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UNIVERSITY OF OTAGO

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Title of Thesis: Aetiology of Denture Sore Mouth: Yeasts and oral hygiene.

Degree for which the thesis is submitted: M.D.S.

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Date: 4 Oct 1971
TITLE: AETIOLOGY OF DENTURE SORE MOUTH: YEASTS AND ORAL HYGIENE.

Author: J. F. Burton, B.D.S. (N.Z.)

Thesis submitted in partial fulfilment of the requirements for the degree of Master of Dental Surgery at the University of Otago, Dunedin, New Zealand, 1971.
PREFACE

Denture sore mouth is a localised inflammation of the oral mucosa seen quite commonly in patients wearing full or partial maxillary dentures.

Trauma and infection with yeast-like organisms are thought to be the most common causes of the inflammation but patients are occasionally told that they are allergic to the material (usually methyl methacrylate) of which the denture is made. It has been established that yeasts of the genus *Candida* are often isolated from the lesions and also that treatment with anti-fungal agents direct to the mucosa will effect resolution of the inflammation. Clinically I have noticed that the inflammation often resolves rapidly, without treatment direct to the mucosa, if the dentures are left to soak overnight in dilute sodium hypochlorite solution. This would suggest that the yeasts are associated with the denture rather than the mucosa. If this is so it would indicate that denture sore mouth is the result of poor oral hygiene.

The investigations in this study were undertaken to determine
1. The association between yeasts present in the saliva, or adhering to the dentures, and denture sore mouth; and

2. The relationship between inadequate denture hygiene and denture sore mouth.
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REVIEW OF THE LITERATURE

1. **INTRODUCTION**

   A condition in which the mucosa under a maxillary denture appeared red and swollen was reported soon after vulcanite was first used as artificial denture base material in 1844. The condition was originally called "rubber sore mouth" because of its association with vulcanite dentures. Cahn (1936), who first drew attention to *Candida albicans* as a possible cause suggested that this was a misnomer and that it should be called "denture sore mouth". The term "denture sore mouth" is still in common usage although many writers consider that this too is a misnomer as the condition is seldom sore. Other names that have been suggested include stomatitis prosthetica, stomatitis contagiosa, stomatitis nudata, stomatopatia allergica, denture stomatitis, and chronic atrophic candidosis. The term denture sore mouth (DSM) will be used throughout this study.

2. **ETIOLOGY**

   According to Nyquist (1952) the aetiology of DSM has been the subject of spirited debate since the middle of the last century. The possible causes have included:
1. Allergic reactions of the mucosa to the denture base material.

2. Chemico-toxic injury.

3. Heat accumulation under the denture base.

4. Traumatic injury to the oral mucosa.

5. Blockage of mucous glands.

6. Infection by C. albicans.

7. Depression of vital resistance due to systemic or deficiency disease and hormonal disturbances.

8. Putrefaction caused by poor oral hygiene and inadequate cleansing of the dentures.

9. The effects of drug therapy.

1. Allergic reactions of the mucosa to the denture base material.

Some early workers (e.g. Detzner, 1905; quoted by Nyquist, 1952) cited allergy to the vermilion used to colour vulcanite rubber. Later workers cited the free monomer in acrylic resin and cadmium and nickel in metal dentures.

Vickers (1949), a dermatologist, described seven patients with DSM and associated angular cheilitis, all of
whom reacted positively to patch tests of acrylic resin. He found that the lesions cleared up when the patients ceased to wear dentures and concluded "these causes estab-
lished the association between denture sensitisation and angular cheilitis". (According to Lyon and Chick (1957)
and Cawson (1966) angular cheilitis and DSM should be regarded as two features of a single disease when they occur together).

Harkness (1954), another dermatologist, reported on 46 patients with angular lesions, 22 of whom had DSM. He concluded that the lesions were caused by sensitivity in 50 percent of the patients but was unable to suggest what the sensitising material was. These two reports are important because of the large number of people who have been led to believe that they are allergic to the denture base material (Fisher, 1956; Lyon and Chick, 1957; Turrell, 1966a).

Nyquist (1952) considered allergy to be an unlikely aetiological factor after carrying out extensive patch and mucosal contact tests on a large number of patients, including a group with diagnosed eczematosus dermatitis and Turrell (1966a) came to a similar conclusion. Fisher (1956) showed that the positive patch tests obtained by strapping an acrylic denture to the forearm can be a non-
specific pressure effect and not one of allergic sensitisation; if the patients own dentures are used irritation may arise from organisms contaminating their surface. He also emphasised that the healing of a stomatitis when a denture is not worn and the prompt recurrence of inflammation when the denture is reinserted are no proof of allergic sensitisation to the base material.

Cawson (1966) and Ritchie, Fletcher, Main and Prophet (1969) consider that true allergy to acrylic resin is very rare if it exists at all but reports continue to be published in the literature (Danilewicz-Stysiak, 1971), claiming allergy as a cause of DSM and advocating a change of denture base material as a cure.

2. **Chemico-toxic injury.**

Residual monomer has been found in processed acrylic resin and it has been suggested that this may cause a toxic reaction in the mucosa. Smith and Bains (1956) found the monomer present in two fractions, one water extractable, the other not. The water extractable part may be associated with the surface of the material but is leached out quite rapidly. The usual time lag before the appearance of DSM would appear to rule this out as a cause of DSM.
3. **Heat accumulation under the denture base.**

There is little evidence for or against heat accumulation under the denture causing DSM. Nyquist (1952) mentions that baseplates of low thermal conductivity, such as acrylic resin, may allow an accumulation of heat and thus permanently raised temperatures which may have a deleterious effect on the mucosa. However Peyton (1968) disagrees and states that the temperature on the tissue side of maxillary dentures is no greater than body temperature.

4. **Traumatic injury to the oral mucosa.**

Many workers have suggested that DSM is the result of traumatic injury to the oral mucosa. Nyquist (1952) carried out an extensive investigation on 1090 patients and related DSM to an assessment of the traumatising factors (including unbalanced occlusion and articulation, rough surfaces and instability) present in each denture. He found the incidence of inflammation increased as the traumatising factors increased and concluded that trauma was the predominant cause of DSM. Furthermore, he was able to cure a number of patients by correcting occlusal discrepancies, by rebasing the lower denture or by constructing a new lower denture. Ritchie et al. (1969) considered that
traumatic factors were present and of sufficient magnitude to cause a primary hyperaemia of the tissues in 93 of 100 patients examined. Budtz-Jorgensen and Bertram (1970a) considered that trauma alone could be responsible for simple localised DSM but that other factors were necessary in order to produce the more generalised inflammations.

Commenting on Nyquist's conclusions Cawson (1963) pointed out that if trauma alone were the cause of DSM the lower denture bearing area would be at least as likely to be involved. Nyquist specifically excludes the mandible from his study and evidence from the literature indicates that DSM rarely involves the mandible. Cawson suggested that a firmly fitting denture might cause trauma merely by its enclosure of the tissues over a period of months or years. He did however state emphatically that DSM can arise in the absence of trauma and other workers have formed similar conclusions (Lyon and Chick, 1957; Budtz-Jorgensen and Bertram 1970a).

5. **Blockage of mucous glands.**

Ostlund (1953) showed that the wearing of a denture is capable of causing dilation of the salivary ducts, especially if the denture base fits well. This occurs
because of pressure on the duct orifices.

Basing his work on Ostlund's study, Newton (1962) likened DSM to the sweat retention syndrome. He suggested that DSM might be due to the lateral spread of saliva into the tissues following occlusion of the duct orifices. As Neill (1965) has suggested, if Newton's theory is correct it would not be unreasonable to suppose that the inflammatory lesion would initially be confined to the posterior part of the palate (i.e. the area of the palatal mucous glands). It would also be difficult to account for the diffuse erythema involving the whole of the denture bearing tissues which is characteristic of the more severe cases of DSM. Clinical evidence suggests that the lesions usually commence more ventrally in the palate and spread to involve larger areas of the denture bearing mucosa.

6. **Infection by Candida albicans.**

DSM had been observed for nearly a century before Cahn (1936) suggested the possibility of yeasts as an aetiological factor. Bartels (1937) studied cultures from Cahn's patients and isolated yeast-like organisms which he identified as belonging to the genus *Candida.*
Since 1936 various workers have carried out mycological examination for Candida species, and the association of DSM with the yeasts has been established. However, the argument as to whether trauma or the yeast organisms are the main factor has continued. Turrell (1966b) has said that the controversy as to whether trauma is primary or secondary to yeast infection is of academic interest only, and it is doubtful if conclusive proof either one way or the other can be given.

In the earliest published case series, Nyquist (1953) isolated Candida species from 29 per cent of patients with DSM and 19 per cent of controls. He concluded that there was no significant difference in the incidence of the yeasts. Lyon and Chick (1957) however, found Candida species in swabs from 72 per cent of patients with DSM compared with 17 per cent of controls. More recently Budtz-Jorgensen and Bertram (1970a) reported isolating yeast-like organisms from 90 per cent of DSM patients and 40 per cent of controls. Ritchie and his colleagues (1969) demonstrated hyphae, which they identified as those of C. albicans, in all of 60 patients with DSM while they were unable to find any evidence of yeasts in 750 smears from the palates of 50 patients with healthy mucosa. They did not state, however, whether or
not these patients were wearing dentures or how the examined material was obtained. Lehner (1965), using the quantitiative immunofluorescent technique, demonstrated significantly raised serum antibody titres against C. albicans in patients with DSM.

Other evidence for the role of yeasts in DSM is found in the success of anti-fungal agents in the treatment of the lesions. Cahn (1936) reported a favourable result from painting the lesions with 5 per cent gentian violet followed by Grams iodine solution. Lyon and Chick (1957) reported some success with sodium caprylate, while later workers have used pruvagol (Chick, 1962), Nystatin (Cawson, 1963), Amphotericin B (Cawson, 1963) and Remiderm (Ritchie, et al., 1969).

Only Davenport (1970) has investigated the yeasts on the whole of the fitting surface of the maxillary denture and he reported no detailed results, merely stating that in all but one case he obtained more colonies from the denture than from the mucosa.

7. Depression of vital resistance due to systemic disease, deficiency disease and hormonal disturbance.

The effects of systemic disease, deficiency disease
and hormonal imbalance on the oral mucosa do not seem to be very well understood, but most workers are in agreement that they result in lowered resistance to infection and poor tissue healing. The systemic influences are therefore thought unlikely to cause DSM, but there is little question that the lesions are much more difficult to cure when the systemic state is depressed.

Certain conditions have been shown commonly to be associated with DSM. Rose (1963) in a study on angular cheilitis and iron metabolism found that 72 per cent of a series of patients with angular cheilitis also had DSM. He found in many cases the DSM disappeared at the same time as the angular lesions; in some instances the only treatment was systemic iron. He concluded that iron deficiency may be concerned in the aetiology of DSM.

DSM has often been noticed in diabetics (Cahn, 1936; Cawson, 1963; Ritchie et al., 1969). Peters, Bahn and Barens (1966) found however, that the diabetic condition did not appear to increase the incidence of inflammation beneath prosthetic appliances and suggested that the primary aetiological factor in such cases was the denture itself.

DSM has been said to occur more than twice as frequently
in women as in men. A high incidence has also been noticed in the fourth and fifth decades. Neill (1961) investigated 9 female patients with DSM whose ages ranged from 41 - 59 years. Both the neutral-17-keto-steroid levels of the urine, and the blood ascorbic acid levels were found to be abnormal and dihydro androsterone levels were markedly reduced. Neill suggested that variation in the tolerance of the oral tissues to different types of trauma may be accounted for in some measure by endocrine factors.

Vitamin C and several of the B complex group of vitamins including niacin, riboflavin, pyridoxine, pantothenic acid, folic acid and vitamin B12 are considered to be related to the health of the oral mucosa (McCarthy and Shklar, 1964). Riboflavin has long been associated with the development of angular cheilitis but there is little in the literature to link it with DSM. Cawson (1963) found no evidence of vitamin deficiencies in his group of patients and stated that there seems no reason to suppose that riboflavin is of importance nowadays as a cause of angular cheilitis. No evidence of blood assays of vitamins in patients with DSM has been reported although Makila (1969) has shown reduced riboflavin and thiamine levels in patients with angular cheilitis, 46 per cent of whom had DSM.
Kimball (1954) considered that nutritional inadequacies seldom cause frank oral manifestations and suggested that, as many are sub-clinical in nature, dentists should rely on subjective symptoms rather than oral signs when considering the nutritional state. Horwitt, quoted by Beatoq and McHenry (1964) suggested that the type of abnormality encountered in riboflavin deficiency may be dependent on trauma, irritation or infection. Riboflavin is essential for tissue repair.

8. **Putrefaction caused by poor oral hygiene and inadequate cleansing of the dentures.**

Several writers have suggested that dirty dentures may cause DSM. Most investigators have examined denture cleanliness in conjunction with DSM but their criteria for deciding whether or not a denture is clean are not clear. Budtz-Jorgensen and Bertram (1970a) examined dentures using disclosing solution (proflavine-monasulfate in 3 per cent aqueous solution) to detect plaque on the anatomical surface of maxillary dentures, and subsequently provided an index for denture cleanliness. This index, however, appears to take no account of the amount of debris but merely allows recording of the area of stain on the denture surface.
They found that patients with severe inflammation usually had poorly cleaned dentures. Gawson (1966) had reported that he had been unable to show a relationship between poor denture hygiene and DSM. Davenport (1970) considered DSM was associated with plaque on the denture and suggested that treatment should be aimed at the denture rather than the mucosa.

9. **The effects of drug therapy.**

Antibiotics, corticosteroids and various kinds of psychotherapy preparations have been considered to predispose the patient to DSM and other oral fungal diseases, particularly thrush. Grill and Mikalowski (quoted by Winner and Hurley, 1964) reported that penicillin chewing gum led to oral thrush in 11 out of 50 patients. Chick (1962) pointed out that DSM gets worse following the administration of antibiotics and that it regresses rapidly to its former state once the antibiotic is withdrawn. McKendrick (1966) studied DSM and angular cheilitis in patients receiving long-term tetracycline therapy, but found no significant relationship between the administration of the drug and the incidence of DSM. He suggested, however, that if a degree of inflammation is already present in the mouth, tetracycline therapy
encourages *Candida* infection and exacerbation. It has also been shown that antibiotic therapy in some cases preceded the detection of DSM and led immediately to the oral changes (Ritchie *et al*., 1969; Budtz-Jørgensen and Bertram, 1970a).

Several workers have commented on the oral side effects of drugs used in psychiatric medicine. These drugs are being used on an increasing scale. Pollack, Buck and Kalnins (1964) found a high incidence of DSM arising one or two months after treatment with tranquilising or anti-depressive drugs. Bliss (1969) noticed xerostomia and DSM in patients on chlorpromazine.
MATERIALS AND METHODS

CLINICAL EXAMINATION

Eighty nine patients were included in this investigation. The only criteria for selection was that the patient had been wearing a full upper denture for at least twelve months prior to examination. Patients were selected from amongst those presenting for treatment at the Otago Dental School, and the examination was carried out under standardised conditions in the Department of Prosthetic Dentistry.

The information recorded for each patient is shown in figure 13 (see appendix ). Most of the entries on this chart are self-explanatory but some require explanation:

(1) General health and drugs. The patients were questioned about their general health and any recent drug history. No investigations were carried out.

(2) Daily duration of wear. Patients were asked whether or not they slept with their dentures in their mouths. Those who usually wore the upper denture at night were recorded as positive, the remainder as negative.

(3) Cleaning Methods. Patients were asked what method of denture cleaning was commonly employed. If the patient made a regular daily habit of soaking the denture in a cleansing solution whether for a short or long period, the
method was entered as "SOAK". Other patients were entered as "BRUSH" or "NONE".

(4) Angular cheilitis. This was recorded as present if any signs of inflammation with or without fissures or ulceration were seen at the corners of the mouth.

(5) Efficiency of dentures. Retention, stability and occlusion were recorded as a means of assessing any traumatic factors which might be present.

Retention was judged by applying the thumb and forefinger to the upper incisor teeth and exerting a tensile force at right angles to the occlusal plane. If the denture was easily displaced it was classed as negative, otherwise positive.

Stability was tested by applying pressure in a direction at right angles to the occlusal plane on each premolar region separately. Again the relative ease of dislodgement was the deciding factor in classifying the stability as positive or negative.

Occlusion was classed positive if the teeth were in centric occlusion when the mandible was in centric relation to the maxilla and the occlusion was reasonably balanced and free in lateral relationships.
(6) **Appearance of mucosa.** Oral inflammation was classified according to the methods described by Newton (1962):—

**DSM Class I.** *Pin-point hyperaemia:* small areas of inflammation in otherwise normal tissue, usually found around the ducts of the palatal mucous glands. Patchy inflammation, confined to the palatal vault, was included in this group.

**DSM Class II** *Diffuse hyperaemia:* a generalised inflammation of the entire denture bearing area; the surface of the mucosa was smooth.

**DSM Class III.** *Granular:* the mucosa had a nodular hyperaemic surface which was sometimes present over the entire denture bearing area but more commonly confined to the central area of the palate.

Any inflammation which did not appear to fit into any of these classes was entered as "Other Inflammation".

The topography of the inflammatory process in each case was plotted on a drawing of the denture bearing area (see figure 1 and figure 14 in appendix).
Figure 1. Diagrammatic drawing of maxilla, denture palate, and agar mould, as used to record information.

Figure 2. Photograph of palate, showing relationship to diagrammatic drawing (figure 1).

Figure 3. Photograph of denture showing relationship to diagrammatic drawing (figure 1).
1, 8, 9, 16 ...... Buccal sulcus
2, 7, 10, 15 ...... Alveolar crest
3, 6, 11, 14 ...... Side of palatal vault
4, 5, 12, 13 ...... Roof of palate
17, 18 ...... Post dam area
A code was used as follows:

Severe inflammation........... red shading
Moderate inflammation......... orange shading
Mild inflammation............. yellow shading

(7) Denture hygiene: a subjective assessment of denture hygiene followed a ten second rinse of the denture under the cold tap. The object of this rinse was to remove any loosely adhering material. The upper denture was examined under standard lighting conditions without magnification and cleanliness was described according to the amount of adherent debris on the fitting surface as

Good.................... no obvious signs of debris
Fair...................... few small patches of debris
Poor...................... widespread debris visible.

During the course of the study it became obvious that additional information was required regarding the assessment of denture cleanliness. For this reason a technique employing a disclosing solution was implemented (in addition to the visual examination) for the final sixty patients. This was carried out after all other tests had been completed and just prior to the dismissal of the patient. The fitting surface of the upper denture was flooded with
proflavine hemi-sulphate in 3 per cent aqueous solution and then rinsed under the cold tap. According to the amount of staining on the denture base the patients could be divided into three groups using the following index of denture cleanliness.

Good...... none or only a few spots of stain

Fair...... more extensive staining involving less than half of the denture base

Poor...... more than half the denture base stained.

The topography of the stained area was then mapped on to a drawing of the denture bearing area (see figure 15 in appendix). To obtain an estimation of the relative distribution of plaque in various parts of the anatomical surface of the denture, a clear numbered template (see figure 15 in appendix) was placed over the topographical drawing of plaque for each denture. Where a third or more of each area showed staining it was recorded as positive, and the total number of positive recordings for each area was obtained.

MICROBIOLOGICAL EXAMINATIONS

(1) Yeasts present in the saliva: a saliva sample was obtained prior to any examination and immediately following initial removal of the dentures from the mouth.
Ten ml of 0.85 per cent saline was rinsed around the mouth for 10 seconds and collected in a sterile container. This was placed in a refrigerator until microbiological examination was commenced. The number of viable yeasts in each mouth rinse was determined by plating 0.1 ml of serial ten fold dilutions on to brain heart infusion agar (Difco) plates (containing 0.05 mg/ml chloramphenicol). Each dilution was plated in triplicate. After 48 hours incubation at 37°C, the average number of colonies per dilution was calculated. This figure was then multiplied by the appropriate factor to give a total yeast count per 10 ml of the original sample.

*C. albicans* was identified by germ tube formation in serum and other yeasts by morphological features, and by fermentation and assimilation tests (Mackenzie, 1966).

(2) Yeasts present on the upper denture: following the subjective examination for cleanliness the upper denture was boxed with utility wax and culture medium (brain heart infusion agar plus 0.05 mg/ml chloramphenicol) was poured into it at a temperature of 40°C (see figure 4). After it had solidified the agar model was removed from the denture, placed in a sterile covered glass dish and incubated at 37°C for 48 hours. The topography of the growth (figure 5), if
any, was plotted on a drawing of the denture bearing area (see figure 16 in appendix). Where possible individual colonies were drawn, but when the colonies were confluent the approximate area was shaded. A quantitative assessment of yeast growth was made and graded. This was as follows:

0........ no growth

+......... a few scattered colonies

++......... larger number of organisms present in a few areas or moderate number over the whole area

+++......... large numbers present over the whole area.
Figure 4. Photograph showing method of obtaining imprint culture of the fitting surface of the upper denture.
RESULTS

APPEARANCE OF THE MUCOSA

Eighty-nine patients were examined. Thirty-seven were considered to have denture sore mouth (DSM), 23 had other inflammation (OI) of the oral mucosa, and 29 were considered to have a normal (N) healthy mucosa. The sex and age distribution within these three groups is shown in Table I. Of the patients with DSM, 22 had Newton type 1, 9 Newton type 2, and 6 Newton type 3 inflammation.

YEAST ISOLATIONS

(a) Mouth rinse: yeasts were isolated from the mouth rinse of 95 per cent of the patients with DSM, 44 per cent of the patients with other inflammation, and 31 per cent of those with normal mucosa. Detailed results are shown in Table II, which also shows the figures for C. albicans alone.

A standard error test on the significance of the differences in the two percentage figures for the OI and N groups confirmed the null hypothesis that there is no real difference in the percentage of subjects for whom yeasts were isolated ($Z = 0.96; P > 0.05$). It was therefore justifiable to pool these two groups into a single control group in which the percentage of subjects from whom the
yeasts were isolated was 37 per cent. When this value was compared with the 95 per cent for the DSM group using a standard error test, the null hypothesis was rejected \((Z = 7.63; \ p < 0.01)\). It may therefore be concluded that the percentage from whom yeasts can be isolated is significantly higher in the DSM group than the control group.

(b) **Imprint culture**: the main feature regarding yeasts was that these organisms were isolated from the fitting surface of the upper dentures of all 37 patients who were considered to have DSM. Positive cultures were obtained from 22 (42 per cent) of the remaining 52 patients. Heavy growths of yeasts were obtained in 81 per cent of the patients with DSM and 19 per cent of the remainder. The detailed results are shown in Table III and figure 6.

By using a standard error test on the significance of the difference between yeasts isolated from the denture of those in the DSM and the \((0I + N)\) groups, it was possible to reject the null hypothesis \((Z = 8.48; \ p < 0.01)\). It may therefore be concluded that the percentage from whom yeasts were isolated is significantly higher in the DSM group than in the \((0I + N)\) group.
Figure 5. Photograph showing sample of yeast growth on imprint culture.
<table>
<thead>
<tr>
<th>Appearance of mucosa</th>
<th>No. examined</th>
<th>Male</th>
<th>Female</th>
<th>Age in years</th>
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<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>&lt;30</td>
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<td>DSM</td>
<td>37</td>
<td>15</td>
<td>22</td>
<td>10</td>
</tr>
<tr>
<td>OI</td>
<td>23</td>
<td>6</td>
<td>17</td>
<td>2</td>
</tr>
<tr>
<td>N</td>
<td>29</td>
<td>17</td>
<td>12</td>
<td>2</td>
</tr>
<tr>
<td>TOTAL</td>
<td>89</td>
<td>38</td>
<td>51</td>
<td>14</td>
</tr>
</tbody>
</table>


**TABLE II: INCIDENCE OF YEASTS PRESENT IN THE MOUTH AS COMPARED WITH THE MUCOSAL APPEARANCE**

<table>
<thead>
<tr>
<th>Appearance of mucosa</th>
<th>No. examined</th>
<th>No. of yeasts / 10 ml mouth rinse</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>&lt;100**</td>
</tr>
<tr>
<td>DSM</td>
<td>37</td>
<td>2 (3)</td>
</tr>
<tr>
<td>OI</td>
<td>23</td>
<td>13 (14)</td>
</tr>
<tr>
<td>N</td>
<td>29</td>
<td>20 (25)</td>
</tr>
<tr>
<td>TOTALS</td>
<td>89</td>
<td>35 (42)</td>
</tr>
</tbody>
</table>

* The first column represents total figures for all yeasts including *C. albicans.* The figures for *C. albicans* alone are shown in parentheses.

** Figures of <100 indicate that no organisms were found.

<table>
<thead>
<tr>
<th>Appearance of mucosa</th>
<th>No. examined</th>
<th>Growth of yeast colonies</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>0</td>
</tr>
<tr>
<td>DSM</td>
<td>37</td>
<td></td>
</tr>
<tr>
<td>OI</td>
<td>23</td>
<td>12</td>
</tr>
<tr>
<td>N</td>
<td>29</td>
<td>18</td>
</tr>
<tr>
<td><strong>TOTAL</strong></td>
<td><strong>89</strong></td>
<td><strong>30</strong></td>
</tr>
</tbody>
</table>

0 ..... No colonies

+ ..... A few scattered colonies

+++ ..... Large numbers of colonies present in a few areas or moderate number over the whole area

+++ ..... Large numbers of colonies over the whole area
Figure 6. Histogram showing association between health of mucosa and yeasts on denture.
AGE OF THE DENTURES

There was no evidence that the age of the denture was related to the appearance of the mucosa. Twenty-one patients were wearing dentures less than 5 years old, while the remainder stated that their dentures were from 5 to 25 years old.

HEALTH OF THE PATIENT

The indicated general health of the patient did not appear to influence the appearance of the mucosal surface: 73 per cent of patients with DSM, 74 per cent of those with other inflammations, and 65 per cent of those with a normal mucosa reported good health.

PRESENCE OF ANGULAR CHEILITIS

Angular cheilitis was observed in 20 (54 per cent) of the patients with DSM, and 4 (8 per cent) of the others. Using the chi square test, with yates' correction, this difference was found to be statistically significant ($x^2 = 21.29$, $p < 0.01$). A moderate or heavy growth of yeasts was obtained from the dentures of 21 (88 per cent) of these patients.

DENTURE HYGIENE

(1) Visual assessment: DSM appeared more prevalent in those patients who did not keep their dentures well
TABLE IV: RELATIONSHIP OF APPEARANCE OF ORAL MUCOSA TO DENTURE HYGIENE: VISUAL ASSESSMENT

<table>
<thead>
<tr>
<th>Condition of mucosa</th>
<th>No. examined</th>
<th>Denture hygiene</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>Good</td>
</tr>
<tr>
<td>DSM</td>
<td>37</td>
<td>5</td>
</tr>
<tr>
<td>OI</td>
<td>23</td>
<td>8</td>
</tr>
<tr>
<td>N</td>
<td>29</td>
<td>17</td>
</tr>
<tr>
<td><strong>TOTALS</strong></td>
<td><strong>89</strong></td>
<td><strong>30</strong></td>
</tr>
</tbody>
</table>
cleaned. Sixty-two per cent of those patients who were considered to have poorly cleaned dentures had DSM, compared with 48 per cent of those whose dentures appeared fairly clean, and 16 per cent of those whose dentures were well cleaned. Statistical analysis using Brandt and Snedecor's Chi square formula confirmed this finding ($\chi^2 = 12.86 \ p < 0.01$). Detailed results are shown in Table IV and figure 7.

(2) Using disclosing agent: the dentures of 60 patients were assessed. Three (5 per cent) were considered to be clean, 34 (57 per cent) fairly clean and 23 (38 per cent) dirty. No conclusions could be drawn with regard to associated tissue inflammation.

It was obvious that patients experienced difficulty in cleaning the undercut areas of the labial and buccal flanges. Of the 60 dentures examined with disclosing solution, only one was completely free of plaque while seven were completely coated showing a total failure of cleaning methods. Figure 8 is a histogram showing the percentage of dentures which exhibited large areas of plaque in 18 subdivided sections of the fitting surface under examination. The quantity of plaque was not taken into consideration.
Figure 7. Histogram showing association of health of mucosa and denture hygiene (visual assessment).
Figure 8. Histogram showing distribution of plaque on fitting surface of full upper dentures.
DENTURE CLEANING HABITS

A total of 16 of the 89 patients claimed to soak their dentures in some cleansing solution regularly. Eleven of these used an overnight method while 5 steeped the dentures for periods of from 10 to 30 minutes. Ten of the 16 patients were subjectively considered to have good denture hygiene and 6 fair. DSM was seen in 3 patients each of whom was found to wear the upper denture overnight and immerse it only for a short period daily. Seventy-three patients used immersion cleansers rarely or not at all; these patients all brushed their dentures using a variety of adjuncts of which toothpaste and denture paste were most frequently mentioned. It was interesting to note that few yeasts were obtained from the dentures of those who used the immersion method. Yeasts were obtained in only three cases and each of these had DSM. Only 17 of the 73 patients who usually brushed their dentures were free from fungal elements.

Although the number of patients who regularly soaked their dentures was small, statistical analysis using the chi square test with Yates' correction indicated that the difference between the incidence of yeasts isolated from the
dentures of these patients and those isolated from the patients who did not soak their dentures could not have occurred by chance ($x^2 = 17.22; \ p < 0.01$). It was concluded that the immersion of the dentures in a cleansing solution was a more effective way of eliminating yeasts from the denture surface than brushing.

**NIGHT WEAR**

Most patients wore their dentures at night although some removed the lower denture and retained the upper. Only 14 (16 per cent) removed the upper denture at night. DSM was seen in 2 of these patients, neither of whom used a cleansing solution.

**TOPOGRAPHICAL DISTRIBUTION OF INFLAMMATION, YEASTS AND RETAINED DEBRIS**

An attempt was made to show a relationship between these three factors but no such relationship could be seen.

**YEASTS ISOLATED**

The dominant species isolated was *C. albicans*. Other species isolated are shown in Table V. Of these, *C. albicans*, *Candida pseudotropicalis*, *Candida parapsilosis* and *Torulopsis glabrata* have been shown to be pathogenic to man.
TABLE V: RELATIONSHIP OF YEASTS PRESENT IN THE MOUTH TO THE APPEARANCE OF THE MUCOSA

<table>
<thead>
<tr>
<th>Yeast species isolated</th>
<th>Appearance of mucosa</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>DSM (37)*</td>
</tr>
<tr>
<td>Candida albicans</td>
<td>34</td>
</tr>
<tr>
<td>C. robusta</td>
<td>2</td>
</tr>
<tr>
<td>C. pseudotropicalis</td>
<td>1</td>
</tr>
<tr>
<td>C. solani</td>
<td>-</td>
</tr>
<tr>
<td>C. zeylanoides</td>
<td>-</td>
</tr>
<tr>
<td>C. melinii</td>
<td>1</td>
</tr>
<tr>
<td>C. rugosa</td>
<td>-</td>
</tr>
<tr>
<td>C. parapsilosis</td>
<td>-</td>
</tr>
<tr>
<td>C. mycoderma</td>
<td>1</td>
</tr>
<tr>
<td>Candida species</td>
<td>2</td>
</tr>
<tr>
<td>Torulopsis glabrata</td>
<td>4</td>
</tr>
</tbody>
</table>

* Number in group
DISCUSSION

It was originally intended to examine at least 100 patients but time and the lack of availability of suitable subjects forced a reduction in numbers. Nevertheless much useful information was provided by those who were eventually examined.

The recognition and classification of inflamed tissues sometimes proved difficult. As there was no definite selection of patients either with or without DSM, a decision had to be made in each case and the patient assigned to one of the three groups. In this study any deviation from the normal healthy appearance of the oral mucosa was recorded. As Hedegard (1970) points out a classification of a slightly inflamed mucosa can be obtained only when there is thinning of the epithelium and an inflammatory reaction in the lamina propria. He suggests that clinical evaluation therefore yields information of definite and rather marked surface changes of the oral mucosa. Thus any errors in the placement of the patients within the three groups in this study are likely to have resulted in patients with DSM being placed in one of the other two groups rather than the reverse.
It should be realised that DSM does not always present as an obvious inflammation of the mucosa and only a small percentage show the deep red inflammation of the complete denture bearing mucosa. The amount of inflammation will vary from time to time with the state of bodily resistance of the tissues and the denture hygiene. For example quiescent DSM may show exacerbation following a bout of 'flu or a severe cold. Under these circumstances the yeasts may even invade the epithelium giving the clinical picture of thrush. Several patients commented that their angular cheilitis was most noticeable when they had a cold and at other times disappeared altogether.

The criteria for clinical evaluation of DSM in previously reported studies have varied. Cawson (1963) included only those lesions which involved the whole of the denture bearing mucosa and were exactly confined to it; Davenport (1970) included inflammation of more than half the covered mucosa, while Nyquist (1952) and Ostlund (1958) used criteria similar to those used in this study.

Yeasts of the genus Candida have been isolated from many parts of the human body, particularly the alimentary tract. Of the various species which have been recovered
from man *C. albicans* is by far the most prevalent. Vanbreuseghem (1966) considers that *C. albicans* is present as a commensal in the intestines of all human beings. Bartels and Bleckman (1962) reported an incidence of 40 per cent in normal mouths. Its occurrence in totally edentulous mouths may be less. Turrell (1966b) found *C. albicans* present in 10 per cent of patients who had been rendered edentulous one month prior to examination and had never worn dentures. The isolation of yeast-like fungi from sites such as the mouth where they are likely to occur saprophytically should not, therefore, be taken as evidence of disease (Mackenzie, 1966).

The question therefore arises, "what role, if any, do candidas, and particularly *C. albicans*, play in the development of DSM?"

Investigators have attributed the ability of *C. albicans* to produce cutaneous disease to two different mechanisms. There are those who maintain that infection through invasion and subsequent digestion of keratin by the fungus is the basic process (Winner and Hurley, 1964; Kapica and Blank, 1957). Such a view has recently received support from electron microscopic observations. Montes and Wilborn
(1968) showed *C. albicans* outside, penetrating and within
cast off epithelial cells in patients with chronic muco-
cutaneous candidosis. On the other hand there are those
who believe cutaneous candidosis is a primary irritant
type of contact dermatitis. In the latter mechanism, as
shown by Maibach and Kligman (1962), occlusion is a necessary
factor.

Although *C. albicans* has been shown to be locally
invasive in thrush lesions, attempts to show tissue invasion
in DSM have not been successful (Cawson, 1964; 1966; Budtz-
Jørgensen, 1970; Davenport, 1970). If the organisms do
not invade the superficial layers of the tissues then they
must be growing on the surface of the mucosa and/or sapro-
phytically within the plaque on the denture base. Many
workers have demonstrated yeast elements in smears from the
mucosa, but several have spoken of the difficulty in
obtaining positive results. Davenport took smears from
both the mucosa and the denture and commented that the
concentration of yeasts on the denture was usually greater
than on the mucosa. Of the 40 people in the present study
who were found to have large numbers of colonies on their
dentures 30 were found to have DSM, 7 had other inflam-
mation, while only 3 had healthy mouths. On the other hand, of
29 patients who were considered to have normal mucosa only 
2 (7 per cent) had a high yeast "mouth rinse" count and only 
3 showed large growth of organisms on imprint denture culture. 
No attempt was made to isolate organisms from the palatal 
mucosa except indirectly by the mouth rinse technique. 
There would certainly seem to be a correlation between a 
high incidence of yeasts on the denture and DSM. 

Support for the denture as the more likely focus of 
infection is found in various attempts at treatment.

(1) Several workers (Lyon and Chick, 1957; Budtz- 
Jorgensen, 1970b), have treated the mucosa without attempting 
to disinfect the dentures which were worn throughout treat-
ment. They have commented that while drug therapy has 
usually been successful, immediate relapse often followed 
withdrawal of the drug. Reinfection from the denture has 
been mentioned as the likely cause.

(2) Cahn (1936) found that resolution of the lesions 
could be effected without any treatment to the mucosa if the 
dentures were left overnight in sodium hypochlorite. This 
has been my experience too. The possibility that the 
hypochlorite affects the yeasts on the mucosa directly is 
thought to be small as the smell and taste of the substance 
is enough to ensure that the denture is thoroughly washed.
before being reinserted in the mouth.

Cawson (1966) suggested that the space between the denture and the mucosa, with no natural cleansing mechanisms and plenty of food debris, acted almost as a culture medium while Hurley (1966) states that Candida species grow easily on almost any medium at body temperature. Cawson, however, was unable to show any correlation between DSM and imperfect cleansing of the denture although he considered that such a correlation would seem reasonable. In this study the results indicated that the standard of hygiene of the DSM group was considerably poorer than that of the other two groups. Thus it would seem that there is some correlation between inadequate denture hygiene and DSM.

Maibach and Kligman (1962) produced experimental lesions in human subjects by applying a dense suspension of dead organisms to skin and covering the area with tape. They found that the lesions occurred only when the area was completely occluded and that they underwent spontaneous regression as soon as the tape was removed. They considered the lesions were due to toxic materials produced by the fungus and stated:
"... this demonstrated clearly that the living organisms do not have to invade the tissues to produce changes and that toxic substances produced by the growth of the organism on the surface are entirely sufficient to produce the inflammatory response ..."

Similar conditions could be set up under a full upper denture or even under the bar of a metal partial denture if hygiene was poor. The inflammation is seldom seen under lower dentures and this may be because the mandibular mucosa is bathed in a constant flow of fresh saliva due to the greater amount of saliva in the floor of the mouth and the habitual movement of the lower denture. In view of the larger number of yeasts found associated with the upper denture and the apparent absence of tissue invasion it seems likely that DSM is a contact irritant reaction.

_Candida_ species have been isolated from the saliva, palatal mucosa, tongue, angles of the mouth, and dentures by many different methods, and the incidence of yeasts found in patients with DSM has varied widely. Nyquist (1953) found yeasts in only 29 per cent of DSM patients while the figure reported by Davenport (1970) was 98 per cent. It is considered probable that the method of isolation and recognition of the yeasts must, in part at least, be the cause of the wide variation.
Few workers have studied the yeast-like flora adhering to dentures and only Cawson (1963), who mentioned six cases, has reported studying yeast growth from imprint cultures of the maxillary dentures. Nyquist (1953) placed unsized paper between the denture and the mucosa for ten minutes, placed the paper in ordinary broth at pH 7.2 and plated the suspension on to blood-agar. Davenport (1970) obtained replica cultures from the fitting surface of the upper denture and the palatal mucosa by using plastic foam sandwiched between the denture and the mucosa. He commented, however, that negative cultures were frequently obtained when smears obtained by scraping the denture were positive. His figure of 98 per cent was obtained by using isolations from the saliva and smears from the denture and the mucosa in addition to the replica cultures.

While no cross checks were done using methods identical to those used by other workers, the method of imprint culture used in this study would appear to be at least as sensitive as any other in demonstrating the presence of yeasts in the oral cavity.

It is considered probable that some patients make a special effort to clean both the dentures and the mouth
prior to visiting the dentist. By such action they would undoubtedly affect the yeast population and thus no single negative test should be taken as definite evidence that yeast-like organisms are not present in the mouth.

The results of the topographical investigation of retained plaque on the dentures indicated that most patients found it difficult to keep their dentures really clean. The evidence showed that the undercut fitting surfaces of the labial and buccal flanges were seldom adequately cleaned and that the palatal area which is readily accessible to a brush was usually well-cleaned. It is perhaps unfortunate that the areas which are most difficult to clean are also those which are likely to receive the most food debris through ingress round the periphery. It was noticeable that those who made a regular habit of soaking their dentures overnight in some cleansing solution had far better denture hygiene than those who did not. It would therefore seem reasonable to suggest that patients should not wear their dentures at night but should leave them in some cleansing solution. Many patients object to doing this on the grounds of vanity. Others however, say they do not like leaving their dentures soaking overnight because they feel "loose" when they are reinserted in the morning. This,
however, is no indictment of the method of cleaning but probably an indication that the dentures no longer fit properly and require relining or renewing. It may also mean that the neuromuscular mechanisms are upset by the removal of the dentures at night and take some time to readjust when the dentures are reinserted.

The conflicting reports of the effects of "night wear" on the development of DSM are of interest. Nyquist (1953), Neill (1965) and Budtz-Jorgensen and Bertram (1970a) found that the incidence of DSM was not affected by the nightly removal of the dentures, but Ritchie et al. (1969) and Davenport (1970) found a greater percentage of cases of DSM amongst those patients who wore their dentures continuously than amongst those who removed their dentures at night.

In this study the percentage of patients who wore their maxillary dentures at night (84 per cent) was greater than that reported by any of the above authors, and the 14 patients who did not wear their upper denture represents too small a number from which to draw any firm conclusions. Certain trends however, were evident. Eleven patients who left their dentures overnight in alkaline hypochlorite, alkaline peroxide, or cetrimide, which all are fungistatic,
showed no evidence of DSM or yeasts. Three patients however, left their dentures either in a glass of water, or under the pillow; two had DSM and yeasts were found in all three. It does seem quite possible therefore that it is not the nightly removal of the denture which affects the incidence of DSM, but rather the way in which the appliance is stored overnight. None of the previous investigators have mentioned studying this aspect and further investigations are needed before any firm conclusions can be drawn.

Patients often ask their dentists whether or not they should wear their dentures at night, and the dentist should be in a position to give information which will enable the patient to decide for himself. The hygiene aspect should be stressed: the answers so often given even in published booklets are unsatisfactory. "Please yourself" (in Advice to patients about Dentures", University of Otago Dental School, 1969) or "the dentures can be left out at night if you so prefer" (New Zealand Dental Association Council of Dental Health Education, 1964, "Make a success of your Denture ") is inadequate advice. Detailed information about hygiene and the possible effects of its neglect should be given to the patient, who may then make his own
decision. Published information of this type appears to be scanty and many dentists are little better informed than the general public.

It should not be thought that the material adhering to dentures is purely food debris. Budtz-Jørgensen and Bertram (1970a) consider that leucocytic emigration and the continuous shedding of epithelial cells from the inflamed mucosa in patients with DSM are more important sources of detritus than food. Smears from dentures and mucosa certainly show large numbers of both (Ritchie et al., 1969; Davenport, 1970). These substances appear to combine with mucin to form plaque which is firmly attached to the denture base. Following the application of disclosing solution to the denture in this study it was necessary to clean the denture prior to returning it to the patient. This was often an extremely difficult task even when no calculus was present. It was frequently necessary to polish the denture on a laboratory lathe to remove the stained deposits from the undercut areas.

"How should I clean my dentures?" is a question commonly asked by patients. The answer may well vary with the material of which the denture is made as well as
its shape, but it is obvious that deposits on the denture can only be removed by brushing when their attachment to the base material is weak and the brush is used with an expertise beyond all but the most skilful and fastidious patient. While some authors (Nyquist, 1953; Budtz-Jorgensen and Bertram, 1970a) found no evidence to link the frequency of denture cleansing with DSM, MacCallum, Stafford, MacCulloch and Coombe (1968) found that the cleanliness of dentures bore no relationship to the frequency of cleaning. It would seem that success in cleaning results from good technique and not necessarily the frequency of cleaning. As most of the immersion cleaners are strongly bacteriostatic and fungistatic and also loosen debris, they must be seen as an essential part of the cleansing process. Brushing is also desirable and patients should be shown, by chairside instruction, how to carry this out effectively. It is somewhat amazing to read that Neill (1968) found that many patients cleaned their dentures in situ.

The proportion of patients with DSM and concomitant angular cheilitis (54 per cent), was in keeping with the findings of previous workers; Cawson (1966) considered that 75 per cent was a closer figure though the angular cheilitis was not always present at the time of examination.
Angular cheilitis can be unsightly and painful and is often the reason why patients with DSM present for treatment. Unfortunately local treatment to the angular lesions is often prescribed and the lesions may clear up but recur at regular intervals because of the failure to reduce the intra-oral yeast population. Several workers have noted that the lip lesions will heal without any local treatment when the associated DSM has been successfully treated. There seems to be adequate justification for the views held by Lyon and Chick (1957) and Cawson (1966) that angular cheilitis and DSM, when present together, should be regarded as two manifestations of the same disease.

An attempt was made to investigate traumatic factors as trauma is considered to be a major cause of DSM (Nyquist, 1952). Excess pressure acting on the denture bearing tissue is extremely difficult to assess. According to Neill (1965) it may be caused by

1. Too great a vertical dimension
2. Errors in centric jaw relationship and unbalanced occlusions.
3. Localisation of pressure due to variable displacement of soft tissues.
4. The use of Stockman Vine ridges and suction
discs.

5. Dimensional changes in impressions and denture
bases.

6. Irregularities in the tissue surface of the
dentures.

In this study 83 of 89 patients (93 per cent) were
considered to have dentures which could be causing excess
pressure through instability or poor occlusal relationships.
Such a figure may be thought to be high but these patients
nearly all required new dentures. Any denture must be
considered to be traumatic to some extent (Ostlund, 1953;
McMillan, 1964): dentures are, after all, foreign bodies
which interfere with the normal physiology of the mouth.
Trauma may well be associated with most if not all patients
who develop DSM, but it is also true that poorly functioning
dentures are worn by many people who do not develop mucosal
inflammations of this type. If trauma alone were the cause
it is considered likely that the mandibular alveolus would
be involved at least as frequently as the maxilla. The
force per unit area acting on the mandible is about twice
that acting on the maxilla and lesions which are definitely
associated with trauma, for example irritative hyperplasia,
are found much more commonly in the mandibular mucosa. DSM is rarely seen in the mandible (Neill, 1965).

Lyon and Chick (1957) and Lehner (1965) amongst others have suggested that chronic traumatic injury to oral mucous membrane predisposes to candidal growth and candidal infection. Thus trauma is seen as a predisposing influence rather than the causative factor.

The interrelationships between oral and general disease are well recognised (Walsh, 1956). Cheraskin (1958) referred to the mouth as the barometer of systemic disease. Oral signs and symptoms may be the first indication of a general disease or deficiency state. Even when the disease or deficiency is of low grade, local irritation in the mouth may be enough to produce complaints or visible findings in the oral tissues. Devan (1952) considered that tissue tone is an index of the circulatory and nutritional condition of the patient and that dentures do not meet biological demands when the mucosa to which they adhere shows signs of inflammation.

The relationship of microorganisms with the human host is an intricate one. Many organisms are essential to life while some are highly pathogenic: others may live as commensals in the body but in certain circumstances multiply
and give rise to disease. Such organisms are said to be opportunistic. Winner (1969), in showing that yeasts were capable of opportunism compared the yeasts to the herpes simplex virus which remains viable in human tissue for long periods and only causes lesions when there is a change in the host. A similar situation is seen in the mouth with regard to the organisms which are presumed to cause acute necrotic gingivitis. Disease due to Candida occurs in certain well-defined conditions of lowered tissue resistance or altered metabolism (Winner, 1966). In other words candidosis is a disease of the diseased and if present often leads the clinician to search for some more deep-seated condition.

It is obvious that the degree of disease varies. As Cheraskin (1953) stated:

"There is no argument that a person can be seemingly very healthy or undeniably sick. These are the blacks and whites of disease. However, too little attention is directed to the fact that there are degrees of sickness and well-being. In all probability, white (perfect health) is never attained. Black, at the other pole, represents disease in its classic form. Between these two limits are an infinite number of shades of grey ... a limitless gradation of health to sickness."

Investigations have shown that DSM and angular cheilitis may be associated with endocrine imbalance (Neill, 1961),
iron deficiency (Rose, 1968), and thiamine and riboflavin deficiency (Makila, 1969). Recent evidence suggests that conditions such as these may not be uncommon in the population at large. Multiple analysis of the blood of 296 subjects, randomly selected from amongst patients attending a general medical practice in England, disclosed iron deficiency anaemia in 40 subjects and diabetes mellitus or renal disease in a further 10. The important fact is that these abnormalities were unsuspected at the time of consultation and would not normally have been diagnosed as the blood tests would not have been requested (Carmalt, Freeman, Stephens and Whitehead, 1970). The possibility that a similar situation may be present in denture patients cannot be overlooked.

Evidence that a high carbohydrate-low protein diet is associated with DSM has been shown by Shuttleworth and Gibbs (1960), Neill (1963) and Ritchie et al. (1969).

Kimball (1954), Kim, Ringsdorf and Cheraskin (1962), Deely (1965) and Osborne (1964) have all stressed the importance of good nutrition to the health of the oral mucosa and have suggested various regimes of diet and vitamin and mineral supplementation as a means of improving
tissue tolerance in the full denture wearer. Ramsey (1970) points out that the mass of nutritional research produced to date clearly identifies the relation of severe dietary deficiencies to oral structures. However this same evidence leaves unsettled the relationship between nutrition and minor disorders of the oral mucosa.

We, in New Zealand, pride ourselves as being the best fed nation on earth and yet Bell (1969) alerts us to anaemia in children and pregnant mothers, scurvy and other diseases among members of the older age groups, alcoholism with its resultant deficiencies, and protein deficiency and other nutritional derangements in medical and surgical cases.

From the available evidence a hypothesis may be advanced. It seems likely that DSM arises from an interaction of factors, both local and systemic, rather than from any single cause. Cheraskin (1958) has expounded that the observable evidence of disease is the product of a systemic substrate and local exciting factors. The systemic substrate can never be perfect. As Dubos and Pines (1966) have pointed out:

"all living things are diseased to some extent ... the real measure of health is not the Utopian absence of disease, but the ability to function effectively within a given environment. As the environment
keeps changing, so good health is a process of continuous adaptation to the myriad microbes, irritants, pressures and problems which daily challenge man."

What we recognise as good health is maintained by a balance between many factors, and evidence of disease is seen only when this balance is upset beyond a certain level. Conditions which have been shown to affect the systemic substrate in such a way as to tilt the balance and predispose the oral tissues to DSM include iron deficiency, endocrine imbalance, nutritional deficiency and drug therapy; these may act either singly or in combination. If the departure from normal is greater than a certain indefinable amount the local factors supplied by a denture and yeast organisms may produce observable evidence of disease.

If the systemic substrate is a fairly healthy one, considerable amounts of trauma and yeast elements are necessary before disease is evident. If, however, the substrate is unhealthy much smaller amounts of trauma and yeasts will produce the same degree of disease. The worst cases can be expected when the substrate is poor and trauma and yeasts are present in abundance. This is borne out by clinical observation. Figures 9, 10, 11 and 12 show this diagrammatically.
If the product of the systemic and local factors in a patient with DSM can be reduced below a certain critical level then the lesions will heal and the tissues remain healthy as long as this level is not exceeded. That this is possible is seen in the many different forms of treatment which have been claimed as successful and in the rate of relapse, which is considered high (Cawson, 1966; Budtz-Jørgensen and Bertram, 1970b).

The above is only supposition but it appears to fit the facts and has an important bearing on treatment.

The aim in treatment must be three-fold:

1. To improve the systemic substrate by correcting deficiency and disease where possible.

2. To reduce the trauma to the mucosa by constructing dentures in harmony with the biological requirements of the tissues.

3. To reduce the yeast population in the mouth which has been shown in this and other studies to be related to the incidence of DSM.

In practical terms this means that treatment should be aimed firstly at improving the general health by investigation and correction of disease and deficiency states with particular emphasis on diet.
At the same time anti-mycotic measures should be directed both at the denture and the mucosa. This can be done effectively by total removal of the dentures for two weeks or more, but this is often unacceptable to the patient. An alternative method involves the disinfection of the dentures by overnight soaking in a fungistatic solution such as 2 per cent sodium hypochlorite or 5 per cent cetrimide together with chemotherapy if necessary. At this stage the occlusion of the dentures should be refined as much as possible. No attempt should be made to renew the dentures until the mucosa has returned to a normal healthy state but it is imperative that new dentures should be made or the old ones remodelled in such a way that they become minimally injurious to the supporting tissues. It is possible that the fitting palatal surface should be made smooth and highly polished and investigations in this direction would be of benefit. Clinical observation has indicated that some patients who have smooth, poorly adapted old dentures develop DSM when new close fitting dentures are made.

The importance of good oral hygiene should be stressed when the new dentures are inserted and the patient should be instructed to remove the dentures at night and place them in a cleansing solution.
Periodic checks should be made over the next six months to ensure that a satisfactory standard of denture hygiene is being maintained and that there has been no relapse.

Figures 9, 10, 11 and 12 (pages following). Three circles represent the factors combining to produce DSM. When these three factors are large enough to produce disease the circles overlap. Only one factorial parameter needs to be in a non-contributory status for the prevention or cure of DSM. It is important to know the status of all three parameters.
Figure 9. Substrate - good
Trauma - minimal
Yeasts - minimal
No DSM
Figure 10. Substrate - fair
Trauma - moderate
Yeast - minimal
No DSM
Figure 11. Substrate - poor
Trauma - minimal
Yeast - moderate

DSM present (mild)
Figure 12. Substrate - poor
Trauma - maximal
Yeasts - maximal

DSM present (severe)
SUMMARY

Yeast populations both in the mouth rinse and on the denture of 89 patients with and without mucosal inflammation were investigated. The cleanliness of the palatal surface of the maxillary denture of 60 of these patients was also examined.

The findings were:

1. Yeasts were found associated with the dentures of 100 per cent of the patients with DSM and 42 per cent of the patients without DSM.

2. Eighty-one per cent of those with DSM were found to have heavy yeast infestation on the denture. The figure for those without DSM was 19 per cent.

3. DSM was also associated with a heavy growth of yeasts from a single mouth rinse sample.

4. Culture from the fitting surface of the denture appeared to be a more sensitive method of demonstrating yeast elements than culture from the mouth rinse.

5. Fifty-four per cent of the patients with DSM had angular cheilitis compared with 8 per cent of the remainder.
6. DSM was more prevalent in those patients with poor denture hygiene.

7. Investigation with disclosing solution showed that few dentures were well cleaned, and that most patients found difficulty in keeping the fitting surface of the labial and buccal flanges clean.

8. *Candida albicans* was by far the most common yeast isolated but 8 other *Candida* species along with *Torulopsis glabrata* were also recovered.

A hypothesis is advanced suggesting how the lesions may arise. Guidelines for treatment are also shown.
Figure 14. Diagrammatic drawing of the palate showing sample entry.

- Yellow: mild inflammation
- Orange: moderate inflammation
Figure 15. Diagrammatic drawing of the palatal surface of the full upper denture showing sample entry. 

- areas of stained plaque
Figure 15. Diagrammatic drawing of the palatal surface of the full upper denture showing sample entry.

Areas of stained plaque
Figure 16. Diagrammatic drawing of imprint agar culture showing sample entry.

--- yeast colonies
APPENDIX

Figure 13.  (Please turn over.)

Method of recording history and examination with sample entries.
NAME: MRS D.C.H  SEX: M/F  AGE: 48 NO: 1

ADDRESS:

DATE OF EXAMINATION: 10.8.70

GENERAL HEALTH: \[ \text{GOOD} \] QUESTIONABLE:

DRUGS: YES. [NO: ] TYPE:

LOWER DENTITION: \(-/F\) \(-/P\) metal/plastic

NATURAL TEETH: [ ] NONE:

AGE OF DENTURES: [23 years] worn at night \([\text{yes/no}\]

MAIN METHOD OF CLEANING: BRUSH SOAK:

ANGULAR CHEILITIS: present/absent

EFFICIENCY OF DENTURES:

retention — stability — occlusion —

CLASSIFICATION OF D.S.M. \([I\) II III

OTHER INFLAMMATION:

DENTURE HYGIENE (visual assessment) GOOD [FAIR] POOR

DENTURE HYGIENE (disclosing solution) GOOD [FAIR] POOR

MICROBIOLOGICAL EXAMINATION:

1. Saliva sample

   No. of organisms: \[100 \text{ C. albicans}\] \(100 \text{ C. species/10ml}\) type

2. Yeasts on Denture: 0 + ++ [+++]
Figures 14, 15 and 16. Diagrammatic drawings of the palate, denture and imprint agar showing method of recording data.
STATISTICAL ANALYSIS

1. Yeast isolations from mouth rinse sample

The statistical difference in yeast isolations from the mouth rinse sample between the DSM group and the OI and N groups was assessed using a standard error test.

This involved confirmation that there was no real difference between the OI and N groups.

\[
\text{Standard Error Difference} = \sqrt{\frac{plq1}{N1} + \frac{p2q2}{N2}}
\]

\[
= \sqrt{\frac{31 \times 69}{29} + \frac{44 \times 56}{23}}
\]

\[
= \sqrt{73.75 + 107.13}
\]

\[
= \sqrt{180.88}
\]

\[
z = \frac{44 - 31}{13.45}
\]

\[
= 0.96
\]

\[p > 0.05\]

The OI and N groups were then pooled and assessed against the DSM group.

\[
S.E. = \sqrt{\frac{95 \times 5}{37} + \frac{63 \times 37}{52}}
\]

\[
= \sqrt{12.84 + 44.83}
\]

\[
= \sqrt{57.67}
\]

\[
z = \frac{95 - 37}{7.6}
\]
(contd. from p. 72) \[ = 7.63 \]
\[ p \ < 0.01 \]

2. **Yeast isolations from the denture**

The statistical difference between yeast isolations from the dentures in the DSM group and the (OI + N) group was assessed using the standard error test.

\[
S.E. = \sqrt{\frac{42 \times 58}{52}} + 0
\]
\[
= \sqrt{46.84}
\]
\[
= 6.84
\]

**Observable difference** \[ = 58 \]

\[
Z = \frac{58}{6.84}
\]
\[
= 8.48
\]
\[ p \ < 0.01 \]
3. **Presence of angular cheilitis**

The statistical difference in the presence of angular cheilitis in the DSM group and the (OI + N) group was assessed using the chi square test with Yates correction.

<table>
<thead>
<tr>
<th></th>
<th>present</th>
<th>absent</th>
</tr>
</thead>
<tbody>
<tr>
<td>DSM</td>
<td>20</td>
<td>17</td>
</tr>
<tr>
<td>(OI + N)</td>
<td>4</td>
<td>48</td>
</tr>
<tr>
<td></td>
<td>24</td>
<td>65</td>
</tr>
</tbody>
</table>

\[
x^2 = \frac{\sqrt{(48 \times 20) - (17 \times 4) - \frac{89}{2}}^2 x 89}{24 \times 65 \times 52 \times 37}
\]

\[
= \frac{\sqrt{960 - 68 - 44.5}}{3001440}^2 \times 89
\]

\[
= 21.29
\]

\[
6.635 < 21.29
\]

\[
p < 0.01
\]
4. **Denture hygiene by visual assessment**

The statistical difference between the denture hygiene of the DSM group and the (OI + N) groups was assessed using Brandt and Snedecors formula

<table>
<thead>
<tr>
<th>Good</th>
<th>Fair</th>
<th>Poor</th>
</tr>
</thead>
<tbody>
<tr>
<td>DSM(z)</td>
<td>5</td>
<td>17</td>
</tr>
<tr>
<td>(OI + N)(b)</td>
<td>25</td>
<td>18</td>
</tr>
<tr>
<td></td>
<td>30</td>
<td>35</td>
</tr>
</tbody>
</table>

\[
x^2 = \frac{\sum a_i^2 - \frac{A^2}{N}}{K(1 - K)}
\]

where \( K = \frac{A}{N} \)

\[
= \frac{\frac{25}{30} + \frac{289}{35} + \frac{225}{24} - \frac{1369}{89}}{\frac{37}{89} \times \frac{52}{89}}
\]

\[
= (0.83 + 9.26 + 9.38) - 15.33
\]

\[
= 12.86
\]

Degrees of freedom = \((\text{rows} - 1)(\text{Columns} - 1) = (2-1)(3-1) = 2\)

12.86 > 9.21

i.e. \( p < 0.01 \)
5. Yeasts isolated from dentures and method of cleaning

The statistical difference in yeasts isolated from those who soaked their dentures and those who did not was assessed using the chi square test with Yates' correction.

<table>
<thead>
<tr>
<th>Yeasts Present</th>
<th>Yeasts Absent</th>
</tr>
</thead>
<tbody>
<tr>
<td>SOAK</td>
<td></td>
</tr>
<tr>
<td>3</td>
<td>13</td>
</tr>
<tr>
<td>Did not SOAK</td>
<td></td>
</tr>
<tr>
<td>56</td>
<td>17</td>
</tr>
<tr>
<td>59</td>
<td>30</td>
</tr>
</tbody>
</table>

\[
x^2 = \frac{\left(56 \times 13\right) - (17 \times 3) - \frac{89}{2}}{59 \times 30 \times 73 \times 16} x 89
\]

\[
= \frac{(728 - 51 - 44.5)^2}{2067360} x 89
\]

\[
= 17.22
\]

\[
p < 0.01
\]
SELECTED BIBLIOGRAPHY


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