTRATIONS mmol/L
001	5.1
002	5.2
003	4.4
004	4.4
005	4.9
006	4.9
007	4.9
008	
009	5.2
010	5.3
011	5.8
012	4.2
013	5.3
014	5.1
015	6.7
016	4.8
017	4.8
018	
019	5.1
020	5.1
021	4.5
022	4.1
023	4.3
024	
025	4.8
026	5.1
027	4.7
028	14.2
029	5.5
030	5.3
031	7.3
032	5.0
033	5.1
034	
035	4.6
036	4.8
037	6.5
038	6.2
039	5.7
040	5.9
041	
042	4.7
043	5.8
044	
045	

APPENDIX: 1
FASTING PLASMA GLUCOSE CONCENTRATIONS

PATIENT REFERENCE NUMBER	FASTING PLASMA GLUCOSE CONCENTRATIONS mmol/L
046	
047	4.9
048	5.2
049	4.7
050	4.3
051	
052	4.4
053	5.5
054	5.3
055	
056	4.0
057	
058	4.8
059	4.9
060	4.8
061	4.5
062	5.5
063	4.6
064	4.4
065	5.6
066	6.5
067	5.1
068	
069	4.8
070	
071	5.9
072	5.2
073	
074	5.4
075	4.7
076	4.6
077	3.9
078	4.6
079	5.4
080	4.5
081	17.7
082	4.1
083	
084	
085	4.9
086	4.8
087	4.9
088	5.3
089	11.1
090	5.0

APPENDIX: 1
FASTING PLASMA GLUCOSE CONCENTRATIONS

PATIENT REFERENCE NUMBER	FASTING PLASMA GLUCOSE CONCENTRATIONS mmol/L
091	11.8
092	
093	5.1
094	4.7
095	5.0
096	5.4
097	
098	4.7
099	4.8
100	4.4
101	4.7
102	4.0
103	
104	5.6
105	6.1
106	4.1
107	4.1
108	5.8
109	4.6
110	5.6
111	5.3
112	5.3
113	5.0
114	5.0
115	4.8
116	
117	4.6
118	5.2
119	7.1
120	5.1
121	5.5
122	4.2
123	4.7
124	4.8
125	4.2
126	5.6
127	
128	5.6
129	5.4
130	5.1
131	5.2
132	4.6
133	4.5
134	5.2
135	

APPENDIX: 1
FASTING PLASMA GLUCOSE CONCENTRATIONS

PATIENT REFERENCE NUMBER	FASTING PLASMA GLUCOSE CONCENTRATIONS mmol/L
136	4.5
137	4.9
138	4.6
139	5.4
140	7.7
141	5.4
142	14.7
143	4.1
144	4.4
145	4.5
146	5.1
147	4.6
148	4.9
149	4.4
150	3.8

APPENDIX: 1
FASTING PLASMA GLUCOSE CONCENTRATIONS

PATIENT REFERENCE NUMBER	RIGHT PAROTID FLOW RATE ml/min	LEFT PAROTID FLOW RATE ml/min
001	0.5	0.5
002		
003	2.5	2.5
004	0.4	0.5
005	0.6	0.5
006	1.0	0.9
007	0.5	0.5
008		
009	1.5	2.0
010		
011	1.1	1.1
012	1.2	1.0
013	0.9	1.3
014	0.9	1.0
015	1.2	1.2
016	>0.7	>0.7
017	0.2	0.7
018	>0.7	>0.7
019	0.1	0.2
020	>0.7	>0.7
021	1.1	1.1
022	1.3	1.1
023	1.8	0.9
024	>0.7	>0.7
025	>2.0	>2.0
026	0.4	0
027	0.4	0.2
028		
029	1.5	1.5
030	0.5	0.5
031	0.5	0.6
032	1.2	1.6
033		
034		
035	0.9	0.8
036	>2.0	>2.0
037	1.6	1.6
038		
039		
040	2.5	2.5
041	2.0	2.0
042	0.8	0.6
043	1.5	1.5
044		
045	1.4	0.8

APPENDIX: 1
STIMULATED PAROTID FLOW RATES

PATIENT REFERENCE NUMBER	RIGHT PAROTID FLOW RATE ml/min	LEFT PAROTID FLOW RATE ml/min
046	1.2	1.2
047		
048	0.7	0.4
049	0.5	0.4
050	0.5	0.5
051	>2.0	>2.0
052	0.6	0.7
053		
054		
055	0.6	0.8
056	1.4	1.5
057		
058	0.7	0.7
059	0.5	0.5
060		
061	1.4	1.5
062	0.9	0.9
063	1.8	1.6
064	1.25	1.25
065	>0.7	>0.7
066	0.8	0.8
067		
068		
069	2.0	2.0
070		
071	0.7	1.3
072	>0.7	>0.7
073	0.3	0.2
074		
075		
076		
077	1.5	1.5
078	>0.7	>0.7
079	1.6	1.4
080	>2.0	>2.0
081	0.7	0.9
082	1.7	1.2
083		
084		
085	1.2	0.9
086	0.5	0.5
087	<0.2	<0.2
088	2.5	2.5
089	>2.0	>2.0
090	1.5	1.5

APPENDIX: 1
STIMULATED PAROTID FLOW RATES

PATIENT REFERENCE NUMBER	RIGHT PAROTID FLOW RATE ml/min	LEFT PAROTID FLOW RATE ml/min
091	1.2	1.2
092	0.8	1.2
093	0.4	0.3
094	1.0	0.8
095	1.2	0.8
096	0.5	0.6
097		
098	1.0	1.0
099	0.9	0.8
100	0.5	2.0
101		
102	0.4	0
103		
104	0.7	0.7
105	1.7	2.4
106	0.7	0.7
107	0.9	1.2
108	0.8	0.8
109		
110	1.3	1.2
111	1.0	1.0
112	0.9	1.0
113		
114		
115		
116		
117	1.6	1.1
118	>2.0	>2.0
119	1.7	1.0
120	1.4	0.8
121	0.2	0
122	0.8	1.0
123	0.8	0.8
124	3.0	2.5
125	1.2	1.2
126		
127	>0.7	>0.7
128	0.5	0.5
129	1.0	1.2
130	0.2	0.3
131	0.5	0.3
132	>2.0	1.0
133	1.2	1.2
134		
135		

APPENDIX: 1
STIMULATED PAROTID FLOW RATES

PATIENT REFERENCE NUMBER	RIGHT PAROTID FLOW RATE ml/min	LEFT PAROTID FLOW RATE ml/min
136	<0.7	<0.7
137	1.4	1.0
138	>2.0	>2.0
139	1.5	1.8
140	1.1	1.1
141	0.5	0.9
142		
143		
144	1.1	1.1
145	1.2	1.6
146		
147	0.5	1.0
148	1.4	1.4
149	0.8	0.8
150	0.8	1.0

APPENDIX: 1
STIMULATED PAROTID FLOW RATES

EDENTULOUS PATIENTS

PATIENT REFERENCE NUMBER	AGE OF PRESENT DENTURES	CLEARANCE	DENTURE WEARING PATTERN	EFFECT OF DENTURE REMOVAL	ONSET RELATED TO PROVISION DENTURES	DESIGN FAULT	REPLACEMENT DENTURES PROVIDED	EFFECT OF REPLACEMENT DENTURES
005	2-5 years	5-10 years	Day only	Improvement	No	Yes	Yes	Improvement
006	<6 months	10-15 years	Socially	Improvement	Yes	No	Yes	No change
007	<6 months	1-2 years	Socially	Improvement	Yes (time of immediates)	Yes	Yes	No change
008	U 5-10 years L 2-5 years	2-5 years		Improvement	No	Yes	No	
009	1/2-1 year	>30 years	Day only	Improvement	Yes	No	Yes	No change
010	1-2 years	>30 years	Day only	Worse	No	Yes	No	
012	1-2 years	>30 years	Day and night	Does not know	No	Yes	No	
013	2-5 years	>30 years	Day only	U improvement L no change	No	Yes	Yes	No change
014	2-5 years	10-15 years	Socially	Improvement	Yes	Yes	No	
015	5-10 years	15-30 years	Day only	No change	No	No	No	
016	5-10 years	15-30 years	Day only	No change	Yes	Yes	No	
017	5-10 years	>30 years	Never	No change	No	Yes	No	

EDENTULOUS PATIENTS

PATIENT REFERENCE NUMBER	AGE OF PRESENT DENTURES	CLEARANCE	DENTURE WEARING PATTERN	EFFECT OF DENTURE REMOVAL	ONSET RELATED TO PROVISION DENTURES	DESIGN FAULT	REPLACEMENT DENTURES PROVIDED	EFFECT OF REPLACEMENT DENTURES
018					No	No	No	
020	1-2 years	>30 years	Day only	No change	No	No	Yes	No change
022	2-5 years	10-15 years	U Day and night L Socially	L Improvement	Surgery to lower alveolus	No	No	
023	5-10 years	>30 years	Day and night	Improvement	No	Yes	Yes	Improvement
024	2-5 years	>30 years	Day only	Improvement	No	No	No	
026	2-5 years	15-30 years	Day only	No change	No	Yes	No	
027			Never		No	No	No	
028	1/2-1 year	2-5 years	Day only	Improvement	Yes	Yes	Yes	Initial Improvement
029	5-10 years	15-30 years	Day and night	No change	Yes	Yes	Yes	No change
031	15-30 years	15-30 years	Day only	Improvement	No	Yes	Yes	Improvement
032	2-5 years	10-15 years	Day only	Improvement	No	Yes	Yes	No change
033	1-2 years	15-30 years	U Day and night L Day only	No change	No	Yes	Yes	No change

APPENDIX: 1 DENTURE STATUS

EDENTULOUS PATIENTS

PATIENT REFERENCE NUMBER	AGE OF PRESENT DENTURES	CLEARANCE	DENTURE WEARING PATTERN	EFFECT OF DENTURE REMOVAL	ONSET RELATED TO PROVISION DENTURES	DESIGN FAULT	REPLACEMENT DENTURES PROVIDED	EFFECT OF REPLACEMENT DENTURES
034	1/2-1 year	15-30 years	Occasionally	Improvement	Yes	Yes	Yes	Improvement
035	1-2 year	15-30 years	Day and night	Improvement	No	yes	Yes	Improvement
036	1/2-1 year	15-30 years		No change	Yes	Yes	Yes	Improvement
040	5-10 years	15-30 years	U Never L Day only	U Improvement L No change	No	Yes	Yes	No change
043	5-10 years		Day and night	Improvement	No	Yes	No	
046	10-15 years	15-30 years	Day and night	Improvement	No	No	Yes	No change
047	1-2 years	15-30 years	Socially	Improvement	No	No	No	
048	1/2-1 year	1/2-1 year		No change	Yes	Yes	Yes	No change
049	1-2 years	15-30 years	Day and night	No change	No	Yes	No	
052	1-2 years	10-15 years		Worse	No	No	No	
053	5-10 years	>30 years	Day only	Improvement	No	Yes	Yes	No change
055	<6 months	15-30 years	Day only	No change	No	Yes	No	

APPENDIX: 1 DENTURE STATUS

EDENTULOUS PATIENTS

PATIENT REFERENCE NUMBER	AGE OF PRESENT DENTURES	CLEARANCE	DENTURE WEARING PATTERN	EFFECT OF DENTURE REMOVAL	ONSET RELATED TO PROVISION DENTURES	DESIGN FAULT	REPLACEMENT DENTURES PROVIDED	EFFECT OF REPLACEMENT DENTURES
056	15-30 years	15-30 years	Day and night	No change	No	No	No	
057	5-10 years	5-10 years	U Day and night L Day only	No change	No	Yes	No	
058	10-15 years	>30 years	Day and night	Improvement	No	Yes	Yes	No change
059	<6 months	<6 months	U Day only L Socially	No change	No	Yes	Yes	No change
062	1-2 years	1-2 years	Never	No change	No	No	No	
063	1-2 years	>30 years	Day only	Worse	No	Yes	Yes	Improvement
064	15-30 years	15-30 years	Day and night	Improvement	No	No	Yes	No change
065	2-5 years	>30 years	Day only	U Improvement L No change	No	No	No	
066	2-5 years	>30 years	U Day and night L Only for meals	No change	No	Yes	No	
068					No	No	No	
069	1/2-1 year	15-30 years	Day only	No change	No	Yes	Yes	Improvement
070				U No change L Totally alleviated	No	Yes	Yes	No change

APPENDIX: 1 DENTURE STATUS

EDENTULOUS PATIENTS

PATIENT REFERENCE NUMBER	AGE OF PRESENT DENTURES	CLEARANCE	DENTURE WEARING PATTERN	EFFECT OF DENTURE REMOVAL	ONSET RELATED TO PROVISION DENTURES	DESIGN FAULT	REPLACEMENT DENTURES PROVIDED	EFFECT OF REPLACEMENT DENTURES
071	<6 months	15-30 years	Day only	U Improvement L No change	No	Yes	No	
072	2-5 years	5-10 years	Never		No	No	No	
073	5-10 years	15-30 years	Day only	Improvement	No	Yes	Yes	Improvement
074	5-10 years	>30 years	Day and night	No change	No	No	No	
077	2-5 years	15-30 years	Day and night	Improvement	No	Yes	Yes	Improvement
078	5-10 years	15-30 years	Day and night	No change	No	Yes	Yes	Initial Improvement
081	5-10 years	15-30 years	U Day and night L Day only	No change	No	No	No	
082	10-15 years	15-30 years	Day and night	Improvement	When dentures relined	Yes	No	
083	10-15 years			No change	No	No	No	
085	U 10-15 years L 15-30 years	>30 years	Day and night	Improvement	No	Yes	Yes	No change
087	5-10 years		Day only		No	No	Yes	Improvement
088	1/2-1 year	2-5 years	Never	No change	No	No	No	

APPENDIX: 1 DENTURE STATUS

EDENTULOUS PATIENTS

PATIENT REFERENCE NUMBER	AGE OF PRESENT DENTURES	CLEARANCE	DENTURE WEARING PATTERN	EFFECT OF DENTURE REMOVAL	ONSET RELATED TO PROVISION DENTURES	DESIGN FAULT	REPLACEMENT DENTURES PROVIDED	EFFECT OF REPLACEMENT DENTURES
089	<6 months	15-30 years	Day and night	No change	No	Yes	Yes	Improvement
090	1/2-1 year	15-30 years	U Day only L Socially	Improvement	No	Yes	Yes	Improvement
091	<6 months	1-2 years	Only for meals	Improvement	Yes with immediate dentures	Yes	No	
092	5-10 years	5-10 years	Occasionally	Improvement	No	No	Yes	No change
093	10-15 years	>30 years	Day only	Improvement	No	Yes	No	
094	1-2 years	1-2 years	U Day and night L Day only	Improvement	No	Yes	Yes	No change
097	1/2-1 year	1/2-1 year		Improvement	No	No	No	
100	1-2 years	15-30 years	U Day and night L Never	Improvement	Yes	No	Yes	No change
103	5-10 years	10-15 years	Day only	Improvement	No	Yes	Yes	Improvement
104	2-5 years	>30 years	U Day and night L Only for meals	No change	No	Yes	Yes	No change
106	1-2 years	10-15 years	Day and night	Improvement	Yes	Yes	Yes	Improvement
111	2-5 years	10-15 years	Day only	Totally alleviated	No	Yes	Yes	No change

EDENTULOUS PATIENTS

PATIENT REFERENCE NUMBER	AGE OF PRESENT DENTURES	CLEARANCE	DENTURE WEARING PATTERN	EFFECT OF DENTURE REMOVAL	ONSET RELATED TO PROVISION DENTURES	DESIGN FAULT	REPLACEMENT DENTURES PROVIDED	EFFECT OF REPLACEMENT DENTURES
114	2-5 years	15-30 years	Day only	Improvement	yes	No	Yes	Improvement
116	2-5 years		Day only	No change	No	No	No	
119	1-2 years		Day and night	No change	Yes extraction $\overline{/8}$	Yes	No	
120	1/2-1 year	>30 years	Day only	No change	No	No	No	
121	1/2-1 year	15-30 years	Day only	No change	No	No	Yes	Improvement
123	1-2 years	>30 years	Day only	No change	No	No	No	
125		10-15 years	Day and night	No change	No	No	No	
126		5-10 years	Day and night	No change	No	No	Yes	No change
127	2-5 years	10-15 years	Day only	Improvement	Yes	Yes	Yes	No change
128	2-5 years	>30 years	Day only	Worse	No	No	No	
129	5-10 years	>30 years	Day and night	Improvement	Yes	Yes	No	
130	2-5 years	15-30 years	Socially	Totally alleviated	No	Yes	Yes	Improvement

APPENDIX: 1 DENTURE STATUS

EDENTULOUS PATIENTS

PATIENT REFERENCE NUMBER	AGE OF PRESENT DENTURES	CLEARANCE	DENTURE HEARING PATTERN	EFFECT OF DENTURE REMOVAL	ONSET RELATED TO PROVISION DENTURES	DESIGN FAULT	REPLACEMENT DENTURES PROVIDED	EFFECT OF REPLACEMENT DENTURES
131					No	No	No	
132	<6 months	>30 years	U Day and night L Day only	U No change L Improvement	No	No	No	
133	<6 months	>30 years		Improvement	Yes	Yes	Yes	Improvement
134	1/2-1 year	15-30 years	Occasionally	Improvement	Yes	Yes	Yes	Improvement
137	5-10 years	15-30 years	Day and night	No change	No	No	No	
140	>30 years	>30 years	U Occasionally L Never	Improvement	No	No	No	
141	1-2 years	15-30 years	Socially	Improvement	No	Yes	Yes	Initial Improvement
144	1/2-1 year	15-30 years		Improvement	No	No	No	
145	1/2-1 year	15-30 years	U Day only L Socially	No change	No	No	No	
147	U 1-2 years L 1/2-1 year	1-2 years	U Day and night L Day only	U No change L Improvement	Yes	No	No	
148	U 5-10 years L 1/2-1 year	>30 years	Day only	Improvement	No	No	No	

APPENDIX: 1 DENTURE STATUS

PARTIALLY DENTATE DENTURE WEARERS

PATIENT REFERENCE NUMBER	TYPE OF DENTURE	AGE OF PRESENT DENTURES	DENTURE WEARING PATTERN	EFFECT OF DENTURE REMOVAL	ONSET RELATED TO PROVISION OF PRESENT DENTURES	DESIGN FAULT	REPLACEMENT DENTURES PROVIDED	EFFECT OF REPLACEMENT DENTURES
001	PU		Day only		No	No	No	
002	PU		Day only		No	Yes	Yes	Improvement
003	PU/PL	1-2 years	Day and night	Improvement	No	No	Yes	Improvement
004	PU/PL		Not clear	Improvement	Yes	No	Yes	Initial Improvement
011	PU	<6 months	Day and night	Improvement	No	No	No	
019	PU				Yes	No	Yes	No change
021	PU	1/2 - 1 year	Never	Improvement	No	No	No	
025	PU				Yes	Yes	Yes	No change
041	CU/PL	1-2 years		U Improvement L Improvement	No	Yes	Yes	No change
044	PU		Never	Totally Alleviated	Yes	No	No	
045	PU Cobalt Chromium		Day only	No change	No	No	No	
050	PU	1-2 years	Occasionally	Worse	No	No	Yes	Improvement

APPENDIX: 1 DENTURE STATUS

PARTIALLY DENTATE DENTURE WEARERS

PATIENT REFERENCE NUMBER	TYPE OF DENTURE	AGE OF PRESENT DENTURES	DENTURE WEARING PATTERN	EFFECT OF DENTURE REMOVAL	ONSET RELATED TO PROVISION OF PRESENT DENTURES	DESIGN FAULT	REPLACEMENT DENTURES PROVIDED	EFFECT OF REPLACEMENT DENTURES
051	PU	2-5 years	Day only	Improvement	Yes	No	No	
054	PU	1-2 years	Day and night	No change	No	No	No	
075	PU/CL	2-5 years 5-10 years	U meals only L day only	Improvement	No	No	No	
076	CU/PL	1-2 years	Day only	No change	No	No	Yes	Improvement
080	PU	1-2 years	Day only	Improvement	No	No	No	
098	PU	2-5 years Cobalt Chromium	Day only	Improvement	No	No	No	
099	PU	Cobalt Chromium		Worse	RCT 5	No	Yes	Improvement
101	PU	2-5 years	Day and night	Improvement	No	No	No	
102	PU/PL	1-2 years	Day only	No change	No	No	No	
108	CU	5-10 years	Day and night	Improvement	No	Yes	Yes	Improvement
110	PU	>30 years	Day only	No change	No	No	Yes	Improvement
115	CU	2-5 years	Day only	No change	Yes	Yes	Yes	Improvement

APPENDIX: 1 DENTURE STATUS

PARTIALLY DENTATE DENTURE WEARERS

PATIENT REFERENCE NUMBER	TYPE OF DENTURE	AGE OF PRESENT DENTURES	DENTURE WEARING PATTERN	EFFECT OF DENTURE REMOVAL	ONSET RELATED TO PROVISION OF PRESENT DENTURES	DESIGN FAULT	REPLACEMENT DENTURES PROVIDED	EFFECT OF REPLACEMENT DENTURES
117	CL		Day and night		Yes Lower Clearance	No	Yes	Improvement
118	CU	<6 months	Day and night	Improvement	Yes	Yes	Yes	Improvement
122	CU	2-5 years	Day and night	No change	No	No	No	
124	PU/PL		Never	No change	No	No	No	
135	CU/PL	15-30 years	U Day and night L Never	U Improvement	No	Yes	Yes	No change
136	PU/CL	1/2 -1 year	U Never L Day time	No change	No	No	No	
142	CU/PL			No change	No	No	No	
143	CU	1-2 years	Day only	Improvement	Yes Upper Clearance	Yes	Yes	No change
146	PU/PL	U 2-5 years L <6 months	Day and night	No change	No	No	No	
150	CU	5-10 years	Day only	No change	No	No	No	

APPENDIX: 1 DENTURE STATUS

APPENDIX 2

BURNING MOUTH STUDY

NAME (Surname first)

HOSPITAL NUMBER ☐☐☐☐☐☐

DATE OF BIRTH Y Y M M D D ☐☐☐☐☐☐

SEX

SYMPTOM SITE 1 = YES 0 = NO

Tongue Tip ☐
Dorsum ☐
R Lat ☐
L Lat ☐
Palate ☐
Upper Ridge ☐
Lower Ridge ☐
Upper Lip ☐
Lower Lip ☐

DURATION OF SYMPTOMS

< 6 months	= 1	5 - 10 years	= 5
6 months to 1 year	= 2	10 - 15 years	= 6
1 - 2 years	= 3	15 - 30 years	= 7
2 - 5 years	= 4	> 30 years	= 8

☐

SEVERITY (0 - 10) ☐☐

SYMPTOMS ON WAKING YES = 1 NO = 0

WORSE AS DAY PROGRESSED " " " " ☐

MAX DISCOMFORT AT NIGHT " " " " ☐

RELATED TO EATING YES = 1 NO = 0 SOMETIMES = 2 ☐

CLIMACTERIC SYMPTOMS

FLUSHING YES = 1 NO = 0 ☐

SWEATING " " " " ☐

I.M.P. " " " " ☐

MEDICAL HISTORY

DIABETES	YES	=	1	NO	=	0	☐
DEPRESSIVE ILLNESS	"		"	"		"	☐
SELF	"		"	"		"	☐
FAMILY	"		"	"		"	☐

DRUG HISTORY

TRANQUILLISERS	"		"	"		"	☐
SLEEPING TABLETS	"		"	"		"	☐
ANTI DEPRESSANTS	"		"	"		"	☐
HYPOGLYCAEMICS	"		"	"		"	☐
OTHERS	"		"	"		"	☐
IF PRESCRIBED ARE THEY TAKEN	"		"	"		"	☐

SLEEP PATTERN

NORMAL	"		"	"		"	☐
DIFFICULTY	"		"	"		"	☐
EARLY MORNING WAKENING	"		"	"		"	☐
DOES BURNING CAUSE WAKENING	"		"	"		"	☐
HAS BURNING CHANGED SLEEP PATTERN	"		"	"		"	☐

IF YES HOW LONG

(SCALE AS FOR DURATION OF SYMPTOMS) ☐

SMOKING

SMOKE	"		"	"		"	☐
CIGARETTES/CIGARS	< 10	= 1					
	10 - 20	= 2					☐
	> 20	= 3					

OCCUPATION

RETIRED	YES	=	1	NO	=	0	P/T	=	2	☐
UNEMPLOYED	"		"	"		"				☐
DURATION OF UNEMPLOYMENT	YEARS									☐☐

HOME CIRCUMSTANCES

OWN HOME = 1

RENTED = 2

RELATIVE = 3

OTHER = 4

SOCIAL CIRCUMSTANCES SCALE 0 TO 10

FINDINGS ON EXAMINATION

ORAL MUCOSA (AFFECTED SITE)
NORMAL = 1 ATROPHIC = 2 ERYTHEMATOUS = 3

SALIVA INADEQUATE IN PATIENT'S OPINION YES = 1 NO = 0

IF YES HOW LONG :

< 6 MONTHS	= 1	5 - 10 YRS	= 5
6 MONTHS TO 1 YR	= 2	10 - 15 YRS	= 6
1 - 2 YRS	= 3	15 - 30 YRS	= 7
2 - 5 YRS	= 4	> 30 YRS	= 8

ORAL RINSE YES = 1 NO = 0

ISOLATED YES = 1 NO = 0

TREATED YES = 1 NO = 0

RESPONSE NO CHANGE = 1

IMPROVED = 2

CURED = 3

WORSE = 4

FLOW RATE < 0.5 ml = 1
> 0.5 ml = 2

LABIAL SALIVARY GLAND

HISTOLOGY PATH. NUMBER

REPORT NORMAL = 0

NON SPECIFIC = 1

CONNECTIVE TISSUE DISORDER = 2

SIALOGRAPHY

NUMBER

REPORT NORMAL = 0

NON SPECIFIC = 1

CONNECTIVE TISSUE DISORDER = 2

HAEMATOLOGY

Hb
FILM
MCV
FERRITIN
GLUCOSE
B12
FOLATE
VIT. PROFILE NORMAL = 0
ABNORMAL = 1
ANF
Rh FACTOR

SPECIFIC DEFICIENCY

RESPONSE TO TREATMENT NO CHANGE = 1

IMPROVED = 2

CURED = 3

WORSE = 4

OTHER INVESTIGATIONS

DENTURES

EDENTULOUS PATIENTS

Age of present upper denture

< 6 months	= 1	5 - 10 yrs	= 5
6 months to 1 yr	= 2	10 - 15 yrs	= 6
1 - 2 yrs	= 3	15 - 30 yrs	= 7
2 - 5 yrs	= 4	> 30 yrs	= 8

Age of present lower denture

< 6 months	= 1	5 - 10 yrs	= 5
6 months to 1 yr	= 2	10 - 15 yrs	= 6
1 - 2 yrs	= 3	15 - 30 yrs	= 7
2 - 5 yrs	= 4	> 30 yrs	= 8

Time since total dental clearance

< 6 months	= 1	5 - 10 yrs	= 5
6 months to 1 yr	= 2	10 - 15 yrs	= 6
1 - 2 yrs	= 3	15 - 30 yrs	= 7
2 - 5 yrs	= 4	> 30 yrs	= 8

Denture materials Entirely acrylic = 1 Not entirely acrylic = 2

If not entirely acrylic specify

Frequency of upper denture wearing

Day and night	= 1	Socially	= 5
Day only	= 2	Occasionally	= 6
Only for meals	= 3	Never	= 7
Except for meals	= 4		

Frequency of lower denture wearing

Day and night	= 1	Socially	= 5
Day only	= 2	Occasionally	= 6
Only for meals	= 3	Never	= 7
Except for meals	= 4		

Effect of denture removal

No change	= 1	UPPER
Improved	= 2	
Totally alleviated	= 3	
Worse	= 4	LOWER

Time to achieve effect

Immediately	= 0
< 1 hour	= 1
1 - 3 hours	= 2
3 - 24 hours	= 3
> 24 hours	= 4

Upper Denture

Fit of denture base

Accurate = 1 Slight movement = 2 Considerable movement = 3 ☐

Postdam Present = 1 Absent = 2 ☐

Peripheral extension

Correct = 1 Overextended = 2 Underextended = 3

Posterior border a) ☐

Buccal periphery 1) Height b) ☐

2) Width c) ☐

Labial periphery 1) Height d) ☐

2) Width e) ☐

Lower Denture

Fit of denture base

Accurate = 1 Slight movement = 2 Considerable movement = 3 ☐

Peripheral extension

Correct = 1 Overextended = 2 Underextended = 3

Distal extension a) ☐

Peripheral extension - lingual b) ☐

Peripheral extension - buccal c) ☐

Peripheral extension - labial d) ☐

Jaw Relationship Occlusal face height Correct = 1
Too high = 2
Too low = 3 ☐

Centric Relation Recording
Correct = 1 Incorrect = 2 Unclear due to worn surfaces = 3 ☐

If incorrect tick appropriate reasons :

Antero-posterior fault a) ☐
Lateral fault b) ☐
Rotational fault c) ☐
Premature contact anteriorly d) ☐
Premature contact posteriorly right side e) ☐
Premature contact posteriorly left side f) ☐
Premature contact posteriorly left and right side g) ☐

Jaw Relationship : contd.

Other h)☐

If other specify

If Correct answer next three questions :

Occlusal balance in protrustion Yes = 1 No = 0 j)☐

Occlusal balance in right lateral Yes = 1 No = 0 k)☐

Occlusal balance in left lateral Yes = 1 No = 0 l)☐

Occlusion locked Yes = 1 No = 0 ☐

Tongue restriction

Posterior tooth position (upper)

Correct = 1 Too far buccally = 2 Too far lingually = 3 ☐

Posterior tooth position (lower)

Correct = 1 Too far buccally = 2 Too far lingually = 3 ☐

Anterior tooth position (upper)

Correct = 1 Too far labially = 2 Too far lingually = 3 ☐

Anterior tooth position (lower)

Correct = 1 Too far labially = 2 Too far lingually = 3 ☐

Occlusal plane

Correct = 1 Too high = 2 Too low = 3 ☐

Does patient feel tongue restricted Yes = 1 No = 0 ☐

Abnormal tongue activity Yes = 1 No = 0 ☐

If YES specify

Parafunctional Habits — Yes = 1 No = 0

Clenching ☐

Grinding ☐

Others

Specify

Pressure neurovascular bundles — Yes = 1 No = 0 ☐

Proven allergy to base materials — Yes = 1 No = 0 ☐

If YES specify

Plastic intolerance — Yes = 1 No = 0 ☐

If YES specify

Effect of replacement dentures :

No change = 1

Improved = 2

Totally alleviated = 3

Worse = 4

At 1 month ☐

6 months ☐

12 months ☐

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