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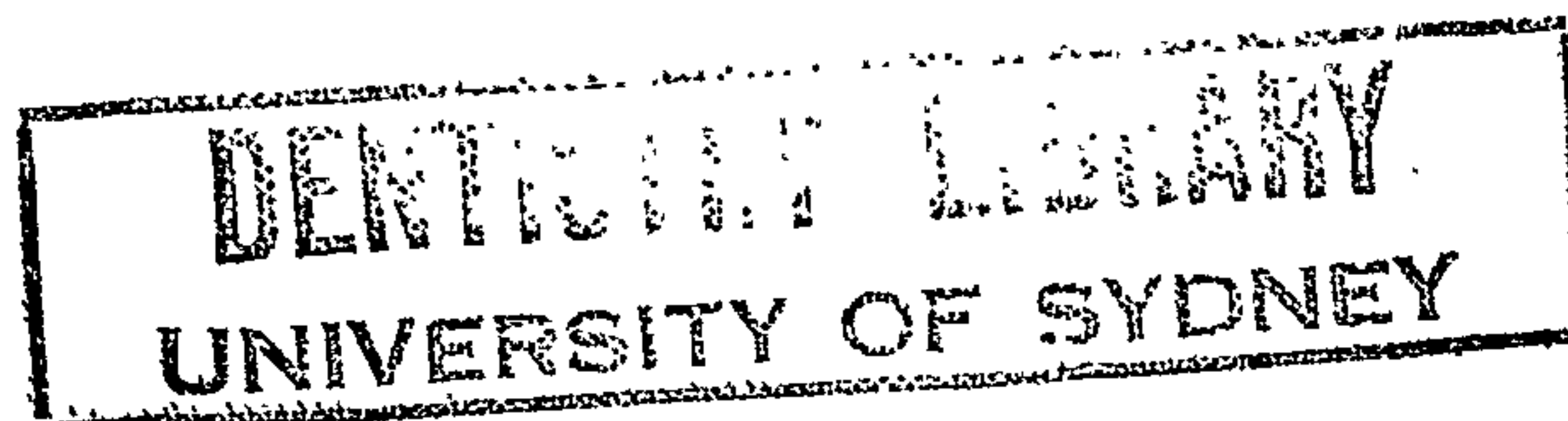
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FLUORIDE and MALOCCLUSION

An Epidemiological Study of Malocclusion
in Tamworth 1963, 1973 & 1988, and Lismore 1989.

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INTRODUCTION

This thesis attempts to test the role of fluoride in preventing malocclusion(s).

Current knowledge is presented on:

- a) the modes of action of fluoride;
- b) the effectiveness of fluoride in reducing dental caries in both the deciduous and permanent dentitions;
- c) the recent secular trend in decay reduction.

The fluoridation of *Tamworth, N.S.W.* is discussed. Past and recent results of *Tamworth* fluoridation are presented.

The problems of defining and recording malocclusions are discussed. Special reference is made to recent indices of malocclusion that have been devised; and critical appraisals are made of the methods used to register malocclusions in Malocclusion Surveys at *Tamworth and Lismore*.

The effects of caries, and premature loss of deciduous and permanent teeth on the occlusion are discussed. From considering these, the reader should be awakened to the possibilities presented by fluoride for preventing malocclusion.

The results of past and recent studies on the role of fluoride in preventing malocclusions are presented. Special references are made to the results of the *Tamworth Malocclusion Surveys*, pre-fluoridation (1963), and continuing-fluoridation (1973) and (1988). The results from the *Lismore Malocclusion Survey* (1989) are presented to serve as a contemporary control.

PART I - FLUORIDE

(i) AIMS OF PART I

Part I of this thesis is intended to give:

- a brief overview of fluoride's occurrence in Nature and its metabolism by humans. This allows assessment of the distribution of ingested (systemic) fluoride throughout the body and especially as it relates to the developing deciduous and permanent dentitions.
- contemporary findings on the mode of action of fluoride in reducing dental caries, especially since several of these modes of action were only conjecture at the time of *Peel's thesis (1973)*.
- a review of all literature available to this author on the role of fluoride in altering tooth morphology. The orthodontic implications of this will be discussed fully in Part II.
- current results of the effectiveness of fluoride, in its various forms and vehicles, in reducing caries.
- an examination of the secular trend in decay reduction that occurred from the late *1970s*. The probable explanations for this are presented. The possible implications of this trend for malocclusion survey results and their interpretation will be discussed further in Part II.

- a discussion of the TAMWORTH FLUORIDATION SURVEYS. Results of DMFT recordings from past and recent surveys are presented, along with the results for *Lismore 1989*.

(ii) Introduction

Prevention of tooth decay is based on 3 concepts:

1. Reducing plaque by chemical or mechanical means;
2. Modifying or controlling diet by urging less intake of fermentable carbohydrates;
3. Increasing tooth resistance.

History shows that option 3. is the most easily achieved. This is by the use of fluoride in one form or another.

The history of the discovery of the link between fluoride and increased caries resistance is beyond the scope of this thesis. It has been dealt with adequately in several overviews on fluoridation, e.g.

<i>Silverstone</i>	(1978)
<i>Mellberg and Ripa</i>	(1983)
<i>American Dental Association</i>	(1989)

Likewise, the chemistry and micro-anatomy of the process of dental caries shall not be covered in this thesis. Good overviews on this subject can be found in *Silverstone (1978); Mellberg and Ripa (1983)*.

Sources of Fluoride

Fluoride is a common compound in Nature, it ranks 13th among the elements in the order of abundance in the Earth's crust. Since fluoride compounds are usually soluble in water, fluoride is commonly found in natural water in varying amounts. Additionally, many foods may be rich in fluoride. Tea, fish, meats, fowl, wheat, rye and rice all contain fluoride. Thus is it commonly ingested. (*WHO, 1984 p25*).

Fluoride Metabolism

Fluoride is readily absorbed into the gastro-intestinal tract and into the bloodstream. (*WHO, 1984 p57*); (*Mellberg and Ripa, 1983 p83*).

Approximately 5-10% of fluoride ingested is not absorbed and exits via the faeces (*WHO, 1984 p37*).

Fluoride not utilised by bone or teeth is excreted in urine and a small amount in the sweat (*WHO, 1984 p44*).

Salivary and gingival fluid level is low and varies with blood plasma fluoride levels. It is usually 65% of blood plasma level of fluoride (*WHO, 1984 p44*).

Fluoride crosses the placenta. The fluoride content of the foetal skeleton and teeth increases with foetal age, and the concentration of fluoride in the drinking water consumed by the mother (*WHO, 1984 p44*).

Levels in human breast milk are similar to those of blood plasma and are thus not considered a major source of fluoride (*WHO, 1984 p4*).

The use of fluoride in preventing dental caries involves 2 categories:

(a) Systemic use of fluoride

This involves ingestion and absorption of fluoride into the bloodstream and its passage to areas where teeth are developing. Here it is incorporated into the enamel of these teeth. In the case of erupted teeth, systemic fluoride is excreted via the saliva and gingival fluid and thus exerts a topical effect as well. Vehicles used for the systemic administration of fluoride are:

- community water fluoridation;
- school water fluoridation;
- daily fluoride tablets or drops;
- salt fluoridation;
- milk fluoridation;
- juice fluoridation;
- flour fluoridation

These will be discussed further below.

(b) Topical use of fluoride

This is the application of various forms of fluoride directly onto the erupted tooth surface.

Systemically administered fluorides also have a certain topical effect as they are swallowed. This effect is especially noted on incisor teeth (*Arnold, 1943*).

The topical application of fluoride is generally by way of various fluoride solutions or gels used as:

- mouthrinses
- professional applications
- varnishes
- dentifrice

These will be discussed further below.

(iii) Mode of Action of Fluoride

The current thoughts on the anticaries mechanisms of fluoride are that fluoride exerts its effects through systemic as well as topical means:

- a) Increased enamel resistance
- b) Increased rate of maturation posteruptively
- c) Remineralisation of incipient lesions posteruptively
- d) Altered tooth morphology
- e) Antibacterial and Intra-oral ecological changes

(a) Increased Enamel Resistance

This is the traditional view of the role of fluoride in increasing caries resistance. Fluoride combines with the hydroxapatite crystal to form fluorapatite. Fluorapatite has been shown *in vitro* (Mellberg and Ripa, 1983 p41) to be less soluble in acid. Thus it was thought that greater amounts of fluorapatite meant greater solubility reduction.

The improvement in crystallinity of apatite to fluorapatite produces a thermodynamically more stable crystal. This imparts greater resistance to acid dissolution and less impurity of structure. Horowitz (1982) and Beltran and Burt (1988) also state that the crystal is slightly bigger. This may affect tooth morphology to be discussed further below.

Mellberg (1977) showed that systemically the tooth acquired fluoride at several times during its formation. During its very early formative period the fluoride concentration may be relatively high but as the tooth matures and begins to mineralise, the fluoride concentration falls. Then follows a period of mineral deposition frequently referred to as pre-eruptive maturation. At this point the enamel becomes more completely mineralised. This is the period of greatest enamel

acquisition of fluoride. This amount will vary with the fluoride concentration of food, water and fluoride supplements. It varies too, with the duration of the pre-eruptive maturation phase.

Therefore, deciduous teeth have a lower fluoride concentration than permanent teeth because they have a shorter period of enamel mineralisation. Differences in fluoride concentration of permanent teeth from similar fluoride areas have been attributed to differences in the length of the pre-eruptive maturation phase (*Mellberg and Ripa, 1983 p88*).

As mineralization proceeds, less fluoride is able to penetrate and thus fluoride deposition becomes increasingly confined to the surface layers. Thus the surface layers show much higher concentrations than interior layers (*Mellberg, 1977; Mellberg and Ripa, 1983 p88*).

Despite enamel being up to 1 mm thick, high fluoride concentrations are normally only found in the outer 30 - 50 μ m.

Some recent studies (*Schamschula et al, 1982; Mellberg et al, 1985*) have detected only slight differences in enamel fluoride concentration between fluoridated and non-fluoridated areas.

It has been felt by observers (*Isaac et al, 1958; Englander and Mellberg, 1976*) that the higher fluoride content in enamel alone could not explain the differences between caries experience in fluoridated and non-fluoridated areas.

The theoretical concentration of fluoride in pure fluorapatite to yield substantial acid solubility is around 38,000 ppm. However, fluoride levels at 2 μ m are only 2,200 - 3,200 ppm in water-fluoridated areas of 1 ppm fluoride (*Aasenden, 1974*). This level

approached 4,800 ppm where the fluoridated water level is 5-7 ppm fluoride (*Aasenden, 1974*). These amounts have even been reported from subjects from low fluoride areas.

DePaola et al (1975) and Mellberg (1977) stated that only 16% of the caries variation between fluoride and non-fluoride areas could be attributed to enamel fluoride. However, the studies reported below confirm the caries-protective effect of systemically acquired enamel fluoride when subjects born and reared in fluoride communities moved to non-fluoride communities.

Up to 20 years' protection has been found by one researcher (*Deatherage, 1943*) and another study (*Isaac et al, 1958*) reported several years' protection.

(b) Increased Rate of Maturation

Post-eruptive enamel maturation is a normal process. Newly erupted enamel has areas of incomplete mineral deposition. These hypomineralised areas become more fully mineralised in the mouth following eruption. Sources of mineral elements include saliva and gingival crevice fluid. These two sources may also thus supply fluoride from ingestion. Additionally, topically applied fluoride, including fluoride toothpaste and mouth-rinsing can be included here. Note also that fluoridated water may also exert a topical effect prior to ingestion (*Mellberg and Ripa, 1983*).

Even very small concentrations of fluoride in solution enhance mineral deposition (*Backer-Dirks, 1966*). In the presence of fluoride the hypomineralised tooth may incorporate more fluoride and become mature within only a few months after eruption. This newly exposed enamel can acquire more fluoride than older enamel and can thus become more resistant to decay. Thus the ready access of newly erupted teeth to fluoride even in tiny amounts, seems to result in greater caries protection than pre-eruptive fluoride incorporation alone. (*Mellberg, 1977; and Mellberg and Ripa, 1983*). This reinforces the importance of topically applied fluoride at this stage, whether it be due to salivary or gingival fluid sources from ingested fluoride, or from fluoride toothpaste, fluoride mouth-rinsing or professionally applied fluorides.

High fluoride concentrations, high temperatures and low pH, all encourage greater uptake of fluoride by enamel during this post-eruptive maturation stage (*Mellberg, 1977*).

Deciduous and permanent teeth behave similarly in this phase. However, penetration appears to be less in permanent teeth where a greater

period of pre-eruptive fluoride incorporation equates to less potential for fluoride uptake topically, post-eruptively (*Mellberg, 1977*).

(c) Remineralisation of Incipient Lesions

Current opinion (*Horowitz, 1983; Mellberg and Ripa, 1983; Beltran and Burt, 1988*) favours this mechanism as perhaps the greatest contribution of fluoride to caries resistance.

Put simply, the process involves both hypomineralised or demineralised enamel. This enamel, in the presence of low concentrations of fluoride, will become remineralised. This remineralisation imparts not only a redeposition of mineral but enamel may actually become more fluoride-rich than before and thus show increased caries resistance.

Saliva performs this function naturally but the process is more efficient in the presence of fluoride.

This fluoride, as already mentioned, may derive from low concentration sources such as saliva and gingival crevice fluid (both derived from systemic fluoride uptake). Fluoride may also come from the direct topical effect of swallowing fluoridated water, or from higher concentration agents such as fluoride toothpaste, gel or mouthrinse. *Mellberg (1977)* feels that the process is likely related to the ability of traces of fluoride to cause precipitation of fluorapatite from supersaturated solutions. Alternatively, it may relate to the ability of porous enamel to accumulate fluoride more readily than sound enamel (as mentioned for the post-eruptive maturation of enamel).

Plaque pH fluctuates from above 7 to below 5 due to intermittent acid production, e.g. after meals. When the pH of plaque falls below 5.5, enamel dissolves. If no additional acid is supplied, the pH rises and any dissolved mineral leached out will reprecipitate. When fluoride is absent the reprecipitated mineral may be more soluble than the

original enamel. Where fluoride is present initially, the enamel is much less likely to dissolve in the first instance (*Beltran and Burt, 1988*). However, in the presence of fluoride (from whatever source) the precipitated mineral is more of the fluorapatite type. This requires a lower pH for dissolution and thus means that the reprecipitate may be more caries resistant than originally. Additionally, if this enamel is demineralised again, the free fluoride released will enhance reprecipitation in the fluorapatite form again. If further fluoride is present, the new enamel remineralised may be even more fluoride-rich. *Beltran and Burt (1988)* also state that the rapidity of remineralisation is also increased.

On the macro scale, experimentally-induced white spot carious lesions have been shown to be easily remineralised *in vivo* if fluoride mouthrinses are used (*Von der Fehr et al, 1970*).

Additional evidence is shown by the fact that there are higher enamel fluoride levels near the gingival margin where plaque levels are highest (*Weatherell et al, 1972*).

In a further example *Beltran and Burt (1988)* state that the fluoride content of highly porous or previously "*primed*" enamel, such as found in recently erupted enamel or "*white spot*" lesions, is much higher than in adjacent sound enamel. When these lesions are subjected to a slightly acidic environment, mineral dissolves from subsurface regions of the sound enamel but not from the remineralised areas.

To promote optimal remineralisation, fluoride should be present at the time of acid attack. A continuous presence of fluoride in the immediate area of activity is ideal.

With prolonged exposure to topical fluoride a "*reservoir*" of CaF_2 may

be formed. This may slowly dissolve to provide further fluoride for remineralisation enhancement. *Mellberg, (1977)* feels that the amount of fluoride in the reservoir will probably depend not only on the frequency of the caries attack, but also on the frequency and concentration of fluoride exposure and the thickness of the overlying plaques.

With the large reservoirs (due to high fluoride concentrations delivered) it is conceivable that released fluoride could produce a bacteria-suppressing feedback effect that would shorten the attack or moderate the pH drop produced. This possible effect will be further discussed below.

Beltran and Burt (1988) state that because frequent low levels of fluoride are essential to this process, then the role of water-fluoridation in this process is very important. Likewise fluoride toothpastes are probably the main agent for fluoride supply for this process in non-water-fluoridated communities.

Unfortunately, the additive effect from combinations of fluoride sources has received little attention in studies (*Mellberg and Ripa, 1983*) and is a very important topic.

(d) Altered Tooth Morphology

Many studies have confirmed that the pits and fissures, especially those of the occlusal surfaces of molars, are the most susceptible to dental caries (*Bossert, 1933; Cooper and Ludwig, 1965; Craig and Powell, 1979; Bagramian et al, 1979; Shaw and Murray, 1980; Ripa, Leske and Varm, 1988*).

Teeth with narrower, deeper fissures are more caries prone than teeth with shallow, wide fissures (*Bossert, 1933; Grainger et al, 1966; Mellberg and Ripa, 1983 p66*).

Research has been performed by many authors into the effect of fluoride on tooth morphology. No conclusive results have been reached on this subject. Current research on this aspect of fluoride action is very minimal and most results date to the early *60s and 70s*.

Previous experiments involved examination of changes in occlusal morphology. Additionally, actual tooth size has been assessed both buccolingually and mesiodistally. One of the problems associated with experiments carried out on this subject is that some were performed on rats (*Paynter and Grainger, 1961; Kruger, 1964*) and thus the problem of extrapolating to the human situation is immediately evident.

Malherbe and Ockerse (1944) noted that teeth from high fluoride areas were lustrous and light-cream coloured. Cusps were low and flat. Grooves and fissures were shallow. None were structurally defective. In contrast, teeth from low fluoride areas were dull, white and chalky. Cusps were high and sharp. Grooves and fissures were deep and often structurally defective.

In 1954 the *Division of Dental Research at the University of Toronto* published early findings of a study involving the measurements of

teeth from a fluoride endemic town, Mt. Forest. These measurements from native-born townschilren were compared with those who immigrated there from non-fluoride areas.

They found females had smaller teeth than males. Additionally they found that the teeth of Mt. Forest-born schoolchildren were smaller buccolingually and mesiodistally and had shallower fissures. This agreed with earlier findings of theirs on rats fed high-fluoride diets.

No subject numbers were mentioned and a subsequent publishing of full results has not been found by this author.

Wallenius (1959) contended that, because of tooth size/basal bone size discrepancies, this link was important for orthodontic reasons. This author will explore this point further later in this thesis, based on the general findings of all authors in this section.

Wallenius felt that despite heavy genetic determinants in tooth size, there was a chance that environmental factors could alter tooth size. She concluded from others' observations of fluorine poisoning that there were changes in tooth size and form. Her study involved measurements on plaster casts of the mesio-distal widths of central incisors and first molars. Measurements were of 419 children from varying fluoride level experiences. Her results showed that above 0.6 ppm fluoride level, the widths of teeth increased and this was especially marked beyond 1 ppm.

She acknowledges that fluoride may not have been the only mechanism at work. Further mention of the secular trend for tooth size increase of children over parents will be elaborated on later in this thesis. This trend may lead us to question *Wallenius'* results. *Wallenius'* results

are in a direct opposition to those of *Omar (1987)*, yet to be presented. Wallenius felt the size increase might be attributable to fluoride "*stimulating the organism and/or salivary depositions of fluoride*". An increase of 1.7% in width was observed. Fluoride levels beyond 1 ppm yielded little further width change.

Paynter and Grainger (1961) reviewed some of their findings concerning caries-susceptible rats. Their molars were of a different shape to resistant rats. They reported also on the experimental feeding of rats at 12 ppm fluoride level from conception. Measurements were made from photographs of: total size, diameter at the cervix, and the shape and size of the mesio-occlusal fissure.

Such rats had smaller molar teeth than controls. They also mention from previous observations, that children from fluoride areas with larger teeth generally are more prone to pit and fissure caries than children with smaller teeth.

Kruger (1964) researched pre-eruptive and combined pre-eruptive/post eruptive effects of fluoride on rat molars. Fluoride-treated rats had teeth with flatter cups, wider and shallower fissures. Lower first molars also tended to be smaller mesiodistally but not significantly so. He thus concluded that fluoride was capable of altering tooth morphology. Additionally, caries tests were performed between control and experimental rats. Reduced caries was found in these experimental rats with altered tooth morphology.

Cooper (1964) and *Cooper and Ludwig (1965)* carried out a study on 100 girls in a fluoride area and 100 girls in a saline gley soil area. Results were compared with 100 controls. Bigonial and bizygomatic widths were measured. Additionally, casts were made of teeth and

mesiodistal and buccolingual widths and cusp heights were measured on lower first permanent molars. No skeletal measurements were different statistically between the 2 groups. The mesiodistal widths of the lower first permanent molars in children exposed to fluoridation were significantly smaller by 0.21 mm mean value. One could thus imply a potential for less dental arch crowding. Buccolingual widths were smaller but not significantly so. This result agrees with previous animal studies. Cusp heights were also reduced in children from the fluoride area.

These authors also contend, that, due to the greater bonding energy of fluoride in the fluorapatite crystal over hydroxyl groups, there would be a decrease in the crystal lattice size. They cite *Minder (1938)* as supporting this in his experiments on bone ash after fluoride administration. However, *Horowitz (1982)*, and *Beltran and Burt (1988)* both mention a larger crystal formed when fluoride substitutes in the hydroxyapatite crystal.

Kruger (1966) and *Adkins and Kruger (1966)*, with further studies using rats, demonstrated further decreases of mesiodistal widths of rat molars in rats with fluoride diets (however this difference was not significant). Fluoride was shown to significantly decrease enamel thickness, and to increase the width of occlusal fissures.

Möller (1967) notes that teeth from fluoride areas were larger and well-formed. Cusps were lower and rounded and rather flat. Occlusal sulci were shallow and grooves and fissures were shallow.

Lovius and Goose (1969) examined maxillary and mandibular dental casts of 258 ten-to-eleven year-old boys. The subjects in the groups came from fluoride and non-fluoride areas respectively, since birth. The

control group came from an area in close proximity to the experimental group, thus eliminating many differences possible between the 2 groups due to a different racial pool.

Teeth measured were 21, 32, 26, 36. Incisors were only measured mesiodistally, but molars were measured buccolingually as well. Mean cusp heights were calculated. The fluoride group showed larger 21s mesiodistally but 32s were smaller mesiodistally. The measured differences were not statistically significant.

All molar measurements showed smaller measurements in the fluoride group with respect to buccolingual and mesiodistal widths and cusp heights. This is a suggestive finding even though the results fail to reach accepted levels of significance.

Simpson and Castaldi (1969) reported their findings from the measurement of models of mandibular and maxillary first permanent molars. Controls came from towns with only traces of fluoride in the water supply; experimental subjects came from a nearby town where the water supply contained 1-2 ppm fluoride. Measurements made were of mesiodistal width, buccolingual width, height of the mesiobuccal cusp above the gingival margin. Additionally, male and female results were kept separate. Racial background was uniform. Control teeth numbered 79, and experimental teeth, 81.

Their results showed that the dimensions of maxillary and mandibular molars in the optimum-fluoride sample are consistently greater than those from the low-fluoride sample. However, only the mesiodistal width of the first permanent mandibular molar in the optimum fluoride group is significantly greater. Another significant finding was that male 5-cusped mandibular molars from the fluoride sample were larger

mesiodistally than the corresponding ones from the low fluoride sample ($P = 0.05$).

Angles at the bases of fissures were consistently more acute in the low-fluoride sample. This was significant in only one group. There appeared to be a trend toward a smaller inter-cusp tip distance in low fluoride groups, but no statistical significance was reached.

In their discussion, they mention that the size of the fluorapatite crystal is larger than hydroxyapatite and may thus be responsible for the size increase found in mandibular molars from fluoride areas. They cite *Posner (1965)* as supporting this. As mentioned earlier, *Horowitz (1982)*, and *Beltran and Burt (1988)* also support this contention. As mentioned, *Cooper and Ludwig (1965)* cite *Minder (1938)* as finding a smaller sized fluorapatite crystal.

The trends toward deeper fissures and smaller angles between cusps of the teeth in low fluoride groups agree with the findings and thoughts of *Paynter and Grainger (1959)*; *Kruger (1962)*; and *Cooper and Ludwig (1965)*. The trend toward wider spacing of cusp tips in the teeth of the optimum fluoride sample would seem to be consistent with the larger intercuspal angles.

Simpson and Castaldi (1969) finish by cautioning that results of significance here should not be taken as indicating fluoride as the only element at work. Possibly other unmeasured elements could also have elicited these changes. *Kruger (1964)* and *Cooper and Ludwig (1965)* both showed that other elements or compounds may also affect tooth morphology. *Simpson and Castaldi* concluded that the importance of fluoride is minimal in effecting a less caries-prone tooth through morphology changes.

Mercado and Bibby(1973) in experiments on rats, tested the effects of trace elements including fluoride, on molar morphology. They concluded that fluoride, but in addition strontium and yttrium, administered during tooth formation will produce changes in molar morphology. Strontium yielded wider fissures and thicker dentine. Yttrium yielded similar but less pronounced effects.

Aasenden and Peebles (1974) surveyed the effects of fluoride supplementation from birth on human deciduous and permanent teeth.

Their Group I had fluoride liquid as a dietary supplement since birth, Group III had consumed 1 ppm fluoride water from birth and Group II had received no dietary fluoride. It was found that Group I participants had had higher fluoride intake than Group III. Group I had 47% of subjects showing atypically shallow pits and fissures. Group III showed 23.9% with such pits and fissures while Group II showed only 4.3% with shallow pits and fissures. This finding occurred in both deciduous and permanent dentitions. Aasenden and Peebles concluded that fluoride had an increasing effect on morphology with increased levels of fluoride intake. It should be noted that scores assigned to teeth in the different groups were made on a fairly subjective basis using explorer "feel".

Brook (1974) studied the prevalence of dental anomalies in 11-14 year-old children who were residents of fluoride (0.8 ppm) and non-fluoride areas respectively. He found no difference in the prevalence of supernumerary teeth, hypondontia, invaginations and mega/microdontia between fluoride and non-fluoride areas. He also mentions that this effect is confirmed also by the U.S Evanston and Oak Park study on the same subject. Brook conjectured that since previous studies on

tooth morphology effects of fluoride had yielded little difference between fluoride and non-fluoride subjects, this study would show similar results. This conjecture was confirmed. Brook cautioned also, that studies of the effect of fluoride on tooth morphology were hindered by inter-population variations in tooth size and the possibility of other factors at play other than fluoride. Additionally errors of measurement were likely.

Grahnen et al (1974) compared mesiodistal widths of teeth on plaster casts. The 213 subjects of the control group came from an area with less than 0.1 ppm fluoride in the water supply. The 151 experimental subjects came from an area with 1-2 ppm fluoride in the water supply. Ages of subjects ranged from 8-11 years.

Results showed no statistically significant difference between the teeth from fluoride and non-fluoride areas could be found with respect to mesiodistal widths. Those with enamel fluorosis (i.e. excess fluoride intake) likewise showed no statistically significant difference compared with controls. Thus they felt that "*optimal*" fluoride concentrations had no effect on the mesiodistal width of human teeth. It is also interesting to note that enamel fluorosis which is normally expected at ≥ 2 ppm fluoride levels was found at significant levels at ≥ 1.2 ppm fluoride. 31% of children with fluoride consumption between 1.0 - 1.2 ppm showed enamel fluorosis.

Goose and Roberts (1979) discussed the confirmed secular trend for increased tooth size of children over their parents. They used this information to design their experiment. They measured buccolingual widths and mesiodistal widths of the teeth of children who had consumed fluoridated water from birth. These measures were compared with measurements of the buccolingual and mesiodistal widths of the

teeth of their parents who had received no fluoride.

Results showed only 9 out of 28 measurements where the offspring had larger teeth in the buccolingual dimension. Half of the offsprings' teeth, only, were larger in the mesiodistal widths. They concluded therefore that the secular trend had been masked by the effect of fluoride on tooth morphology. They also backed this with comparisons of children who had received fluoride from birth on the island of *Anglesey*. The measurements of these children were compared with those of children from the nearby unfluoridated mainland. In 33 out of 56 comparisons, the *Anglesey* childrens' teeth were smaller. Additionally, nearby *Liverpool* (unfluoridated) showed good agreement with the secular trend for children to have larger teeth than their parents. In 123 families tested, anterior teeth were significantly larger in the children tested.

Omar (1987) compared mesiodistal and buccolingual tooth widths of 6-7 and 14-15 year-old schoolchildren. Subjects from one group had consumed water with 2.5 ppm fluoride throughout life. The other subjects living in an adjacent area had consumed water with 1.06 ppm fluoride throughout life. He hypothesised that increasing fluoride levels above optimal would result in a reduction in crown size.

Significantly smaller maxillary central incisors mesiodistally were found. However, mandibular incisors showed no significant differences mesiodistally. Significant differences were also found for maxillary and mandibular second premolars ($P < 0.05$). However, mesiodistal measurements of maxillary and mandibular molars showed no significant difference between high and normal fluoride consumption.

Omar highlighted difficulties in such studies involving accurate,

reproducible measurement from study casts. Also mentioned was the difficulty of attributing the results obtained to fluoride alone. He also notes that intraoral measurements are less accurate than measurements from models.

Omar concludes from his study that there is a tendency for a higher level of fluoride to produce a correspondingly greater reduction in tooth dimensions as compared with an optimum level Maxillary central incisors and second premolars showed significant reductions in mesiodistal dimensions in the higher fluoride sample. This disagrees with the findings of *Lovius and Goose (1969)*, who found larger maxillary central incisors in an optimum fluoride level community.

On the basis of his present overall results, however, Omar concludes that an inverse relationship does not exist between crown size and the level of fluoride in drinking water.

Some of the studies mentioned above should be read in conjunction with the two articles: *Garn et al (1968)* and *Lavelle (1972)*. Both studies have shown that a secular trend exists between the tooth sizes of parents and their offspring. In general, offspring have significantly larger teeth than their parents. Lavelle attributes this to the environmental effect on genetic expression. Thus, some of the findings of studies previously mentioned may have had this mechanism working as an unknown factor affecting their results. In fact, *Goose and Roberts (1979)*, as mentioned, structured their study around this trend.

Lavelle, and Garn et al fail to comment on whether there is an accompanying increase in skeletal dimensions as well, as part of this

trend. The tooth size/basal bone size implications are obvious with respect to crowding potential being increased if there is not an accompanying skeletal size increase. That is, offspring should show a greater potential for crowding.

The orthodontic significance of the possible effect of fluoride on tooth morphology and thus malocclusion will be explored further in the section on malocclusion to follow.

One important point which should be made at this stage is that the consumption of optimally fluoridated water yields no significant increase in the frequency of dental anomalies of number, form or size (*Brook, 1975*).

For any effects to occur with respect to tooth morphology, it is necessary to have consumed fluoride systemically.

(e) Antibacterial and Intraoral Ecological Changes

Möller (1965) stated that teeth, in areas with fluoridated water, look cleaner. Presumably from less plaque accumulation.

Additionally, *Birkeland (1972)* found less plaque accumulation in children using fluoride mouthrinses.

Aasenden and Peebles (1974) also found that, in their study, the children receiving regular fluoride supplements had lower plaque scores than the group exposed only to fluoridated water. This latter group, however, had a lower plaque score than the non-fluoride control group.

Aasenden and Peebles tried to explain this effect in terms of an elevated fluoride level in adjacent enamel causing a lowering of plaque accumulation. If this were so, they proposed this as another possible caries-preventive effect of fluoride. Alternatively, they postulated that the differences between the fluoride-supplement, fluoridated-water and control groups, could be related to more conscientious oral hygiene in the fluoride-supplement group due to increased parental interest which was part of the interest involved in the regular intake of fluoride supplements.

Mellberg (1977) feels that there is a distinct bacteria-suppressing effect from fluoride adsorbed onto enamel, and also from fluoride in solution in saliva and crevicular fluid.

A *World Health Organization (1984)* summary of the anticaries effects of fluoride supports antibacterial effects by way of retarding plaque growth. Additionally, enzymes are affected which causes a decrease in acid production and extracellular polysaccharides.

Mellberg and Ripa (1983) state that an interference with the metabolism of micro-organisms has long been a proposed possible mechanism of the topical effect of fluoride.

Studies on this subject have dealt with single bacteria, salivary sediment and plaque itself.

Evidence from Saliva

Salivary fluoride may derive from salivary gland secretions or from food, water or other fluoride-containing materials such as mouthrinses or dentifrices.

Normal salivary fluoride levels due to fluoride ingestion are low (0.02 ppm fluoride). Topical doses of 12,000 ppm fluoride as acidulated phosphate fluoride solution yield 100 ppm fluoride level in saliva after 5 minutes. This level falls rapidly, but at 10 hours is approximately 0.25 ppm fluoride and normal levels may not be reached for up to a week (*Aasenden et al, 1968*).

On the other hand, high concentration fluoride mouthrinses show very quickly diminishing levels in saliva after 5 minutes (*Aasenden et al, 1968*).

Fluoride can affect salivary micro-organisms in several ways. At low concentrations it inhibits acid production especially at low pH when demineralisation is favoured. Increasing concentrations of fluoride affect growth and metabolism, and very high concentrations are lethal (*Bibby and van Kesteren, 1940; and Shiota, 1956*).

At pH 5 only 6 ppm F are needed to prevent acid formation from glucose. At lower concentrations, acid formation is partly inhibited (*Jenkins, 1959*).

It has been proposed that undissociated molecules of HF which are in high concentration at acid pH, produce the antibacterial effects. However, atmospheric conditions also determine the efficiency of fluoride effects. 200 ppm F in saliva under anaerobic conditions, inhibits bacterial metabolism by 40%. Under aerobic conditions there is no effect (*Roberts and Rahn, 1946; Moland and Hartles, 1966*).

The fluoride effect can be related to its inhibition of the enzymes enolase and phosphoglucomutase. Also suggested is the interference with the transfer of glucose across the cell wall (*Sandham and Kleinberg, 1969, 1973*).

In vivo studies to support this antibacterial effect of fluoride involve:

- a) Lactic acid formation was reduced for 10-20 minutes by 0.1 - 0.5% NaF mouthrinses (*Ericsson and Hellstrom, 1952; and Hellstrom, 1960*).
- b) The number of viable microorganisms in whole saliva diminished for several hours after a 0.1 to 0.5% SnF_2 mouthrinse (*Andres et al, 1974*).
- c) Brushing with 0.1% MFP dentrifice reduced salivary streptococci numbers (*Woods, 1969*).
- d) Topical 1% NaF decreased lactobacillus levels by 5/6. This effect lasted to some degree up to 24 days (*Kozouskova et al, 1967*).

Evidence from Plaque Studies:

As with salivary effects, the effect of fluoride in plaque is related to its concentration in plaque, and the length of time it is present.

In non-fluoride consumers plaque levels of fluoride may approach 3.5 - 5 ppm F, whereas fluoride consumers may show plaque levels of 10-14 ppm F. This is far higher than salivary levels and demonstrates a fluoride-concentrating effect by plaque.

However, most plaque fluoride is not free (only 0.08 - 0.8 ppm F in plaque fluid). Remaining fluoride appears to be bound to metal ions, dialysable material and possibly bacterial cell walls and interstices.

Thick plaque generally has a lower fluoride concentration than thin plaque.

The use of fluoride-containing compounds topically will increase plaque fluoride levels and this effect may still be present up to 24 hours later. Amine fluorides keep plaque fluoride level more elevated than do sodium fluorides.

Mellberg and Ripa (1983) cite one report *Birkeland (1972)* where frequent mouthrinsing with sodium or amine fluorides reduced plaque accumulation on teeth 20 - 30%. Less plaque was noted on subjects' teeth in another study cited by Mellberg and Ripa, where there was a high dental fluorosis rate.

In another experiment plaque accumulation 3 days after an APF gel application was significantly less on experimental teeth.

Streptococcus mutans levels are reduced up to 75% in such cases and lower levels of *strep. mutans* may persist for up to 2 weeks. This effect may be related to the duration of loss of fluoride from treated enamel. Enamel fluoride loss after 5 and 10 daily APF gel applications was found to last up to 90 and 150 days respectively.

Keene et al (1971) found interproximal sites *strep. mutans*-free for up to 2 months following flossing with SnF_2 on dental floss.

It thus appears that the loss from enamel of unbound fluoride, over time, to plaque, can maintain high plaque fluoride levels and thus the potential for a high bacterial-inhibitory effect.

Mellberg and Ripa find the effect of fluoride on plaque metabolism may be reflected in reductions in:

- i) numbers of micro-organisms;
- ii) amounts of intra- and extracellular polysaccharides;
- iii) acid forming ability of plaque.

Plaque in subjects from areas with fluoridated drinking water is different to non-fluoride consuming subjects. In the fluoride area, the plaque contains fewer polysaccharide producing *streptococci*. All fluoride rinses have been shown to reduce the intracellular polysaccharide content of human plaque. Intracellular polysaccharides can be utilised to produce acid when extracellular glucose is unavailable. Sodium MFP rinsing also reduces extracellular polysaccharide which is important in providing plaque matrix. Acid production by plaque is less in subjects from fluoride areas than in subjects from non-fluoride areas.

Apparently little of the fluoride in plaque derives directly from enamel in the longer term. Mellberg and Ripa cite one experiment by *Zwemer (1957)*, where teeth were isolated and treated with 4% NaF for 20 minutes. Acid production *in vivo* was inhibited 56% immediately after application. This reduced to only 14% inhibition after 24 hours. One week later no inhibition was detectable. Also cited is a similar experiment by *Wooley and Rickles (1971)* which showed a similar result.

The duration of effect seems to be related to the concentration of fluoride and the method and frequency of application as previously mentioned. Rinses may have an effect of less than one day while a series of APF gel applications has been shown to release fluoride to plaque for as long as 8 months.

It seems that once non-bound enamel fluoride is fully released, there is no further contribution by enamel to plaque fluoride levels. This is supported by the fact that plaque grown on shark enamel (33,000 ppm F), which is nearly pure fluorapatite does not contain higher fluoride levels than plaque grown on untreated human enamel (*Gron et al, 1969*).

On the other hand, enamel under plaque with a high fluoride content may also have a high fluoride content (*Charleston et al, 1974*). This seems to be due to the concentration of fluoride in the surface enamel, which is due to the effect of demineralisation and remineralisation mentioned earlier under the mechanics of fluoride action.

Additionally, fluoride has been shown to desorb protein and bacteria from hydroxyapatite and may thus change the enamel pellicle. This would appear to be related to surface energy changes or surface charge changes.

Ericsson (1977) noted that high concentrations of fluoride such as in mouthrinses can kill *strep. mutans*. In addition fluoride can also reduce bacterial adherence to each other and to the tooth. Fluoride can thus interfere with colonisation. Fluoride is also reported to reduce bacterial acid production (*Mellberg and Ripa, 1983, p66*). The source of such fluoride can be in solution or adsorbed CaF_2 . It may also derive from fluorapatite in enamel and be released to solution.

The findings of *Mellberg and Ripa (1983)* above do not support this last point of Ericsson's.

Beltran and Burt (1988) feel that fluoride may also create a change in "herd immunity". That is to say, that fluoride may, via its antibacterial effects, create a longer term intraoral ecological change. They highlight that caries is transmissible. *Strep. mutans* is transferred from the mother to the child soon after tooth eruption. They postulate that fluoride may interfere with this transmission by reducing *Strep. mutans* levels in mothers, so that few bacteria are transmitted. Fluoride in low concentration can induce *Strep. mutans* to become less cariogenic. Thus, some years of this effect may lead to much less overall caries due to intraoral microflora alterations. This may explain why maximal caries reduction is not achieved in the first cohort after fluoridation begins.

(iv) THE EFFECTIVENESS OF FLUORIDE IN THE REDUCTION OF DENTAL CARIES

Since the link between fluoride and dental caries was established conclusively there have been a multitude of studies worldwide, of the effectiveness of fluoride in reducing dental caries. These studies examined fluoride in different forms and by different vehicles of delivery.

Historically, community water fluoridation was the earliest medium of fluoride provision. Later, other vehicles were investigated to assess their effectiveness of use in areas where for political, geographic or other reasons, community water fluoridation was not possible.

Community Water Fluoridation

Results with community water fluoridation have ranged from a 30 - 70% reduction in decay where fluoridated water was consumed from birth. *Silverstone (1978); and Haugejorden and Heloe (1981)* concluded that the caries protective effect of water fluoridation averaged 50% approximately for both dentitions. No studies showed less than 20% reduction, and reductions in caries above 80 - 90% are rare (*Murray and Rugg-Gunn, 1979*).

Effects on Primary Teeth

More specifically the primary teeth show a 40 - 50% reduction in decay prevalence. *Newbrun (1989)* reports 30 - 60% reductions.

In addition *Mellberg and Ripa (1983)* mention that 6 - 9 year-old children in optimally fluoridated *Newburgh* had six times the number of caries-free primary cuspids and molars compared with their counterparts in *Kingston* (fluoride deficient).

Effects on Permanent Teeth

Water fluoridation will effect an average caries reduction of approximately 50% (*Mellberg and Ripa, 1983*). The *World Health Organisation (1983, p62-3)* listed average caries reductions ranging from 50 - 75% for consumers of fluoridated water from birth.

Benefits are greatest when fluoride is available pre-eruptively during mineralisation. However, erupted teeth also benefit e.g. 17 year-old *Newburgh* children exposed to water-fluoridation for only 9 years, had 28% less caries than *Kingston* controls (*Mellberg and Ripa, 1983*).

Hardwick et al (1982) found that children too old to experience pre-eruptive benefits from water fluoridation, which commenced when they were 12 years-old, still showed a 27% reduction in decay after 4 years. This demonstrates the important topical effect of consuming optimally fluoridated water.

Several sources (*Stamm and Banting, 1980; Haugejorden and Heloe, 1981; WHO, 1984 pp 63-4; Newbrun, 1989; and A.D.A, 1989*) emphasise that adults experience the caries-preventive effect from continued fluoride exposure from birth, but, in addition, this effect is obvious after permanent tooth eruption, provided they are lifetime consumers of fluoride.

In fluoridated communities, the number of caries-free children is 6 times that of non-fluoride communities, and there are 75% less extracted first molars in fluoridated communities. Additionally, there is a 95% reduction in smooth surface caries in anterior teeth in fluoridated communities (*Arnold, 1943*). The obvious orthodontic implications of decreased first molar extractions will be elaborated upon later in this thesis.

Adult Benefits

Life-long consumers of fluoridated water from birth will enjoy high caries resistance. As mentioned, those whose fluoride exposure was started after the eruption of the permanent teeth will also enjoy some caries resistance. These effects will be lifelong if fluoridated water consumption is continued (*W.H.O., 1984 p 64; and A.D.A., 1989*).

The results of *Englander and Wallace (1962)* are of DMFS comparisons between 18 and 19 year-olds from optimally fluoridated and low fluoride areas. DMFS values in the fluoride areas were 60% less. *Russell and Elvove (1951)* found similar results when they examined 20-44 year-olds living in similar areas.

The *A.D.A. (1989)* claims adults benefit from water fluoridation commenced after permanent tooth eruption due to the remineralisation of carious lesions which can be halted or even reversed. Adults from *Aurora* (high fluoride) have 40% lower DMFT values than nearby *Rockford* (low fluoride).

Another benefit to adults is the reduction in the amount of cemental caries occurring in middle-age to old-age. This effect is found not only in fluoride consumers from birth but also adults exposed to the topical effect of fluoride only (*Mellberg and Ripa, 1983; W.H.O., 1984; and A.D.A., 1989*).

School Water Fluoridation

This system is mainly operative in the U.S.A. in rural areas where piped central community water-fluoridation is impossible. Reductions in DMFT/S in 6 - 17 year-olds after 8 - 12 years at school are around 33 - 40% (*Haugejorden and Heloe, 1981*). *Mellberg and Ripa (1983)* report caries reductions from 20 - 40%.

Generally, water is delivered at a level of 3 ppm F to make up for consumption elsewhere at low levels of fluoride. The disadvantages according to *Haugejorden and Heloe (1981)* and *Mellberg and Ripa (1983)* are:

- * *the child doesn't enter the program until 5- 6 years-old;*
- * *there is no consumption at weekends and holidays;*
- * *there is no consumption beyond school age.*

School and Home-based Fluoride Tablet and Drop Programs:

In school-based programs decay reductions of 16 - 84% have been found after 2 - 8 years of administration. Children ranged from 3 - 11 years at the start. Most studies show 20 - 40% reductions. (*Haugejorden and Heloe, 1981*).

Again for school-based programs the disadvantages are the same as those listed above for school water-fluoridation programs.

In home-based situations the greatest reductions in caries are recorded for those receiving supplements from birth.

Generally, reductions parallel those for water-fluoridation (*Silverstone, 1978; Mellberg and Ripa, 1983; W.H.O. 1984*).

Aasenden and Peebles (1974) cited *Hamberg (1971)* who administered 0.5 mg F per day to infants 2 - 3 weeks after birth. Dosage was kept

constant. Deciduous teeth of 4 - 5 year-olds had 50 - 70% less caries than controls. Additionally, Aasenden and Peebles found that fluoride tablets produced a greater caries reduction than fluoridated water.

This was clouded by experimental problems:

- *oral hygiene status of subjects was not assessed;*
- *there were marked variations in water consumption by subjects;*
- *fluoride levels in the water varied during the study.*

They concluded both should give equivalent caries reductions. The fluoride tablet group showed a caries reduction of 75 - 80%.

Many preparations for infants are combined with vitamins in drop form. There appears to be no difference in effectiveness of fluoride-vitamin drops when compared with fluoride drops alone (*Mellberg and Ripa, 1983*).

Horowitz in the *A.D.A. article (1989)* noted a 60% caries reduction in one *Virginia* county where fluoride tablets and supervised rinsing were combined.

The obvious weakness of home-based administrations is that of continuing parent and child co-operation in following dosages.

Salt Fluoridation

Although not widespread, salt fluoridation has been found to give significant levels of caries reduction. 200 - 300 mg F/kg yields similar urinary fluoride levels as 1 ppm F water. Caries reductions have ranged from 48 - 67% in 7 - 14 year-olds in studies over 8 - 10 years (*Haugejorden and Heloe, 1981*).

Additionally a 48% reduction in dmft for life-long consumers at 4 - 5 years of age has been noted for levels of intake of 250 mg F/kg. These findings of *Haugejorden and Heloe (1981)* are generally supported by *Mellberg and Ripa (1983)*.

In contrast, *Silverstone (1978 p 24)* found salt fluoridation to be only half as effective as water-fluoridation.

Milk and Flour Fluoridation

Inconclusive results with these two vehicles don't permit any valid statements regarding their general effectiveness in caries reduction (*Haugejorden and Heloe, 1981; Mellberg and Ripa, 1983; and WHO, 1984 pp 67-8*).

The above fluoride vehicles work mainly as systemic agents. Below are the predominantly topical agents for fluoride delivery.

Toothbrushing with Fluoride

Haugejorden and Heloe (1981) conclude from school-based programs that results with annual or semi-annual brushing with fluoride prophylaxis paste are inconclusive.

They note caries reductions of 3 - 44% for supervised brushing 4-5 times annually over 2 - 3 years with 0.5 - 1.23% NaF gels or solutions. However, they feel 2 - 3 years is too short a time to make valid estimates of the value of the procedure over the period of compulsory school attendance.

Mellberg and Ripa (1983) generally feel that due to problems with young children swallowing these solutions, and because other methods are generally more effective, this method of fluoride delivery is not of great value.

Flouride Mouthrinses

The *World Health Organisation (1984, pp 68-9)* lists mouthrinsing as effecting caries reductions of the order of 20 - 35% depending on the regime followed.

Haugejorden and Heloe (1981); and *A.D.A. (1989)* found on average, for school-based supervised rinsing programs, reductions of up to 40%. This varied with the regime followed. *Haugejorden and Heloe (1981)* note that, particularly in Scandinavian countries, this has been a most effective caries-preventive method. This is particularly so where water-fluoridation is difficult or not legally sanctioned. They also mention the problem of low follow-on rates once participants leave school. In addition, co-operation problems are common with high-school-age students.

Silverstone (1978) and *Mellberg and Ripa (1983)* report *Torrell and Ericsson's (1965)* findings that a 49% caries reduction occurred with 0.05% NaF solution daily over a 2 year period in 10 - 12 year-olds. This compared better than a supervised rinsing program once a fortnight using 0.2% NaF. This later study was using 10 - 12 year-olds followed over a two year period and yielded only a 21% reduction.

Rugg-Gunn et al (1973) found a 37% decrease in caries with 0.05% NaF mouthrinse supervised daily over a 3 year period.

Aasenden cited by *Mellberg and Ripa (1983)* found an increased caries preventive effect from fluoride rinsing in a fluoride area. *Mellberg and Ripa* feel that such procedures, even though showing greater statistical effectiveness, really don't mean any great clinical difference in most cases.

Koch (1969) warns of the likely results of cessation of mouthrinsing programs. In his study 2 years after cessation, control and experimental groups showed no difference in caries incidence.

Professionally Applied Topical Fluoride Solutions

These include solutions, gels and varnishes.

3 main agents have been used in most studies. Namely NaF, SnF₂ and APF.

For sodium fluoride, usually a 2% solution variously applied 2 - 4 times per annum results in average caries reductions of 20% (*Mellberg and Ripa, 1983*). *Silverstone (1978)* mentions the results of *Bibby (1942)* of a 33% caries reduction using a 0.1% NaF solution 3 times per annum. *Knutson (1948)* recorded 30 - 40% caries reductions using 2% NaF solutions.

Stannous fluoride is usually used at 8 - 10% strength with 1 or 2 applications per annum. This regime produces an average caries reduction of 30 - 35% (*Mellberg and Ripa, 1983*).

Acidulated Phosphate Fluoride is usually composed of 1.23% NaF buffered to pH 3 - 4 in 0.1M phosphoric acid. It is normally applied 1-2 times per annum and reduces caries by an average of 30% (*Mellberg and Ripa, 1983*).

Silverstone (1978) reported reductions in caries due to APF topical applications of from 41 - 70%.

Fluoride varnishes supposedly increase the enamel exposure time to fluoride by several days (*Silverstone, 1978; Mellberg and Ripa, 1983*). Currently available preparations are Fluor Protector 0.7% F and

Duraphat 2.2% F. *Koch and Peterson's (1975)* study showed a 75% reduction in new caries in a one year study. The children involved received 2 applications.

Heuser and Schmidt (1968) achieved a 30% reduction over a 15 month period using Duraphat.

At the time of writing *Mellberg and Ripa (1983)* felt that varnishes were not sufficiently tested to be found superior to other topical applications.

On the other hand, *Haugejorden and Heloe (1981)* mention that recent studies have shown a tendency to abandon school-supervised rinsing and brushing programs in favour of 6-monthly, professional Duraphat applications. Supposedly results are equivalent to weekly rinsing with 0.2% NaF solution.

Craig and Powell (1979) draw attention to an alternative to fluoride varnishes. They use the prolonged fluoride application (PFA) method to protect fissures considered susceptible to caries. The agent is stannous fluoride in Orabase ointment. Once the slurry of the above components is placed, it is covered with an isolation Stomahesive wafer for 1 hour.

The technique falls midway between the fluoride varnish and normal topical application of fluoride, with respect to the time the fluoride compound is intimately related to the fissure.

The effectiveness of this method, over a 4 year period, is noted by the authors.

Fluoride Toothpastes

These act via a topical route, mainly in older children (> 5 - 6 years). However, in younger children up to 1/3 of the toothpaste used may be swallowed. Thus, for fluoride toothpaste with levels approaching 1000 ppm F, there is a distinct and sometimes unwanted systemic action in younger children (*Mellberg and Ripa, 1983; Ophaug and Singer, 1988*). Additionally, younger children frequently consume toothpaste at other times. *Silverstone (1978)* felt they should not be used until children were 4 years old for this reason.

In optimally-fluoridated communities, this ingestion may be linked to enamel fluorosis. However, in fluoride-deficient communities where there are no other systemic sources of fluoride, ingestion may be beneficial (*Mellberg and Ripa, 1983*).

In non-fluoride communities, fluoride toothpastes may reduce caries up to 15 - 30% with an average of 20% DMFS decrease (*Silverstone, 1978*). *Mellberg and Ripa, (1983)* and *W.H.O. (1984)* reported caries reductions from 20 - 30% in non-fluoride areas when fluoride toothpaste was used. In fluoride areas the average caries reductions were 17%. These authors, as well as *Beltran and Burt (1988)*, comment that this additive effect in such areas is a worthwhile contribution to overall caries reduction; presumably if it is not swallowed.

Mellberg and Ripa (1983) summarised their ideas on the mode of action of the various topical agents and methods of fluoridation. In general, the greater the concentration of fluoride available, the greater the anticaries effect will be. This results in:

- i) increased fluoride incorporation in sound enamel and incipient lesions;

ii) greater depth of penetration of fluoride;

iii) greater bacterial inhibition.

It would seem that greater subsurface penetration and incorporation of fluoride should result in greater decay resistance. This is due to the site of initial ion exchange being subsurface.

Additionally, the greater frequency of fluoride use, the greater the anticaries effect. This results in increased fluoride levels in plaque and enamel. Mellberg and Ripa feel that fluoride prophylaxis pastes and semi-annual and annual applications of fluoride only deposit fluoride in sound enamel or incipient lesions. They have little antibacterial effect, and remineralisation effect of more progressed lesions.

Fluoride mouthrinses are felt to have a minor fluoride-depositing effect on sound enamel, but rather more tend to remineralise enamel, inhibit plaque bacteria and increase plaque fluoride levels. This also applies to fluoride toothpastes.

Fluoride tablets or drops, if chewed and swished prior to swallowing, will, in addition to the systemic effect, have a topical effect similar to that of mouthrinses.

Overall, authors (*Silverstone, 1978; Murray and Rugg-Gunn, 1979; Haugejorden and Heloe, 1981; Mellberg and Ripa, 1983; W.H.O., 1984; Beltran and Burt, 1988; Newbrun, 1989*) still conclude community water-fluoridation to be the most effective method of reducing caries. The advantages of water-fluoridation are:

i) It is the least expensive per capita. This is especially so when it is remembered that its benefits extend to

adults as well as children. (*Garcia, 1989; Newbrun, 1989*).

ii) It is equitable, reaching all levels of society, especially those of the lower social classes who can least afford other methods of caries reduction and have the greatest need of caries reduction. There is a difference of 26% caries experience between children from upper social classes from fluoride and non-fluoride areas. On the other hand there is a 71% difference between caries experience of children from low social classes from fluoride and non-fluoride communities. Thus, those lower class children benefit the most (*Carmichael et al, 1980*).

iii) It is safe.

iv) It is eminently effective (*Newbrun, 1989*).

v) Little co-operation is needed.

vi) Lifetime benefits accrue if there is continual consumption of the fluoridated water (as mentioned earlier).

vii) Reduced dental care costs with respect to restorative, prosthetic and surgical needs. These are both on the individual and community levels.

As mentioned earlier, in fluoride areas there will be found an increased reduction of caries from topical agents used concurrently e.g. fluoride toothpastes and fluoride rinses. This effect is not purely additive, but nevertheless, statistically significant. *Mellberg*

and Ripa (1983) warn that this significance does not usually entail clinical significance, and thus they can't recommend public programs of supervised rinsing or brushing on a cost/benefit basis in areas already having optimally fluoridated water.

(v) SECULAR TRENDS IN DECAY PREVALENCE

Worldwide over the last 10 - 15 years there has been a marked secular decline in caries prevalence. Also during this same time period, the difference in caries experience of children from fluoride and non-fluoride communities, has narrowed. (*Zacherl and Long, 1979; Rise and Haugejorden, 1980; Glass, 1981; Brunelle and Carlos, 1982; Brown, 1982; Latcham et al, 1982; Anderson et al, 1982; Bohannan et al, 1985; Ophaug and Singer, 1988; A.D.A., 1989, Newbrun, 1989*).

This effect was first observed in the mid-to late-1970s in some areas of the world. By the early 1980s, it was quite obvious in many parts of the world.

In the *U.S.A.* there was a 37% decline in dental caries in children 5 - 17 years-old between 1971-4 and 1979-80 (*Bohannan et al, 1985*). The *A.D.A. (1989)* reported that in the *U.S.A.* in 1986-7, 49.9% of 5 - 17 year-olds were caries-free. This figure was 36.6% in 1980 and 28% in 1970.

Haugejorden and Heloe (1981) report on the marked caries decline evident by the late 1970s in Norway. They also note that this effect has been documented as well in Sweden.

Anderson et al (1982) noted reductions of 32 - 57% in caries prevalence from the early 1970s to early 1980s for English children 5-12 years-old. This applied in both optimally fluoridated and fluoride-deficient towns.

Brown (1982) presented further evidence of this trend in decay prevalence in New Zealand. The particularly large decrease in decay prevalence was noticed mainly between 1977-82.

Latcham et al (1982) recorded similar findings to overseas in their survey of 15 year-old schoolchildren in non-fluoridated *Brisbane*. They found approximately a 50% decline, which was principally related to the smooth surfaces of posterior teeth.

Health authorities throughout the world, as well as members of anti-fluoridation lobbies, were all keen to clearly establish the cause of this marked caries decline.

An *F.D.I. (1985)* Joint Working Group discussed this secular trend as well as likely causes. Various possible aetiological factors raised by this group as well as other authors are discussed below.

a) Water-Fluoridation

The increased utilisation of this vehicle for fluoride delivery is no doubt responsible in a large way for the continued and marked decline in caries prevalence generally in fluoride communities. It is felt that the possibly more important post-eruptive topical effect of fluoridated water is the main factor at work in water-fluoridated communities and; therefore, this is responsible for the later expression of this mechanism of caries reduction (*Beltran and Burt, 1988*). In addition, on a national basis in the *U.S.A.*, over the time period being discussed, greater implementation of water-fluoridation programs had been occurring.

b) Supervised Fluoride Rinsing Programs and School Water-Fluoridation

These have increasingly been implemented in areas where fluoridation is opposed or impractical. It would appear that the increased utilisation of these programs correlates well

with the secular decline of caries (*F.D.I., 1985; Beltran and Burt, 1988*).

Rise and Haugejorden (1980) attributed this trend in *Norway* to the increasing use of fluorides both at school and in the home. During the *1970s* school fluoride programs potentially covered more children, and more children actively participated. For *1960-71* the average surfaces filled were 5.5 each year, by *1976* this dropped to 3.6.

c) Oral Prophylaxis Programs and Dental Health Education

The increasing emphasis on these types of programs was noted by the *F.D.I. (1985)* Joint Working Group. The possibility of this being strongly correlated with the trend in question was not discounted but its impact with respect to the scale of caries level improvement was thought to be minimal. the *F.D.I.*

(1985) Joint Working Group felt that toothpaste manufacturers, through their marketing campaigns, made people more dentally aware of preventive possibilities.

In *Norway*, *Rise and Haugejorden (1980)* felt community preventive programs were largely responsible for decay reductions.

Anderson et al (1982) felt increasing use of dental health education and active campaigning against the misuse of vitamin syrups were other variables which could have played a part in the observed caries decline.

d) Sugar Consumption

Opponents of fluoridation have claimed that there is less

sugar consumption generally and that this is a major mechanism at work in the marked caries decline, and underlies the "small" difference now between non-fluoride and fluoride caries scores in children (*Beltran and Burt, 1988*).

Worldwide, sugar consumption, in the countries which have experienced this secular trend, is still up to 4 times the world average. In some of the countries concerned e.g. *Australia*, there is decreased domestic sugar use but increased use in food processing (*F.D.I., 1985*).

It is possible that subgroups of the population now consume less sugar and thus have a less cariogenic diet than 10 years ago (*Kalsbeek, 1982; F.D.I., 1983*). *Rise and Haugejorden (1980); and Koch (1982) in F.D.I. (1985)*, stated that the frequency of sugar intake has decreased. *Brown (1982)* felt that little input from sugar consumption in *New Zealand* was involved. Also *Brunelle and Carlos (1982)* felt there was no evidence of reduction in sugar consumption in the *U.S.A.* in the *1970s*. *Beltran and Burt (1988)* also agreed with this.

e) Fluoride Tablets

There appears to be a correlation between the proportion of caries-free children and the consumption of fluoride tablets in a *Netherlands* study (*F.D.I., 1985*). At the same time, sales of fluoride tablets rose from 50 million per annum in *1965* to 500 million per annum in *1975*.

Glass (1981) felt fluoride tablets could be related to the caries trend observed.

However in 1977, only 21% of sampled *Brisbane* primary schoolchildren used fluoride tablets regularly. 35% reported sporadic use (*Latcham et al, 1982*).

Little effect in the marked caries decline in *New Zealand* was attributed to fluoride tablets by *Brown (1982)*.

f) Fluoride Dentrifices

Fluoride rinsing programs, fluoride tablet consumption (whether school or home-based) and fluoride dentrifice usage are held as the main cause of the trend in question.

In non-fluoride communities it is felt that the 3 agents may, if used in combination, exert an additive effect on caries reduction (*Glass, 1981; Rise and Haugejorden, 1980; F.D.I., 1985*).

Also it has been noted that many children will derive a significant systemic intake from fluoride toothpaste that is swallowed (*Ophaug and Singer, 1988*).

Children 2 - 4 years-old will swallow 26 - 35% of their toothpaste due to immature swallowing patterns. By 18 months of age, 78% of children are instructed in brushing by their parents and are either brushing their own teeth in some way or have this done for them by their parents, usually twice-daily (*Ophaug and Singer, 1988*).

In fluoridated communities, it is felt that the topical effect of fluoride dentrifice usage combined with any extra fluoride ingestion from swallowed toothpaste is responsible for

increased caries reduction figures recently obtained, above what has normally been expected.

Additionally the market domination by fluoride toothpastes was minimal in the late *1960s* and early *1970s* but rose to very high levels by the mid-to-late *1970s* in most countries (*F.D.I., 1985*).

Anderson et al (1982) noted the correlation of increased fluoride toothpaste use from 1970 (5% of sales) to 1977 (> 95% of sales), as did *Glass (1981)* in the *U.S.A.* and *Latcham et al (1982)* in *Australia*, and *Brown (1982)* in *New Zealand*. *Beltran and Burt (1988)* also note this occurrence worldwide.

The *F.D.I. (1985)* Joint Working Group noted that, worldwide, this trend for increased fluoride toothpaste use correlated very strongly with the observed decrease in decay prevalence registered in many countries in both fluoride and non-fluoride communities.

g) Availability of Dental Services

During the time periods in question, all countries exhibiting the trend in decay prevalence, had readily available dental services in place. So in this respect access to dental care could not be related to the trend (*F.D.I., 1985*).

There has always been high utilisation of the *New Zealand School Dental Service* according to *Brown (1982)*, so that any input from this aspect in *New Zealand* could be ignored.

h) Increased Preventive Approach by Public and Private Dentistry

The greater use of fluoride supplements accompanying such a change in emphasis of practice is no doubt linked to the caries decline in question. However, the *F.D.I. Joint Working Group* said it was difficult to identify the impact of this approach.

Additionally, a more optimistic approach to early caries has been adopted generally. The "*wait and closely monitor approach*" has become more widespread (*Glass, 1981; Brown, 1982*).

Rise and Haugejorden (1980) also felt less extractions were performed, with restorations preferred. They also felt a better level of diagnosis may have been present with perhaps a greater reliance on radiographs.

i) Changes in the Diagnostic Criteria of Studies and Associated Statistical Validity

Only *New Zealand's School Dental Service* adopted a changed diagnosis of caries. Other participating countries in the *Joint Working Group* study by the *F.D.I. (1985)* reported no differences in their diagnostic criteria as it might relate to the observed secular trend of decay prevalence.

Brown (1982) and Latcham et al (1982) felt that a change in diagnostic criteria for other studies could be partly related to the decline. However, they felt this change would be mainly related to occlusal surfaces only. Inter-examiner variation is also involved here.

j) Widespread Use of Antibiotics

An inverse relationship between caries experience and antibiotic experience has been reported by *Loesche et al (1982)* in *F.D.I. (1985)*. Couple this observation with the greater use of antibiotics generally. Additionally, *Strep. mutans* has exhibited suppression in saliva of children receiving antibiotics for other conditions (*F.D.I., 1985; Beltran and Burt, 1988*).

The F.D.I. Joint Working Group was unable to measure retrospectively this potential factor. They believed that these agents couldn't explain the sudden and dramatic caries decline observed in most caries studies from the mid-1970s.

Glass, (1981); and *Beltran and Burt (1988)* felt unknown changes in oral microflora may be partly responsible for the observed caries decline.

k) Herd Immunity

The possibility of Man adapting to the pathogens involved in dental caries has been proposed by *Beltran, (1978); Bowen, (1981)* and *Beltran and Burt (1988)*.

The Joint Working Group discounted this as a possibility with respect to the trend, since this process would exhibit a slow decline. Herd immunity thus can't account for the dramatic declines in caries observed in the mid-1970s and later.

l) Increased Use of Fissure Sealants

The possibility of this procedure effecting a dramatic decline in DMF scores alone has not been given great credence. However, the increased use of these sealants was associated

with the time period in question (*Bagramian et al, 1979*). This mechanism was not discussed by the F.D.I. Joint Working Group.

m) Increased Consumption of Foods Grown or Processed in Fluoridated Water from Fluoride Areas

With the greater mobility of rural products and processed foods in recent years, it is conceivable that foods from fluoride areas may exhibit additive effects in decay reduction in fluoridated areas. In non-fluoridated areas these fluoride-containing foods may yield another systemic source of fluoride (*Brown, 1982; Ophaug and Singer, 1988; Newbrun, 1989*).

This aspect was not discussed by the F.D.I. Joint Working Group. In the *U.S.A. and Australia*, with most major cities fluoridated, the outflow of processed foods (containing fluoride) to other fluoride and non-fluoride areas, presents an important source of fluoride.

n) Infant Formulas

Ophaug and Singer, (1988) mention that infant formulas were a problem prior to 1979 due to their potential for causing dental fluorosis. Also in the early 1970s there was a greater reliance on formulas for infants. In 1971, 77.4% of newborn infants in the *U.S.A.* received formulas. The pattern of formula use has decreased. However, these earlier recipients of formula often suffered fluorosis if in a fluoride area. This was due to the manufacturers using fluoridated water in their formula. There was thus a concentrating effect of this fluoride.

It is interesting to note that this increased usage of high

fluoride formula was most widespread during the period of the dramatic caries declines in question. Since 1979, formulas manufactured in the U.S.A. have used deionised water.

c) Part of a Natural Waxing/Waning Phenomenon

Anderson et al (1982) really didn't have much evidence as to the cause of the sudden decline in caries prevalence but felt that it was possibly related to a natural rhythmic cycle which was independent of preventive measures.

They draw attention to the example of Finland, where since 1934, caries prevalence improved, deteriorated and improved again. Similar changes have occurred in England over the same time as well. However, it was conceded that this phenomenon may be intimately related to the Great Depression, and to sugar consumption prior to and after World War II.

Beltran and Burt (1988) mention this phenomenon as a possibility but have no evidence for this.

Conclusion

The F.D.I Joint Working Group found common factors in all the countries who experienced the dramatic caries reductions -

- 1) widespread availability of fluorides - especially fluoride dentrifices;
- 2) availability of dental resources;
- 3) increased dental awareness coupled with greater utilisation of dental services;
- 4) preventive approach practitioners.

3 other factors were common to some of the countries involved:

- i) water fluoridation
- ii) fluoride supplements
- iii) reduction in sugar consumption

The Joint Working Group concluded that the prime reason for this decay reduction was the dramatically increased use of fluoride dentrificates during the 1970s. In areas that were already fluoridated there was an additive effect from the systemic and topical effect of the dentrifice fluoride (Haugejorden and Heloe, 1981).

Brunelle and Carlos (1982) stated that the decline in caries prevalence was greatest in the interproximal areas of permanent teeth. This is where fluoride has its greatest effect. They thus felt this was good evidence to link the decline to fluoride.

Most other researchers concluded likewise, that the increasingly widespread use of all types of fluoride was making remineralisation of incipient lesions more effective due to more frequent exposure to increased amounts of fluoride (Rise and Haugejorden, 1980; Glass, 1981; Haugejorden and Heloe, 1981; Brunelle and Carlos, 1982; Bohannan et al, 1985; and Beltran and Burt, 1988).

Carlos cited by the A.D.A. (1989) cautions that there is still a 25% difference in caries experience between children in water-fluoridated areas and unfluoridated areas. DMF scores for children of all ages in fluoride areas are still significantly lower than for children in non-fluoride areas. Thus, there should be no challenge to water-fluoridation regarding its effectiveness, which is augmented by the additive effects of other fluoride sources.

Additionally, Beltran and Burt (1988) feel that this decline is still

not over. If this trend in decay reduction continues, some researchers feel that caries will be virtually eliminated in one or two more decades (A.D.A., 1989).

(vi) FLUORIDATION IN TAMWORTH

Adjustment of *Tamworth's* water supply to a level of 1 ppm was commenced in *October, 1963*. Before the commencement of fluoridation, the first widespread *Tamworth Fluoridation Survey* was undertaken to assess the dental health of children who were lifelong residents of the immediate Tamworth area.

The results of this assessment of dental health in *1963* were to serve as controls for subsequent fluoridation surveys.

Other major surveys were undertaken in *1973* and *1979* using the same criteria. These were followed again by another fluoridation survey in *November, 1988*.

This thesis is concerned with the results of the DMFT index scored in *Tamworth* for the years *1963-1988* for 12 and 15 year-olds, and to test for changes in malocclusion prevalence and experience.

However, it was felt by the present writer that the inclusion of dmft index scores for the 2-7 year-old age group for the years *1963-1988* would also be beneficial. Other "*worked*" results derived from the DMFT and dmft indices results are also presented; namely, the mt (deciduous teeth missing due to caries) component of the dmft score; the percentage of children with deciduous tooth extraction due to caries; the percentage of children with a caries-free deciduous dentition; the percentage of children with permanent teeth extracted due to caries; and the percentage of children with a caries-free permanent dentition. The DMFT and dmft (for 5 and 6 year-olds) are presented below as graphs. The DMFT and dmft are also presented below as tables, along with those worked-result categories just mentioned above.

It is the intention of the present writer to highlight not only the changes in DMFT scores from *1963-1988*, but also the changes that have occurred in dmft, mt and caries-free scores (both dentitions) from *1963-1988*.

The present writer contends that it is the effect of unrestored (or poorly restored) caries, or extractions of deciduous and permanent teeth (mainly) from the ages of 4 to 7 years that is responsible for a significant number of malocclusions. Evidence to support this contention will be presented in PART II of this thesis. Thus, it is more the decline over the years *1963-1988* in the dental caries prevalence in the deciduous dentition (from the ages of 4 - 7 years) that may be responsible for most reductions in malocclusion prevalence in 12 and 15 year-old *Tamworth* children from *1963-1988*.

Thus, the caries-preventive effect of fluoride on tooth enamel is assumed to be the main agent involved in any changes in the malocclusion status of children from *1963 to 1988 in Tamworth*.

TABLE: 1 - 1

12 year-olds in 1988	dmft	DMFT	% caries-free decid. teeth	% caries-free perm. teeth	mt	% perm. teeth exo due caries	% decid. teeth exo due caries
when 4 years in 1980	*	*	*	*	*	0.00**	*
when 5 years in 1981	2.0**	0.0**	51.4**	99.1**	.22**	0.0**	6.8**
when 6 years in 1982	2.0**	0.1**	45.4**	95.3**	.37**	0.0**	14.0**
when 7 years in 1983	*	*	*	*	*	*	*

TABLE: 1 - 2

15 year-olds in 1988	dmft	DMFT	% caries-free decid. teeth	% caries-free perm. teeth	mt	% perm. teeth exo due caries	% decid. teeth exo due caries
when 4 years in 1977	*	*	*	*	*	*	*
when 5 years in 1978	2.0**	0.0**	51.4**	99.1**	.22**	0.0**	6.8**
when 6 years in 1979	2.0	0.1	45.4	95.3	.37	0.0	14.0
when 7 years in 1980	2.6*	0.4**	40.7**	78.3**	.48**	0.0**	20.2**

* no figures for years 1980-1987 available

** used 1979 figures where it was felt reasonable to do so.

TABLE: 1 - 3

<i>12 year-olds in 1979</i>	<i>dmft</i>	<i>DMFT</i>	<i>% caries-free decid. teeth</i>	<i>% caries-free perm. teeth</i>	<i>mt</i>	<i>% perm. teeth exo due caries</i>	<i>% decid. teeth exo due caries</i>
<i>when 4 years in 1971</i>	2.1	0	57	98.9	.16	0	5.4
<i>when 5 years in 1972</i>	2.3	.1	51.3	96.5	.25	0	8.5
<i>when 6 years in 1973</i>	2.9	.4	42.4	78.3	.47	0	16.4
<i>when 7 years in 1974</i>	n/av	n/av	n/av	n/av	n/av	n/av	n/av

TABLE: 1 - 4

<i>15 year-olds in 1979</i>	<i>dmft</i>	<i>DMFT</i>	<i>% caries-free decid. teeth</i>	<i>% caries-free perm. teeth</i>	<i>mt</i>	<i>% perm. teeth exo due caries</i>	<i>% decid. teeth exo due caries</i>
<i>when 4 years in 1968</i>	3.2	0	38.1	100	.15	0	6.7
<i>when 5 years in 1969</i>	3.1	0.1	38.8	95	.26	0	9.2
<i>when 6 years in 1970</i>	3.8	0.5	29.0	72.7	.49	0	17.2
<i>when 7 years in 1971</i>	4.1*	1.2	25.5	48.7	.68	0.6	24.0

TABLE: 1 - 5

12 year-olds in 1973	dmft	DNFT	% caries-free decid. teeth	% caries-free perm. teeth	nt	% perm. teeth exo due caries	% decid. teeth exo due caries
when 4 years in 1965	5.5	0.1	26.6	98.4	.38	0	14.1
when 5 years in 1966	6.0	0.2	16.1	89.4	.43	0	16.1
when 6 years in 1967	5.6	0.9	18.4	57.7	.78	0	24.1
when 7 year in 1968	6.2	1.8	15.4	33.1	1.02	1.5	35.8

TABLE: 1 - 6

15 year-olds in 1973	dmft	DNFT	% caries-free decid. teeth	% caries-free perm. teeth	nt	% perm. teeth exo due caries	% decid. teeth exo due caries
when 4 years in 1962	n/av	n/av	n/av	n/av	n/av	n/av	n/av
when 5 years in 1963	5.9	0.2	16.5	89.5	.72	0	21.1
when 6 years in 1964	7.1	1.4	9.5	47.1	1.14	0	32.8
when 7 years in 1965	7.0	2.3	6.3	24.5	1.47	0.7	42.9

TABLE: 1 - 7

(Estimated from the country figures of Barnard (1956)

12 year-olds in 1963	dmft	DNFT	% caries-free decid. teeth	% caries-free perm. teeth	mt	% perm. teeth exo due caries	% decid. teeth exo due caries
when 4 years in 1955	n/av	n/av	n/av	*	n/av	n/av	n/av
when 5 years in 1956	n/av	n/av	n/av	*	n/av	n/av	n/av
when 6 years in 1957	n/av	0.8	n/av	65.1	n/av	n/av	n/av
when 7 years in 1958	n/av	2.04	n/av	29.00	n/av	n/av	n/av

TABLE: 1 - 8

15 year-olds in 1963	dmft	DNFT	% caries-free decid. teeth	% caries-free perm. teeth	mt	% perm. teeth exo due caries	% decid. teeth exo due caries
when 4 years in 1952	n/av	n/av	n/av	*	n/av	n/av	n/av
when 5 years in 1953	n/av	n/av	n/av	*	n/av	n/av	n/av
when 6 years in 1954	n/av	.8	n/av	65.1	n/av	n/av	n/av
when 7 years in 1955	n/av	2.04	n/av	29.00	n/av	n/av	n/av

There is also interest in the DMFT results from the assessment of Kadina High School students from *Lismore*. These 12, 13 14 and 15year-old students were examined in *October, 1989*. *Lismore* has negligible fluoride in the water supply. *Lismore* subjects were examined to act as contemporary controls. *Bohannan et al (1985)* stress the need for such experimental design especially since the secular decline in caries.

One important point to consider in any analysis of the results below is that *Australia* also experienced the significant secular decline in caries prevalence as discussed earlier in this thesis (*Latcham et al, 1982*). Therefore, one would expect to see a further significant reduction in DMFT scores from the late *1970s* to the early *1980s*. This of course would be mainly due to the increased use of fluoride dentrifice that happened in the time period mentioned. Thus the results of the Tamworth Fluoridation Survey are not due to the effects of water fluoridation alone. This point needs to be remembered when reading further in this thesis and interpreting any results. This was the reason for including contemporary controls from a non-fluoridated city (*Lismore*).

A comparison of the DMFT scores for *Tamworth* in *1963, 1973* and *1988*; and *Lismore, 1989* is to be found in Table 1 - 9. Results are listed according to age.

TABLE: 1 - 9

Age	Tamworth 1963 DMFT	Tamworth 1972 DMFT	Tamworth 1988 DMFT	Lismore 1988 DMFT
12	8.3	4.2	0.9	overall 12-14 yrs. combined 2.0
13	9.6	5.7		
14	10.6	6.4		
15	12.3	7.7	1.5	
combined	10.2	6.0	1.2	

Note: that for *Lismore* the results are calculated on a total sample of 160 subjects of whom 120 were suitable for the subsequent **Malocclusion Survey**, which served as a contemporary comparison with *Tamworth*. The 40 subjects excluded had received or were receiving orthodontic treatment.

There were 40.6% of children with caries-free permanent dentitions in *Lismore*, 1989. This was for combined sexes and age groups 12 - 14 years. *Tamworth*, 1988 showed 65% of 12 year-olds, and 52% of 15 year-olds caries-free.

FIGURE I-1

DECREASE IN DENTAL CARIES - DMFT
TAMWORTH DENTAL SURVEY 1963 - 1988

School children aged 6, 9, 12, 15 yrs.

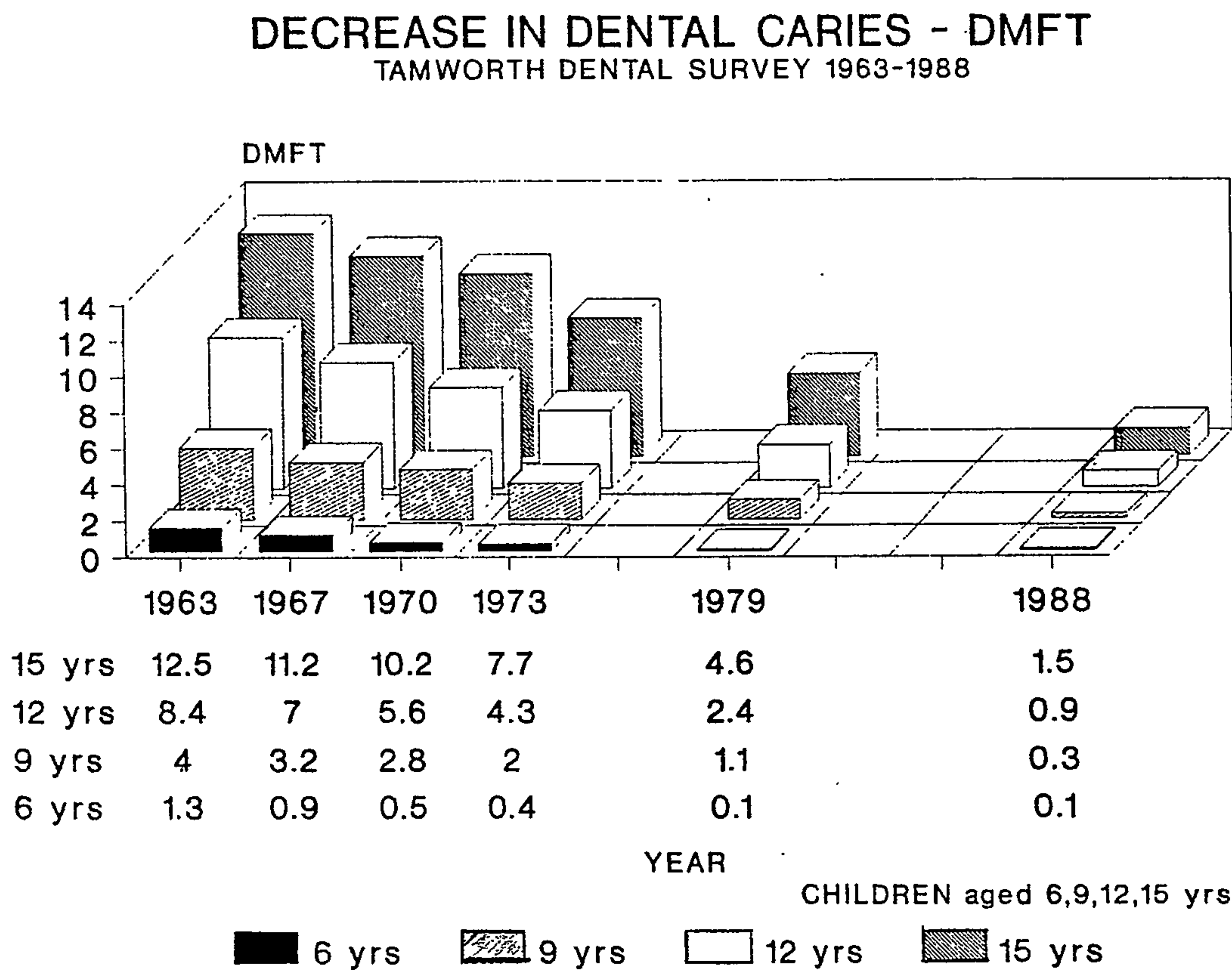


FIGURE I-2

DENTAL CARIES - dmft COMPONENTS
TAMWORTH DENTAL SURVEY 1963 - 1988

School children aged 6 yrs

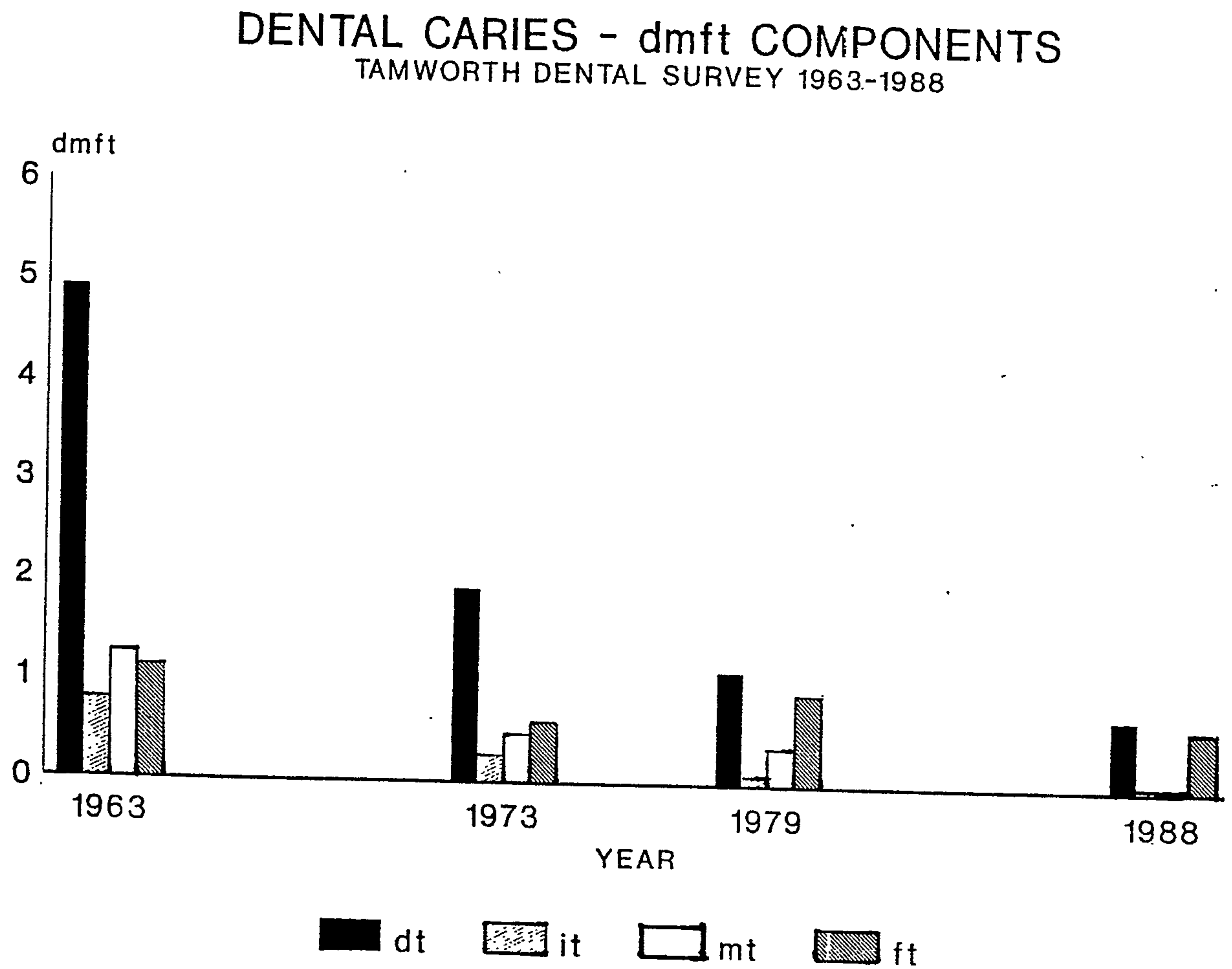


FIGURE 1-3

DENTAL CARIES - dmft COMPONENTS
TAMWORTH DENTAL SURVEY 1963 - 1988
School children aged 5 yrs

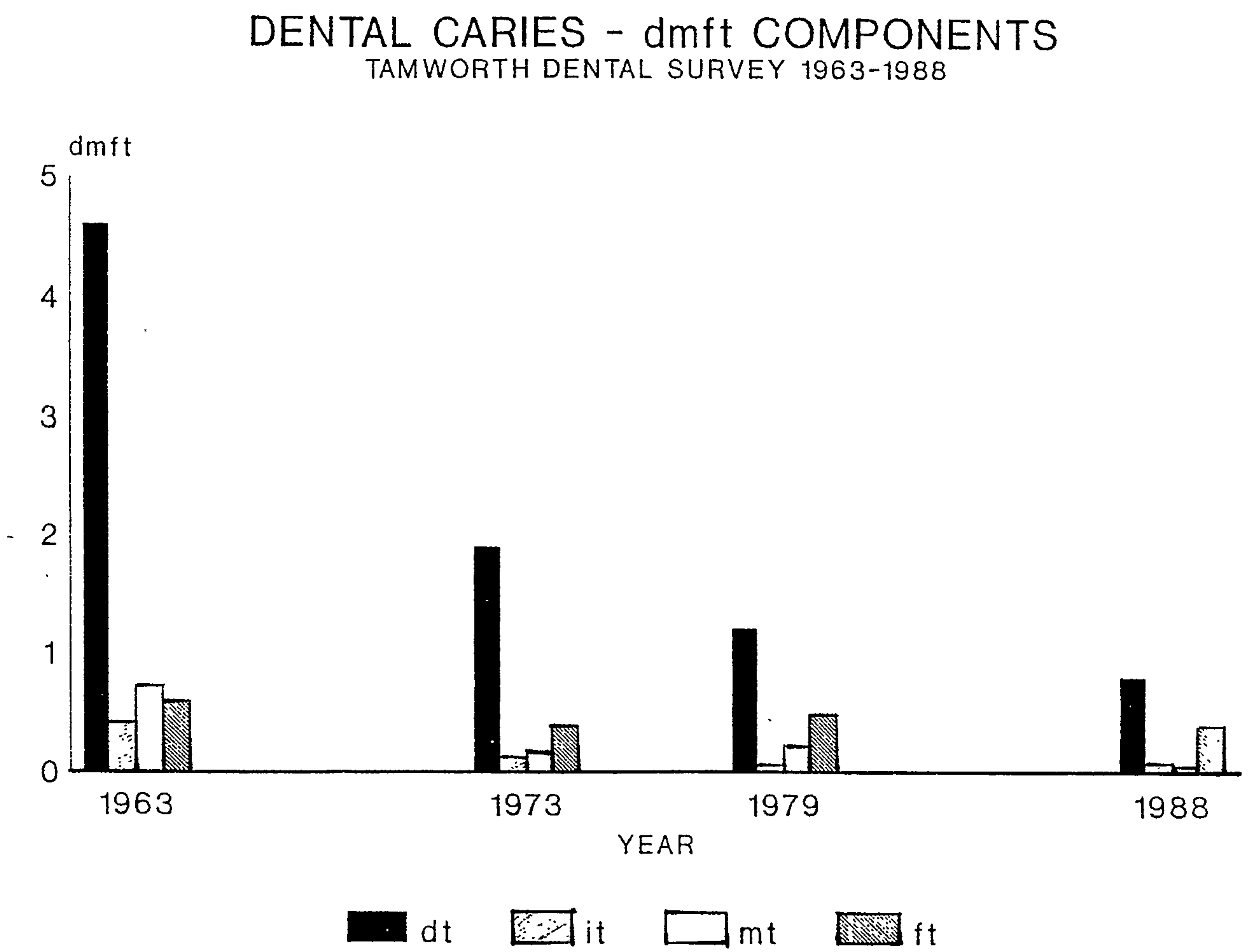


TABLE: 1 - 10

TANWORTH DENTAL SURVEY 1963 - 1973, 1979, 1988 (after Barnard, 1988)

Group All Schoolchildren		Index: Decayed, Missing or Filled Deciduous Teeth/Child (dmft)												
Age Group (years)	1963	1964	1965	1966	1967	1968	1969	1970	1971	1972	1973	1979	1988	
4	5.5	6.3	5.5	3.8	3.2	3.2	1.9	2.1	2.1	1.7	1.1	-	n/o	
5	5.9	6.6	6.3	6.0	4.5	4.5	3.1	3.0	2.9	2.3	2.5	2.0	1.3	
6	7.2	7.1	7.2	6.9	5.6	5.6	4.4	3.8	3.7	3.2	2.9	2.0	1.4	
7	7.0	7.3	7.0	7.1	6.2	6.2	4.9	4.9	4.1	4.0	3.3	2.6	n/o	
8	6.5	6.8	7.0	6.9	6.3	6.4	5.4	5.0	4.8	4.2	3.9	2.4	n/o	
9	5.4	6.0	5.5	5.8	5.2	5.2	5.1	5.1	4.6	4.2	3.6	2.8	1.5	
10	3.6	3.9	3.8	3.7	3.5	3.5	3.4	3.8	3.3	3.2	3.0	2.0	n/o	
11	1.8	2.3	1.9	2.0	1.8	1.8	1.8	2.0	2.1	1.6	1.6	1.4	n/o	
12	0.7	0.9	0.7	0.7	0.6	0.6	0.6	0.7	0.9	0.8	0.7	0.5	0.3	

n/o = not obtained

TABLE: 1 - 11

Index: *mt* (deciduous teeth missing due to caries) 1963-1973, 1979, 1988
(after Barnard, 1988).

Group All Schoolchildren		Index: <i>mt</i> (deciduous teeth missing due to caries)											
Age Group (years)	1963	1964	1965	1966	1967	1968	1969	1970	1971	1972	1973	1979	1988
4	.39	.58	.38	.33	.28	.15	.11	.15	.16	.03	.2	-	n/o
5	.72	.7	.6	.43	.52	.39	.26	.29	.24	.25	.18	.22	negligible
6	1.25	1.14	1.19	.94	.78	.83	.69	.49	.53	.41	.47	.37	negligible
7	1.71	1.70	1.47	1.46	1.27	1.02	.95	.89	.68	.62	.52	.48	n/o

n/o = not obtained

TABLE: 1 - 12

TANWORTH DENTAL SURVEY 1963 - 1973, 1979, 1988 (after Barnard, 1988)

Group All Schoolchildren Index: % Children with Deciduous Teeth Extracted due to Dental Caries													
Age Group (years)	1963	1965	1965	1966	1967	1968	1969	1970	1971	1972	1973	1979	1988
4	12.0	21.1	14.1	12.8	7.5	6.7	4.8	8.5	5.4	2.0	6.7	-	*
5	21.1	20.3	17.8	16.1	14.7	14.0	9.2	9.0	9.5	8.5	8.7	6.8	2
6	37.2	32.8	34.7	28.0	24.1	23.9	22.4	17.2	17.3	15.2	16.4	14.0	3
7	48.4	48.1	42.9	47.7	37.3	35.8	31.3	31.8	24.0	25.7	20.9	20.2	*
8	57.1	56.7	57.2	53.2	51.0	44.4	40.6	38.4	34.6	31.4	27.5	19.8	*
9	50.5	60.9	49.2	53.2	45.8	47.6	42.2	41.7	39.9	32.8	28.3	24.4	*
10	38.9	40.2	38.7	39.0	30.3	27.9	30.9	37.6	30.6	31.1	26.1	15.4	*
11	21.9	31.0	19.6	24.7	17.1	16.9	18.5	26.1	21.8	16.4	15.0	11.5	*
12	9.1	14.4	6.7	8.2	3.8	7.2	5.6	10.1	9.2	8.7	6.9	4.3	*

n/o = not obtained

TABLE: 1 - 13

TANWORTH DENTAL SURVEY 1963 - 1973, 1979, 1988 (after Barnard, 1988)

Group All Schoolchildren		Index: % Children Caries Free deciduous Dentition (% dmft = 0)											
Age Group (years)	1963	1965	1965	1966	1967	1968	1969	1970	1971	1972	1973	1979	1988
4	21.7	15.8	26.6	25.6	43.0	38.1	61.4	57.4	57.0	55.9	66.7	-	n/o
5	16.5	12.6	14.6	16.1	27.4	31.9	38.8	42.5	43.9	51.3	43.4	51.4	64.0
6	7.5	9.5	10.9	9.8	18.4	22.1	24.2	29.0	33.3	34.9	42.4	45.4	63.0
7	6.5	7.0	6.3	7.7	10.2	15.4	18.3	19.5	25.5	26.8	29.5	40.7	n/o
8	5.5	4.8	3.5	6.0	8.1	8.5	13.1	13.4	0.2	22.4	20.8	36.8	n/o
9	9.4	6.5	8.0	6.8	9.8	11.3	8.9	10.6	17.4	16.0	21.8	28.4	54
10	20.2	18.0	20.8	20.5	21.9	23.4	22.1	21.2	13.2	25.3	27.2	38.2	n/o
11	47.2	37.9	46.9	44.3	48.8	46.8	48.99	43.0	22.8	47.7	47.6	53.3	n/o
12	72.0	64.9	74.6	71.5	73.3	72.0	74.9	72.4	41.9	70.1	78.9	76.3	99

n/o = not obtained

TABLE: 1 - 14

TANWORTH DENTAL SURVEY 1963 - 1973, 1979, 1988 (after Barnard, 1988)

Group All Schoolchildren Index: Decayed, Missing or Filled Permanent Teeth/Child (DMFT)													
Age Group (years)	1963	1965	1965	1966	1967	1968	1969	1970	1971	1972	1973	1979	1988
4	0.1	0.2	0.1	0.1	0.0	0.0	0.0	0.0	0.0	0.0	0.0	-	n/o
5	0.2	0.2	0.2	0.2	0.1	0.1	0.1	0.1	0.1	0.1	0.0	0.0	0.0
6	1.3	1.4	1.2	1.3	0.9	0.8	0.6	0.5	0.5	0.4	0.4	0.1	0.1
7	2.5	2.6	2.3	2.5	1.8	1.8	1.3	1.4	1.2	1.0	0.9	0.4	n/o
8	3.1	3.6	3.2	3.2	2.7	2.6	2.3	2.1	1.7	1.5	1.4	0.7	n/o
9	4.0	4.3	4.0	3.7	3.2	3.1	2.9	2.8	2.5	2.2	2.0	1.1	0.3
10	5.0	5.5	4.9	4.8	3.9	3.8	3.5	3.4	3.0	2.9	2.5	1.4	n/o
11	6.4	7.0	6.9	6.8	5.3	5.0	4.59	4.3	4.0	3.7	3.3	1.8	n/o
12	8.4	9.3	8.7	8.7	7.0	6.5	6.2	5.6	5.0	4.7	4.3	2.4	0.9
13	10.3	11.4	10.8	10.2	8.9	8.5	7.7	7.4	6.4	6.1	5.5	3.1	n/o
14	11.6	12.9	12.6	11.4	10.0	9.9	9.2	8.9	8.0	7.2	6.6	4.1	n/o
15	12.5	13.7	13.9	12.9	11.2	10.9	10.3	10.2	9.0	8.5	7.7	4.6	1.5
16	13.3	14.0	14.5	13.7	12.0	12.0	11.0	10.7	9.9	9.3	8.7	5.1	n/o

n/o = not obtained

TABLE: 1 - 15

TAMWORTH DENTAL SURVEY 1963 - 1973, 1979, 1988 (after Barnard, 1988)

Group All Schoolchildren Index: % Children with Permanent Teeth Extracted due to Dental Caries													
Age Group (years)	1963	1965	1965	1966	1967	1968	1969	1970	1971	1972	1973	1979	1988
4	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	-	n/o
5	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	n/o
6	0.0	0.0	1.0	0.7	0.0	0.2	0.0	0.0	0.0	0.0	0.0	0.1	0.0
7	0.5	1.0	0.7	2.1	0.8	1.5	0.7	0.6	0.6	0.2	0.0	0.4	n/o
8	4.7	4.3	2.7	3.9	6.4	1.7	2.5	1.4	1.1	1.2	0.7	0.7	n/o
9	10.0	7.8	5.4	5.4	10.0	6.7	4.0	4.3	4.1	2.5	1.8	1.1	1.0
10	15.3	16.3	8.3	11.9	9.7	12.7	9.8	4.4	6.4	5.5	3.4	1.4	n/o
11	18.7	18.0	19.3	13.5	17.1	16.7	16.89	12.2	8.4	8.6	4.1	1.8	n/o
12	35.5	26.2	23.9	30.1	22.7	25.6	20.9	18.9	13.8	13.9	4.3	2.4	2.0
13	42.2	39.5	33.2	33.7	29.2	31.3	30.1	26.8	19.4	21.3	9.4	3.1	n/o
14	52.0	44.5	45.6	35.7	37.3	40.3	32.2	33.0	29.1	25.1	12.3	4.1	n/o
15	49.2	50.6	41.2	47.6	35.6	39.3	42.8	30.0	20.4	31.0	11.5	4.6	1.0
16	51.5	39.4	15.0	33.6	34.6	39.4	34.3	34.4	29.5	28.1	15.6	5.1	n/o

n/o = not obtained

(vii) SUMMARY AND CONCLUSIONS OF PART I

Part I of this thesis has dealt with current information on fluoride and the respective modes of action. At the time of *Peel's thesis* (1973) such information was not available.

Additionally, two modes of action have been explored in greater detail, namely "*altered*" tooth morphology; and anti-bacterial intraoral ecological changes. The possibility and implication of improved tooth morphology producing malocclusion reductions will be discussed further in Part II.

Further, earlier and current studies of fluoride's effectiveness (in its various vehicles and compounds) in reducing dental caries were presented so that the reader could better interpret contemporary results.

The existence and importance of the secular trend of decay reduction were highlighted, along with the effect this trend has on interpretation of fluoridation surveys. The likely causes were pinpointed; chiefly the increased use of fluoride dentrifices from the later 1970s. Thus, most children from a very early age are receiving fluoride both systemically and topically whether they live in a water-fluoridated or non-water-fluoridated area. In non-water-fluoridated areas most young children receive a systemic effect due to swallowed toothpaste as outlined by *Ophaug and Singer (1988)*. Additionally, they receive the usual topical effect from the dentrifice during brushing. In fluoridated areas, it is likely that some increases in dental fluorosis are due to the early ingestion of fluoride dentrifice along with normal ingestion of optimally fluoridated water (*Ophaug and*

Singer, 1988).

Finally the TAMWORTH FLUORIDATION SURVEYS have been outlined along with a presentation of results from *Tamworth 1963-88 and Lismore 1989.*

These results show that DMFT scores for 12 year-olds in *Tamworth* have declined from 2.4 to 0.9, from 1979 to 1988. For 15 year-olds, DMFT scores dropped from 4.6 in 1979 to 1.5 in 1988. This approximates a 60% reduction in DMFT scores in both age groups between 1979 and 1988. On the other hand 12-14 year-old *Lismore* children examined in 1989 in the same age range had combined DMFT scores of 2.0. There is a difference between the 2 towns of around 40% for combined DMFT scores for the combined age groups. Additionally, for *Tamworth* for the years 1963-1988, there have been significant reductions in the dmft and mt scores, and percentages of children with deciduous teeth extracted due to caries. As well, there have been significant increases in the number of children with caries-free deciduous and permanent dentitions. The difference between scores in 1973 and 1988 is particularly marked. Considering that 1973 was the last time a major malocclusion assessment was made in *Tamworth*, it will be interesting to see whether this further decline in DMFT scores is reflected in a reduced incidence of malocclusion from 1973 - 1988.

PART II

MALOCCLUSION

i) AIMS OF PART II

Part II of this thesis is intended to -

- 1) Highlight the difficulties in distinguishing normality from abnormality; and "*ideal*" from "*normal*". These will be discussed with respect to the problems in defining malocclusion and its significance.
- 2) Highlight the reasons for gathering data on malocclusion prevalence and types.
- 3) Discuss recent attempts at improving malocclusion recording through indices and models.
- 4) Present critical appraisals of the 3 methods of malocclusion assessment used in the TAMWORTH and LISMORE MALOCCLUSION SURVEYS.
- 5) Discuss the effects of unrestored caries and premature loss of deciduous and permanent teeth.
- 6) Discuss the possibilities for the prevention of malocclusion.
- 7) Discuss the role of fluoride in preventing malocclusion. Reference will be made to previous controlled studies including the TAMWORTH MALOCCLUSION SURVEYS.

ii) WHAT IS A MALOCCLUSION?

In his 1973 thesis, Peel (p 8) discussed the definition of malocclusion. From the range of definitions discussed it is obvious that he believes malocclusion is a continuum of conditions

"which may range from a slightly rotated lower incisor to that of a gross skeletal and dental anomaly".

Peel also feels that controversy and limitations in formulating and agreeing on certain parameters of malocclusion measurement are due in a large part to a lack of clarity regarding what constitutes a malocclusion. This is supported by Houston (1983) and Graber and Swain (1985).

Unfortunately, it seems that orthodontists, and dentists in general, still regard a malocclusion as any deviation from ideal rather than a range of tooth/jaw relationships within a standard deviation of ideal. Even this may be putting an arbitrary value on normality. Maybe there should be more patient input in deciding whether a malocclusion is even in existence or not. Once the patient is convinced that they have a malocclusion then maybe this is the time for us to comment and classify.

Proshek et al (1978) thought that treatment required for each particular case might act as a determinant in defining malocclusion.

The World Health Organization (1962) cited by Foster and Day (1974) defined malocclusion under the heading of *Handicapping Dentofacial Anomaly*. This is defined as an anomaly which causes disfigurement or which impedes function, and which requires treatment

" if the disfigurement or functional defect is, or is

likely to be an obstacle to the patient's physical or emotional well-being".

Salzmann (1968) defines a handicapping malocclusion as one which adversely affect aesthetics, function or speech.

Houston (1983), p 1) discussed malocclusion, ideal occlusion and normal occlusion. Ideal occlusion is a hypothetical concept based on the anatomy of the teeth. It is rarely if ever found in nature. However, it provides a standard by which other occlusions can be judged. Normal occlusion is commonly described as

"an occlusion within the accepted deviation from the ideal"

This, unfortunately, gives no clear limits to the range of normal. In general, minor variations in the alignment of the teeth which are not of aesthetic or functional importance are consistent with a normal occlusion.

Thus malocclusion is an irregularity beyond the accepted range of normal. The dilemma, however, as *Houston* admits, is determining the "cut off" point between normal occlusion and malocclusion. *Houston* adds that patient factors are of concern in this definition.

Here he brings in treatment as a possible element involved in malocclusion. He raises patient factors such as likely benefit from treatment, possible co-operation and interest, compromised function and aesthetics.

Foster (1974), p 19) emphasises that most malocclusions do not have a pathological basis and are thus

"variations in the range of normal"

Peel (1973) cited *Graber (1972)* as stating that normal in biological studies is a range, never a point. From these reasonings then, maybe one could argue that malocclusions are rare, but objective indices (which are based on ideals) have found the opposite.

To complicate matters when discussing normality *Foster and Day (1974)* actually found Class II dental relationships and crowding were more the "*normal*" than Class I, uncrowded dentitions in one *English* area they studied.

Foster (1974, p 34) then goes on to cite conditions which for him, help to define malocclusions:

- adaptive postural positions
- translocated closure from rest, or from an adaptive postural position to centric occlusion
- tooth positions necessitating adverse reflex avoiding mechanisms during articulatory function
- damage to oral soft tissues
- predisposition to periodontal and dental disease
- adverse personal appearance due to tooth position
- tooth position interferes with speech.

These points he says, serve as a basis of treatment-need.

Massler and Frankel (1951) also arrived at the view that ideal versus normal occlusion should be differentiated. They felt the term malocclusion should be reserved for patients actually requiring orthodontic treatment, and for any case having more than 10

malpositioned teeth according to their criteria.

Normal was reserved for less than 10 malpositioned teeth, or where teeth were very slightly out of perfect alignment.

Graber and Swain (1985, pp 11,13) state that normal occlusion and malocclusion must be visualised as overlapping frequency distributions of various morphological traits. Thus, varying degrees of occlusal harmony or disharmony must be understood as portions of a continuum rather than discrete entities.

Perhaps "*degrees of occlusal harmony*" should be used rather than the polar terms occlusion and malocclusion. They go on to say that malocclusion should be used where there is a need of active orthodontic intervention. Then stems the problem of who determines "*need*", and how?

Prahl-Andersen (1978) also mentions that our traditional views of malocclusion represent any deviation from ideal, rather than deviations around a normal value. Additionally, there is little consideration of patient sociopsychological factors in making this assessment. This is supported by *Freer et al (1973)* and *Bowden and Davies (1975)*.

Gray and Demirjian (1977) also struggled with the problem of defining malocclusion and cautioned that any definition should be time - or age -dependent, since some malocclusions may worsen or improve through the mixed dentition period and beyond.

From the writings of many authors, it appears a difficult question - what is a malocclusion? It would seem that to date no definition effectively covers the term "*malocclusion*". This lack of agreement

thus extends to indices for assessing malocclusion prevalence and severity. This aspect will be more fully covered later in this thesis.

iii) WHY ARE WE INTERESTED IN RECORDING MALOCCLUSION?

Helm (1973) answered this question by stating three reasons -

- i) to assess the prevalence of malocclusions, and this author would assume the types of malocclusions.
- ii) to assess need of treatment and the type of need.
- iii) to organise treatment methods and resources.

Foster (1979) answered this question another way: he felt it was important to assess and document malocclusion to enable:

- i) clinical treatment regimes to be planned
- ii) treatment need assessment, prioritising patients and/or conditions
- iii) resource planning - manpower and material infrastructure
- iv) funding arrangements and estimates.

Prahl-Andersen and Moore (1977) discussed the recent emphasis on, and concern for elevating the total health level of the World's population. This is coupled with increased public awareness of the possibilities and benefits of professional health care. These feelings have thus increased demand upon these services. Orthodontics is no exception.

More and more, professional health care is perceived as being a right and not just a privilege. This notion is being felt in the provision of orthodontic care world-wide. This is especially so in many developed countries where caries and periodontal disease reductions have diverted peoples' attentions to improving the aesthetics of their

occlusions especially.

Prahl-Andersen and Moore (1977) discuss the need to determine an orthodontic needs-decision model. They base their model on:

- a) Normative need. This is objective professional evaluation.
- b) Comparative need. This relates the patient to social norms for facial and dental appearance.

Additionally one might add to all of the reasons, above, the following:

- a) to try and implicate or pinpoint causes, and other effects, which may be preventable or able to be reduced.
- b) to assess the efficacy of past and present treatment and preventive programs.

Most of the reasons given for documenting malocclusion would seem on the surface to be applicable only to public health orthodontics, but several of the reasons are especially important to private practice orthodontics. Chief among these are the need to be able to classify and treatment-plan malocclusions on the individual level. Also, the necessity of having to communicate malocclusion details of a particular case to colleagues is equally important.

Grewe (1973) felt that when assessing occlusions we should be mindful of what we are trying to achieve - is it to determine:

- a) prevalence of different occlusal conditions?
- b) severity of malocclusion?
- c) treatment priority?

The problem, once documented, must then be worked through in the light of the approach to treatment taken by the health system, i.e.

i) all are eligible and all are treated;

ii) all are eligible but part are treated;

iii) part are eligible and part are treated.

He felt that ideal indices will come with time but he felt an ideal method of establishing treatment priorities in all situations will not come.

iv) HOW ARE MALOCCLUSIONS RECORDED?

a) Introduction

In his 1973 thesis, Peel reviewed the various malocclusion indices current at that time. He also discussed the different approaches and emphases that their originators employed in devising their respective indices.

It is the intention of the present writer to review only those malocclusion indices or approaches to recording malocclusion which have been devised since 1973. Discussion of these more recent indices should make the reader aware of changes in approaches to malocclusion recording, as well as continuing problems with indices, and possible future directions for malocclusion recording.

Following on from this discussion will be a critical appraisal of the 3 different methods of malocclusion assessment used in each of the Tamworth Malocclusion Surveys, and also at Lismore in 1989. These are the Angle's Classification, Subjective Assessment of Treatment-Need, and the Godfrey Index (GI).

b) Methods of Recording Malocclusions since 1973

Eismann (1977) felt that conventional indices for measuring malocclusion really only measured dysmorphological traits and that it is difficult to translate this information into treatment-need. Largely this must be determined subjectively with the aid of information gathered from measurement of these traits above.

He feels that until an objective method of determining

functional, aesthetic and mental status exists then we are bound to use these morphological traits only as a guide to treatment-need.

His "*index*" is largely analogous to that of *Björk, Krebs and Solow (1964)*. However, using his experience with malocclusion assessment and treatment he has set limits to arrive at groupings from 0 (regular dentition) to IV (severe malocclusion). Group 0, 0 points ranges up to Group IV - 66 points or more. His calculations for most populations rate treatment-need above 15 points generally. This means that only mild malocclusions are excluded from treatment.

One should read this in the context that much of what Eismann talks about relates to the *German Democratic Republic* where all orthodontic treatment is free but generally confined to removable appliances.

This indexing system he puts forward appears to offer no advantage over earlier indices.

Lundstrom (1977), at the invitation of the *Swedish National Board for Health and Welfare*, attempted to upgrade the Board's indexing system which indicates various levels of orthodontic treatment-need.

Lundstrom developed in its place an "*indication*" (for treatment) index. He stresses that this treatment does not necessarily have to be orthodontic in nature. He used the form shown below (FIG. II-1) to set out the factors which he felt important. Beside each factor an indication-for-treatment score would be placed.

Degree 1 is used when a moderate disturbance is considered to exist and when there is a possibility of more serious consequences arising from the existing disturbance.

Degree 2 denotes manifest disturbances.

Degree 3 denotes urgent need-for-treatment.

For the scoring of "*desire for treatment*":

Degree 1 denotes little interest

Degree 2 denotes marked interest

Degree 3 denotes intense interest

Cases with the greatest number of *Degree 3* scores are naturally considered in need of most urgent treatment.

He concedes that evaluations of this kind may be difficult in many cases due to our limited knowledge of the consequences of malocclusion. This is supported by *Jago, (1974); Shaw et al, (1980); McLain and Proffit, (1985).*

FIGURE II - 1

AFTER LUNDSTROM (1977):

Treatment Indication	Degree of indication (actual or predicted)		
	1	2	3
Mastication deficiency			
Speech defect			
Mouthbreathing			
Oral Health			
Caries susceptibility			
Periodontal disease			
Mandibular dysfunction			
Root-resorption			
Follicular cysts			
Traumatic injury			
Prosthetic considerations			
Aesthetic disturbance			
Sum total:			
Special observations			
Desire for treatment			

The ranking procedure presupposes that the same weight should be given to all eleven indication factors. Lundstrom felt that with present knowledge we really have no way of discriminating between factors on a weighting basis. This is also supported by (Jago, 1974) and Shaw et al (1980).

Lundstrom compared his system with that of the Board's index after the completion of clinical trials.

From the point of view of diagnostic output, he felt his index

superior since more evaluation was done and information was gathered.

He found little difference between specialist operators using his system. This was slightly better for the Board's Index which is simpler to apply but yields less information.

The Board Index produces 4 groups without any instructions on how to rank cases intragroup. He felt his "*indication*" index could allow reasonable subgrouping.

Registration time for his Index is an average of 2 minutes longer. He felt this is of no great concern overall, in the context of overall treatment time. Both indices he felt, could be improved. He felt his Indication Index was amenable to change and may even stimulate research which may precipitate such changes.

The Indication Index tries to be objective and to include all aspects of assessing a malocclusion, i.e. physical factors, aesthetic factors and attitude-to-treatment factors. However, this author feels that true objectivity is difficult when assessing the aesthetic and treatment-desire factors in the way Lundstrom gathered his information. Firstly, the patients were seen in a clinical environment which may have altered patient responses. Largely, the method still relied on some subjective input in scoring these last 2 factors. Lundstrom concedes as much himself.

Prahl-Andersen and Moore, (1977) and *Prahl-Andersen, (1978)* developed a treatment-need-decision model from the use of line

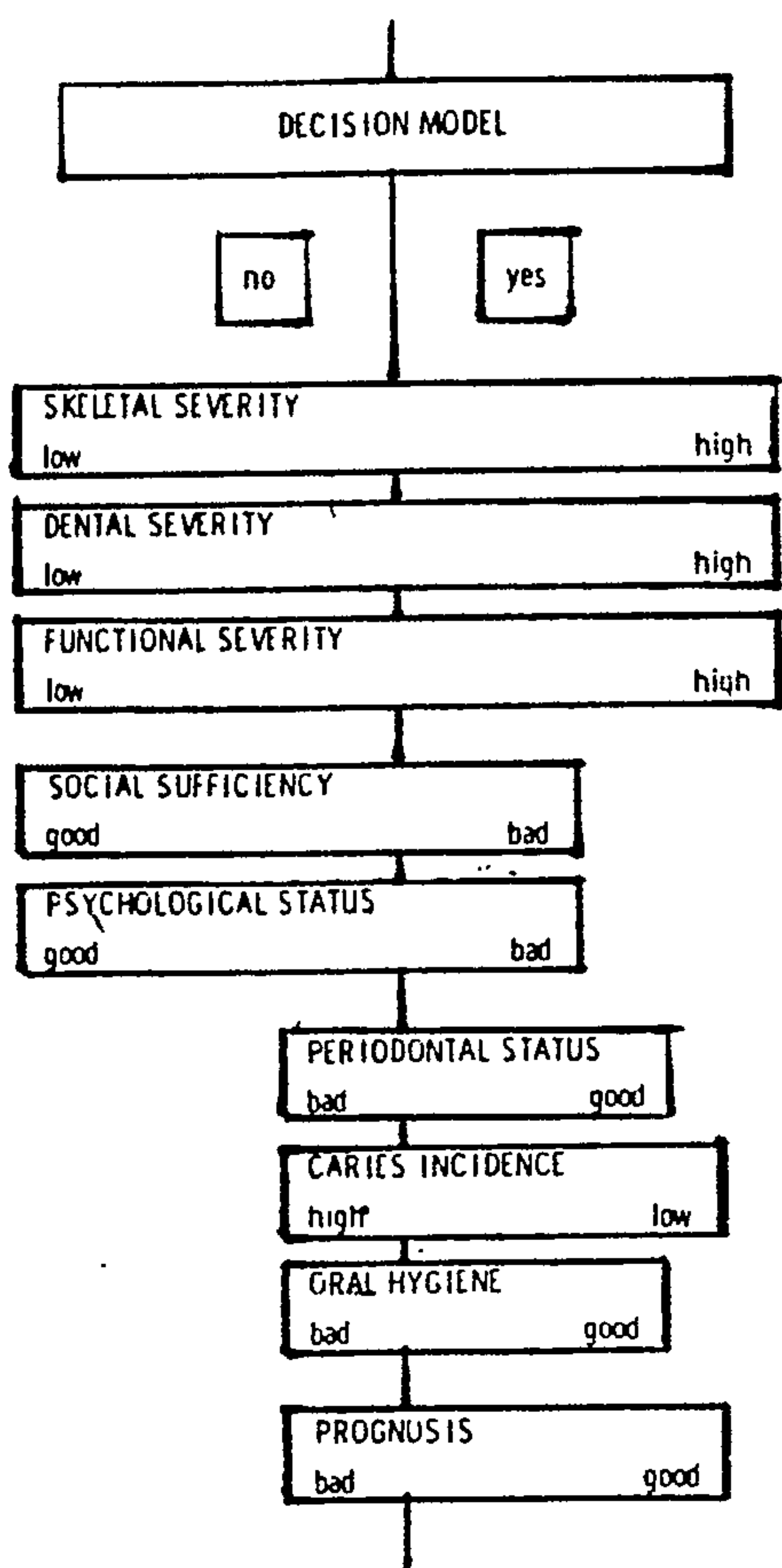
drawings of various profiles and the use of photographs of smiling lips/teeth displaying varying occlusal patterns.

They felt that such models at this must be flexible to adapt to the changing needs and attitudes of the population group to be served. Decision Model Factors were selected and weighted according to professional criteria. This author feels the validity of the professional criteria is questionable in the light of previous and current thought (*Jago, 1974; Shaw et al, 1980; McLain and Proffit, 1985; and Shaw et al, 1986*).

Cut-off points between treatment and non-treatment must be negotiated according to the "*climate*" the model will be used in. For example, underdeveloped countries will naturally set higher cut-off points if their financial and manpower resources are limited. According to the authors, these cut-off points can be easily calculated given accurate information on future manpower prediction, current child populations, birth and death rates.

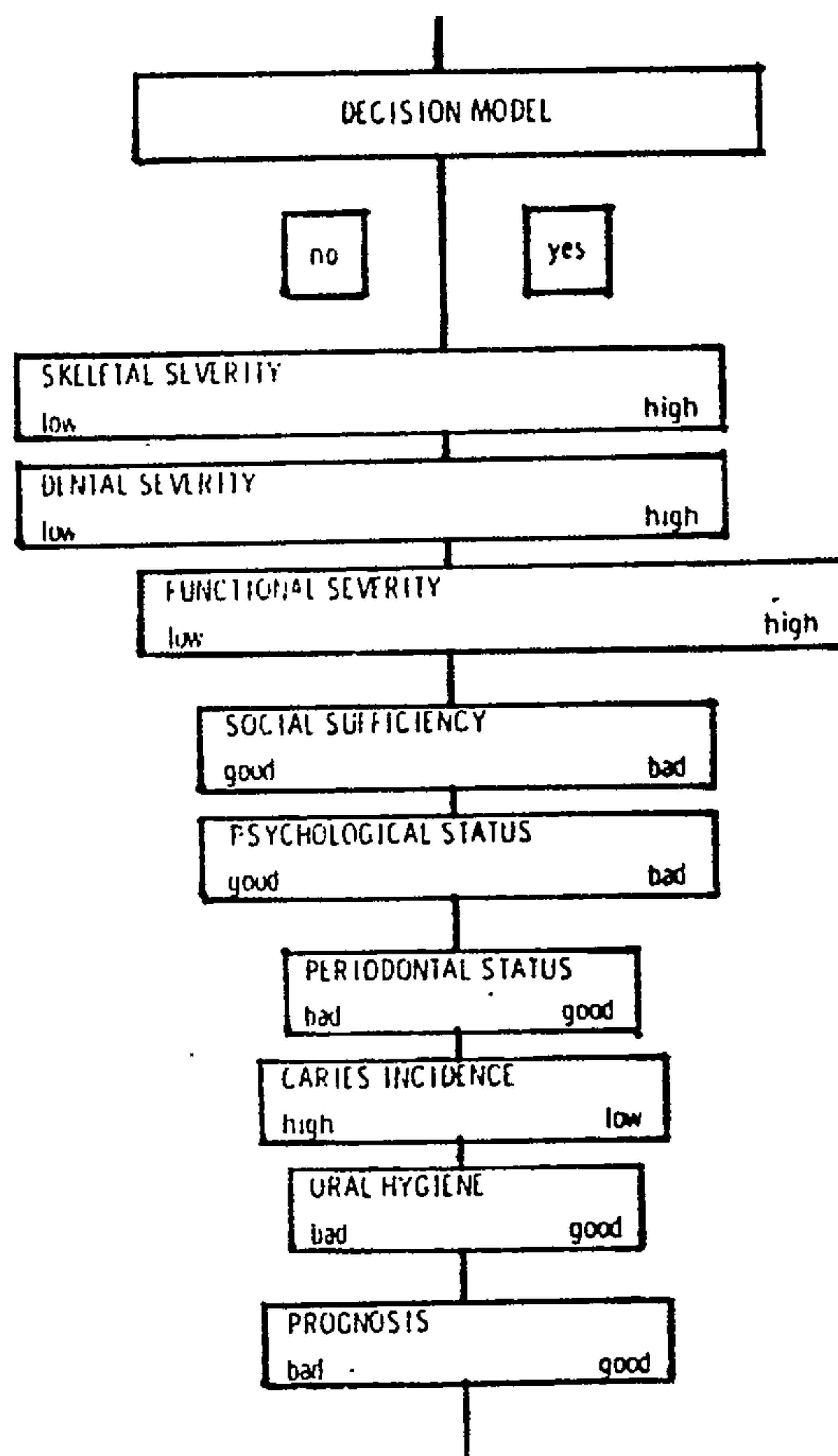
Patient scores are ranked along a continuum in each cell. The cells and their assessed factors are pictured below (Fig. II-2) As mentioned, cut-off points and weightings, as seen from the relative skewing and size of cells iv variable and adaptable to the population it is to be used on.

FIGURE II-2



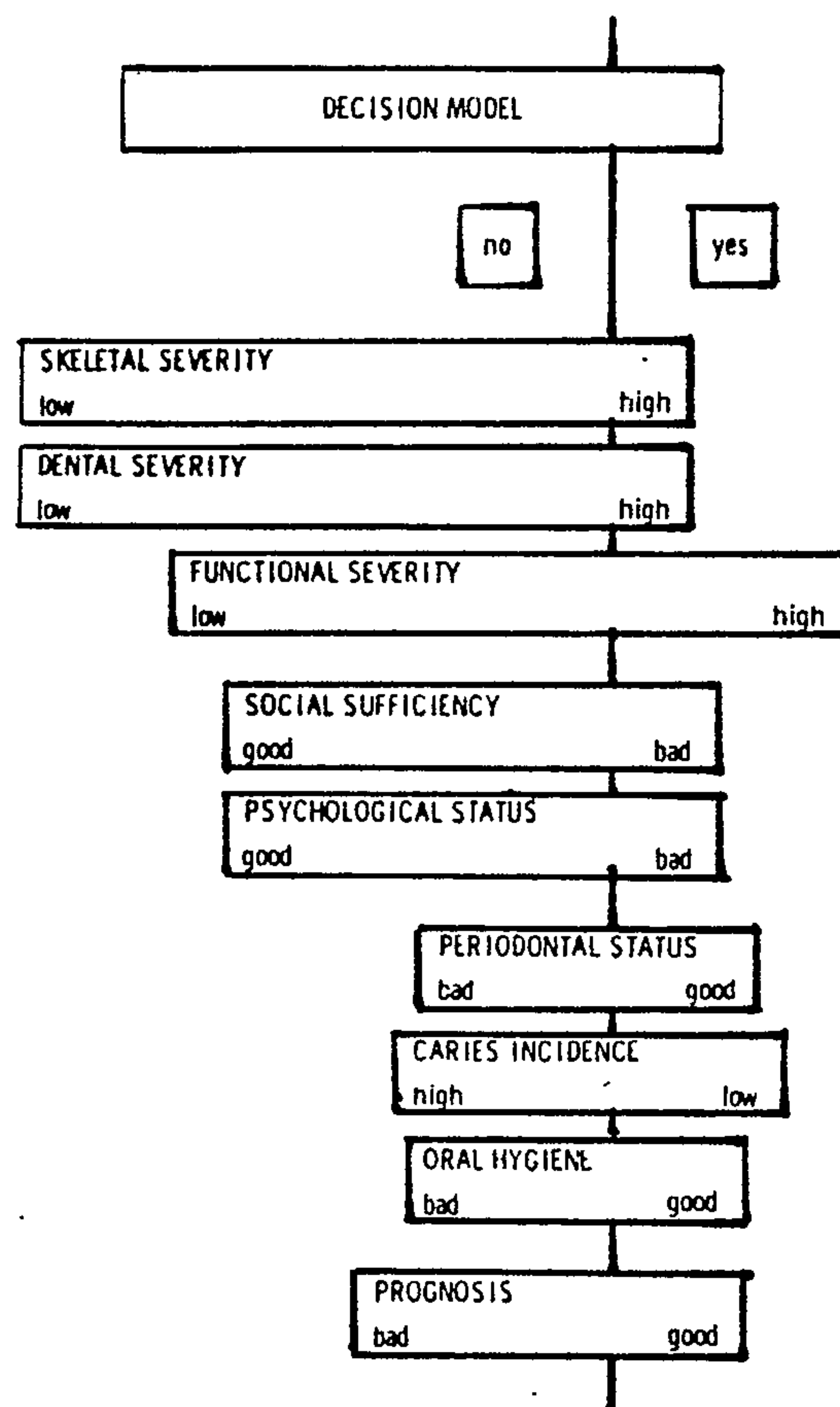
Patient clinical criteria for a decision model.

FIGURE II-3



Weighted clinical criteria for a decision model in a developed country.

FIGURE II - 4



Weighed clinical criteria for a decision model in an undeveloped country.

It appears the authors have originated and tried to validate this model. They contend the model could be customised to the target group and then used as a more concrete index of treatment-need. As with other indices, there has been a *"subjective"* assessment as to what factors should be included to be assessed. This may later be found to be invalid.

The schema is comprehensive in that it does not dwell totally on recording occlusal traits only, but includes such factors as skeleton, function, social sufficiency, psychological status and even prognosis. However, several of these are still assessed somewhat subjectively.

Campbell-Wilson (1985) reported on a specially developed index to satisfy health authority screening needs with respect to severe malocclusions. The index had to be very quick and simple with no special training required. These conditions were imposed due to economic and manpower constraints.

This screening index for potentially severe malocclusion was carried out at 9 years of age.

The following was placed on the patient's general dental record card as a stamp:

O.J. more than 10 mm	
Soft tissue trauma	
<u>3/3</u> not palpable	
<u>1/1</u> unerupted	
6s poor	
Nil Relevant	

Only positive responses are recorded.

This author feels that such a screening index would detect those most at need. Any treatment would obviously depend on patient/parent/provider factors.

This author would also suggest the possible inclusion of:

- i) abnormal permanent tooth wear;
- ii) anterior crossbite

Results of screenings with this index indicate that approximately 4.5 - 5.5% of children fit one or more of the categories of the index. Campbell-Wilson notes that this is more a screening tool than an epidemiological tool. She also concedes that the incorporation of a severe crowding indicator into this tool was found difficult. This author questions the use of such a crowding indicator for 9 year-olds anyway. *Hiles (1985)* questions the economics of a dental officer performing such screenings routinely for 10 year-olds. However, this author feels that such a service performed by dental therapists, as part of a normal examination, may be cost-effective.

The Dental Aesthetic Index (D.A.I.) [*Cons, Jenny and Kohout, 1986*].

This contemporary malocclusion index system was published in 1986 and focusses its attention on the aesthetic aspects of occlusal traits.

The proponents of this type of index argue that most patients seek orthodontic treatment primarily for aesthetic reasons and less commonly due to functional problems associated with the masticatory system. Rarely do dire physical consequences follow the lack of orthodontic treatment.

Increasing evidence from social psychology studies shows that unacceptable dental (and one would assume skeletal) appearance when compared with societal norms may affect the individual's socialisation process.

This may:

- 1) stigmatise the individual;
- 2) impede career chances;
- 3) affect peer group acceptance;
- 4) encourage negative stereotyping;
- 5) adversely affect self concept.

Based on these findings, the authors developed a regression equation that relates objective measurements of certain occlusal traits to societal norms of facial appearance.

The underlying concept is that the more a person's dental appearance deviates from social norms, the more likely it is that the person will experience social handicaps, and therefore need orthodontic treatment. Thus the greater the D.A.I. Score, the greater the likely need of orthodontic treatment.

The index can be used to rank individuals in terms of urgency for public orthodontic programs. It can also be used as a decision-making tool by administrators for a given population. In this sense it can help establish the urgency of treatment need of the population and the general level of malocclusion frequency/severity in the community. This is underpinned by the fact that the D.A.I. is based on the contemporary finding

regarding the importance of aesthetics with respect to occlusion as perceived by the public.

The index is not a diagnostic tool, it is a screening tool. Eligible individuals uncovered by the D.A.I. are referred for diagnosis, clinical evaluation and subsequent treatment planning and treatment.

The authors claim that the D.A.I. is:

- (a) valid. Due to rigorous double cross-validation carried out in the D.A.I. development. It has been shown to be relevant cross-culturally;
- (b) objective. 10 orthodontic traits are objectively measured.
- (c) reliable. Inter-and intra-examiner reliability is good.
- (d) equitable. The scoring is completely unbiased and ranks only according to the severity dictated by the social norms used in devising the index.
- (e) inexpensive. It can be used by dental auxiliaries. No special instruments are needed. However, this author would recommend the use of graduated periodontal probes to simplify and speed up millimetre measures when assessing the D.A.I.
- (f) speedy. All 10 traits can generally be recorded in less than 3 minutes in the field. This author could also add that special clinical conditions are not essential to enable good recordings.

Suggested additions to the recording form are that of previous residency history, and social class as determined by parents' occupations. Also, in cases such as this thesis, history of fluoride intake in its various forms (*Craig, 1989*).

Urgent, severe and/or traumatic conditions that are untreated at the time of evaluation, are registered and should be referred for further specialist evaluation.

A prior or present history of any type of orthodontic intervention is noted. This may give valuable information on the actual utilisation of orthodontic care past and present in the target group. As an aside, interesting information would be obtained if the various occlusal characteristics that promoted patients/parents to seek treatment could be obtained.

Following on, the D.A.I. then registers:

- 1) The total number of missing teeth mesial to the first permanent molars where spaces remain in both the maxillary and mandibular arches.
- 2) Incisor crowding. Whether present or not:

0	=	no crowding
1	=	1 segment crowded
2	=	2 segments "
- 3) Incisor spacing. Whether present or not:

0	=	no segment spaced
1	=	1 segment spaced
2	=	2 segments "
- 4) Maxillary median diastema if present, is measured to the nearest whole millimetre.
- 5) The largest rotation and/or labio-lingual displacement in the maxillary anterior segment in millimetres.
- 6) The largest rotation and/or labio-lingual displacement in the mandibular anterior segment in millimetres.
- 7) The largest anterior overjet (in mm).
- 8) The largest reverse overjet (in mm).
- 9) Anterior openbite (in mm).
- 10) Anteroposterior molar relationships in either direction (i.e. pre-or post-normal).

0	=	normal
1	=	½ cusp irregularity
2	=	full cusp irregularity

One should always assign the lesser score when any doubt exists.

Traits are totalled as per the form and a score arrived at. Scores for interest groups can then be compared with larger group scores to enable ranking, severity assessment and prevalence assessment by appropriate authorities.

This author (R.M.) feels one weak point is that skeletal problems in the dentofacial region are not assessed. This is a problem in most malocclusion indices. They can be inferred from the severity of certain trait scores, such as incisor overjet and molar relationship (but only indirectly).

The other criticism of lack of completeness is related to the D.A.I.'s concentration only on 10 aesthetics-related traits without allowing the opportunity to register other orthodontically-interesting traits which may be of use to planners and treaters. Such items as midline deviations, crossbite, degree of anterior crowding and posterior crowding. Granted, the D.A.I. is primarily an aesthetics-related index.

This author also takes issue with the registration of openbite as an aesthetically-compromising trait. *Prahl-Andersen (1978)* in her paper found little aesthetic preference was expressed by lay-raters with respect to the presence or absence of openbite. An obvious contrast with the D.A.I. and its basis.

Brook and Shaw (1989) state that it is important to distinguish those indices which:

- 1) classify malocclusions into types such as the Angle's Classification;
- 2) record prevalence of malocclusions in epidemiological studies;

- 3) attempt to record treatment-need and priority;
- 4) record treatment-success or treatment-difficulty.

Brook and Shaw's interest was in developing an orthodontic treatment priority index which concentrated on the dental health and functional indications for treatment; as well as the deviation from social norms which may accompany a malocclusion.

Dental Health Component

The index of treatment priority used by the *Swedish Dental Board* was used as the basis for grading the functional and dental health indications for treatment.

There are 5 grades; grade 1 representing little or no need for treatment and grade 5 representing great need-of-treatment.

An attempt was made to try to establish from the literature meaningful values for cut-off points between grades for each occlusal trait that represents a quantifiable threat to the dentition.

Most traits are recorded using a millimetre rule. Crowding was recorded by measuring the largest displacement between teeth in the arches. In use, only the highest scoring trait need be recorded as this determines the grading of the patient.

Aesthetic Component

Aesthetic impairment was recorded using the *SCAN INDEX (Standardised Continuum of Aesthetic Need) (Evans and Shaw, 1987)*.

This scale was constructed using dental photographs of 1000 12 year-olds collected during a large multidisciplinary survey. Six non-dental judges rated these photographs on a visual analogue scale. At equal intervals along the judged range, representative photographs were

chosen, giving a 10 point scale from 0.5 (attractive) to 5.0 (unattractive).

Brook and Shaw claim the index is easy and quick to apply and could be used by auxiliaries. Additionally, it had acceptable reproducibility. They feel that the index is able to be adapted as new information on aesthetics and dental health impairment, with respect to malocclusion, comes to hand.

The authors have not set cut-off points at which patients should or should not be offered treatment. (This would depend upon the amount of treatment resources available).

This author questions the validity of the use of dental photographs where the lips are retracted into an unnatural state. This is hardly the situation a lay-person would be facing when unconsciously assessing a person's aesthetics in a normal situation in social interactions. This author feels that dental aesthetic impairment results as much from tooth lip interaction as it does from tooth position *per se*. Here the D.A.I. (*Cons et al, 1986*) seems to "*fit the bill*" better, although it includes little that relates to a dental health component as with Brook and Shaw's index.

Brook and Shaw are at least attempting to use objectivity to measure both occlusal traits and aesthetic impairment, thus removing subjectivity from the assessment of treatment priority.

(c) Critical Appraisals of the Methods used to assess Malocclusions in the Tamworth Malocclusion Surveys (1963, 1973, 1988) and in Lismore (1989)

Angle's Classification - a critique

This classificaiton has been part of modern orthodontics for over 80

years and used extensively for both treatment-need assessment and malocclusion classification by both epidemiologists and orthodontists. However, the Angle's Classification suffers from several shortcomings.

Basically the Angle's Classification was devised to assess the clinical treatment required for the orthodontist; that is, a prescription for treatment. It was not meant as an epidemiological tool (Jago, 1974). Additionally Peel (1973) feels that the classification doesn't lend itself well to statistical breakdown.

Because this classification does not quantify the elements of the occlusal disharmony and can't measure the severity of disharmony, it should only be used as an indicator and not as an index of malocclusion (Peel, 1973; Jago 1974; Brook and Shaw, 1989). It neither implies the cause of the malocclusion or the effects. Neither is the possibility of jaw involvement determined.

Additionally, it has been criticised because it emphasises dental occlusion only and not actual tooth positions relative to the face (Bennett, 1912; Rinchuse and Rinchuse, 1989).

Angle's Classification was based on ideal occlusion which is the exception rather than the rule. Thus, use of the Angle's Classification alone registers between 75 - 95% of the population in a malocclusion category (Horowitz and Hixon, 1966). Here the concept of "ideal" versus "normal" occlusion needs clarifying. Unfortunately, normal occlusion is difficult to define whereas ideal occlusion is easily defined. Normal occlusion may vary not only racially but geographically; hence the problem is compounded (Jago, 1974; Foster and Day, 1979).

Difficulties also arise in applying the Angle's Classification in mutilation cases (Peel, 1973; Rinchuse and Rinchuse, 1989).

Confusion is often found with some Class II molar relationship cases, where different operators frequently have difficulty in allotting a case to Class II Div. 1 or Div. 2 (Gravely and Johnson, 1974).

There is no idea conveyed of crowding, vertical and transverse relationships with many malocclusions (Bennett, 1912; Peel, 1973).

Gravely and Johnson (1974) question the reliability of the Angle's Classification as well. They cite the large differences found for each of the different malocclusion classes in different studies of similar populations. They feel that a large part of this difference is due to inter-examiner variation in allotting subjects to the respective malocclusion classes.

They cite Ast et al (1962) as finding inter-examiner variability of 30% with respect to the Angle's Classification in their study.

On the other hand, Murray (1969) was able to classify 50 sets of models without disagreement on 2 separate occasions.

Gravely and Johnson (1974) concluded in their study that great difficulties arise when using the Angle's Classification when there are underlying arch asymmetries left to right. In this instance one side may have a Class II molar relationship while the other side may be Class III. Additionally, crowding and/or early deciduous tooth loss also can complicate a classification. These contentions are supported by Rinchuse and Rinchuse (1989). As well, some Class II malocclusions may rate Division 1 on one side and Div. 2 on the other side. In these situations examiner judgement must be used and it is here that

interexaminer errors arise.

Gravelly and Johnson also concluded that if measurements are to be made in different communities, they are more likely to be reliable and thus statistically valid if the same examiner is used. They felt that because of these problems with Angle's Classification, its diagnostic value to the clinician is questionable. Thus, its epidemiological use must be more questionable.

Katz' (1978) study found that in relation to oral self-image satisfaction, the Angle's Classification correlated the best of 8 malocclusion indices studied. This applied both to chi-square and linear regression analyses. *Katz* felt this finding demonstrated the ability of the Angle's Classification to relate to the different soft tissue facial profiles and anterior tooth positions which overlie the basic dental malocclusion categories. Since in a majority of cases these Angle's classes possess characteristic anterior and frontal appearance, it is understandable that these Angle's classes relate so well to lay-person assessments of their own appearance.

Also in defence of this method for assessing malocclusion; it is quick and usually easy in most modern occlusions that have not suffered the ravages of caries and subsequent tooth loss.

It essentially conveys a mental picture of the subject's appearance and some of the occlusal features that one might expect to see. There is also a hint of the potential treatment regime that might be followed.

In this respect, this author regards the Angle's Classification as a "valuable adjunct" rather than the chief means of assessing a malocclusion for epidemiological purposes.

Assessing Occlusions according to Subjective Treatment-Need - a critique

Being subjective, the obvious problem with this type of registration is its variability from assessor to assessor. This variability can change according to the assessor's background, that is whether a lay-person, subordinate health worker, general dentist or orthodontist. Additionally, the assessor's experience, zeal (*Albino, Lewis and Slakter, 1978*) mechano-therapy preferences, as well as the assessor's concepts of aesthetics and concepts of adequacy/normality of function and appearance may affect assessment of treatment-need (*Kohout et al, 1983; and Jarvinen and Vaataja, 1987*).

As mentioned earlier, the assessment of treatment-need is important for resource planning, staffing, funding and so on (*McLain and Proffit, 1985*).

Bowden and Davies (1975) highlight the great variation in the results of treatment-need assessed subjectively. They cite the need for standard criteria to ease this problem.

They feel that because treatment-need is a multifaceted entity i.e. function, static occlusion, skeleton and psyche; the subjective interactive effect of the orthodontist must have some place in assessing treatment-need.

In their study 25% of interexaminer disagreements were due to value judgements over what constituted a pleasing arrangement of anterior teeth, and the relationship of the teeth to lip musculature. Generally disagreements concerned the less severe malocclusions.

However, on the other hand, *Jenny et al (1983)* found that public and

professional perceptions approximate each other to a significant degree as regards assessments of severity and treatment-need. The reasons for this are probably explained by our socialisation norms, common to most members of society. Where differences arise, it is most likely due to clinical criteria such as dysfunction, potential for tissue destruction, and so on.

The speculative nature of these clinical criteria is also proposed as an area where differences may arise.

This author's own feeling is that for years dentistry has felt that most patients seek treatment for primarily aesthetic reasons to avoid adverse, social stereotyping, consciously or unconsciously; be it restorative dentistry or orthodontics. Now this feeling has been validated by several studies (*Adams, 1977; Albino, 1980; Albino et al, 1981*). Any person now involved in assessing orthodontic treatment-need is probably cognisant of these studies and their implications. This may very well now carry through to affect their criteria for assessing, or their overall judgement of, orthodontic treatment-need. That is to say, because we now know some Class II Div. 1 patients are likely to suffer negative stereotyping and reduced life success, then we are more likely perhaps than before to recommend treatment for them. However, bear in mind that previously we may have recommended treatment anyway due to likely improvement in lip function, general appearance and decreasing the chances of trauma to protruding incisors.

Draker (1960) had his feeling substantiated that an increase relationship between inter-and intra-examiner variation and malocclusion severity existed.

Freer et al (1973) found that for subjective assessments of treatment-need, that, the less severe the malocclusion, the greater was the variation exhibited both inter-and intra-examiner. This was also supported by *Bowden and Davies (1973)* earlier.

The impression this author gets is that it is preferable to include a subjective assessment of treatment-need with any objective assessment being attempted. This subjective assessment should be based on checking certain clinical criteria as outlined by *Jarvinen and Vaataja (1987)*. The study of *Jarvinen and Vaataja (1987)* revealed that different objective indices selected different children for orthodontic treatment. One might also say that the validity of each index in all cases is questionable if this occurred. It is here that this author feels the inclusion of a subjective assessment of treatment-need combined with an objective index would help to yield a more **"balanced"** overall assessment of treatment-need. This is supported in the conclusions of *Ingervall and Hedegaard (1975)*. Add then, the Angle's Classification as a guide to self-image perception as advocated by *Katz, (1978)* and a well-balanced overall index is created. The Tamworth Malocclusion Surveys conform to these criteria.

The Godfrey Index (GI) - a critique

This critique is intended to highlight adequacies/inadequacies of the Godfrey Index in its practical application and interpretation of the data collected. An explanation of the actual GI traits, recorded and their method of scoring, can be found in PART III.

A major fault of nearly all indices examined by this author is their ignorance in not including assessments of skeletal deviations. The Godfrey Index and other indices can, from crossbite, overjet, overbite

and molar relationship scores, infer a skeletal deviation. However, this can only be inferred and not confirmed. Additionally, the G.I. scoring may not reveal such a problem where the malocclusion has been largely compensated.

Suggested by the present author is some palpation method to assess skeletal deviation. Another option could be a "T" type clear plastic template that could have one axis for the soft tissue Frankfort Plane and another movable axis perpendicular to this. This would be placed on soft tissue Nasion. When viewed from the lateral aspect this could easily reveal maxillary and/or mandibular deviations in the sagittal and/or vertical plane.

The G.I. has no assessment of midline deviation. In a treatment-related sense this would be important.

The mandibular anterior crowding score really gives no general picture of the severity of displacement or rotation and thus may not allow fully for the extent of the cosmetic problem or the treatment difficulty of the particular subject. For example, all anteriors may be severely rotated, yet, only yield a low mandibular anterior crowding score. Yet, treatment difficulty may be much increased. Additionally, cosmetic problems may be great.

This author also feels that the crowding score of the upper anteriors should also be scored and perhaps the existence of a maxillary median diastema. A good number of occlusions have been seen by this author where the lower anteriors and lower arch in general may be in good alignment yet the upper teeth may be displaced and crowded possibly due to a Bolton's Ratio problem. In this situation the Godfrey Index mandibular anterior crowding component by itself would imply a better

occlusion than was actually present.

Godfrey (1990) explained that for him the condition of the lower arch is all-important to successful or not-so-successful achievement of orthodontic stability. He also took the view that slipped contacts of lower anteriors were the essential problem rather than the amounts of rotation. This is the reason for him concentrating solely on the lower arch for crowding. The present author is prompted to ask

" is there such an animal as orthodontic stability?

Whether there is or isn't is the basis of orthodontic survival" according to Godfrey

Good maxillary anterior alignment can also exist yet severe retroclination and *"relative crowding"* of the lower anteriors can exist. Again the interpretation of the scoring could lead to a false idea of treatment requirements in such a case.

In defence of the mandibular anterior crowding measure, however, this author considers this method of scoring circumvents having to make actual millimetre measurements which can give false interpretations of actual crowding where the subjects' teeth can vary in size markedly.

Additionally, the scale of scoring is easy even though somewhat subjective in its assessment for an objective measure of crowding. Also, the method appears to have sufficient fineness.

This fineness of measurement and use of proportions rather than millimetre measurements can also be put forward as pluses for the measurements of posterior crowding of the lower arch. Again, this overcomes the variation in tooth sizes between individuals. However, the non-inclusion of upper arch posterior crowding may give a false interpretation of the actual malocclusion present.

Similarly overjet and overbite scoring has adequate fineness. overbite also is assessed without using millimetre measures. Overjet is largely independent of tooth size in this author's opinion, hence millimetre measurements are valid here.

Openbite scoring would appear to be appropriate. However, no account is taken of posterior openbite. *Godfrey (1980)* explained that he did not include posterior openbite because of its minimal effect on aesthetics and because it was not readily observed by lay-people.

Buccal anteroposterior relationship scoring in this author's opinion is unnecessarily fine. This means that some occlusions that overall are "*nearly excellent*" may score higher than a less acceptable occlusion with crowding, crossbite or overjet. This is due to the scoring for normal molar relationship up to less than a $\frac{1}{2}$ unit deviation in either direction, being overly weighted in this author's opinion. The criteria are tighter than the Angle's Classification. *Godfrey (1990)* explained that he was looking for the stable locking of the occlusion here.

The scoring of posterior crossbite would appear to be appropriate. It is the present reviewer's opinion that there are anomalies between total Godfrey Index scores and the severity of the malocclusion. This becomes apparent when registering these scores in the field.

As previously mentioned, some occlusions that appear clinically good may actually fare worse than some occlusions with midline diastemas, upper anterior crowding and upper posterior crowding when using the G.I. It is conceded that there may be some subjective input here in what the examiner visualises as clinically good.

Without the use of the Subjective Treatment Need Assessment and the Angle's Classification, the type of malocclusion one would expect to visualise, can only be inferred from an assessment of each individual trait score.

The other problem if using the G.I. alone, is to work out the dividing line between mild occlusions and severe malocclusions from the G.I. score. Obviously, the greater the G.I. score the severer the malocclusion, but as previously mentioned it is this author's experience that mild and even very mild malocclusions may rate as high as 6 on the G.I. score. From this author's experience, G.I. scores above 6 generally require orthodontic treatment on the basis of their objectively-assessed traits deviating from orthodontic ideals. However, some severe cases such as gross crowding may register under this G.I. score yet not be interpreted as requiring treatment from the G.I. score alone.

In this respect then, comparison of G.I. scores alone with a larger population's G.I. scores may resolve difficulties here. Another approach is to compare G.I. scores with a Subjective Treatment-Need Assessment and use the comparison to resolve any difficulties. Again the problem arises of subjectivity and objectivity being married to answer a question that objectivity alone should answer. However, given the limitations and difficulties associated with devising the perfect, objective, all-encompassing, quick, simple, easy, orthodontic trait assessment, perhaps the decision on ultimate severity and treatment-need is best modified by some input from subjectivity.

Godfrey (1990) himself admits that the G.I. could have been better thought out, but this was limited by the time available, and the

limited availability of index models at the time, to prepare such an index for the first Tamworth Malocclusion Survey in 1963. Thus some of the weighting inadequacies are understandable. In the G.I. defence, it is quick, easy, objective and uses only one millimetre measurement.

Additionally, it is suitable for field use where no proper clinical conditions exist since only a mirror, a disposable millimetre measure stick (toothpick) and normal daylight are adequate to enable scoring. Also, since mainly only lower arch and anterior traits are registered (with the exception of buccal segment relationships), the measurements are easily obtained without a supine patient position.

It should also be pointed out that the G.I. was developed at a time when there was less orthodontist interest generally in the psychosocial inadequacies of the individual's occlusion with respect to aesthetics.

The severity, prevalence and thus treatment needs of orthodontic deviations of occlusal relationships which were emphasised in 1963 are now different today. Thus more emphasis is now placed on the aesthetic inadequacies of an individual's occlusion. Thus the D.A.I. (*Cons et al, 1986*) is probably more in vogue as a way of assessing treatment-need from the aesthetics aspect. However, the D.A.I. lacks information on traits that are always of orthodontic interest with respect to mechanics and function, for example, posterior crossbite and posterior crowding. This is where the G.I.-type indices help provide an overall picture of the malocclusion as it relates to its owner. This is especially so if these aesthetics-related indices are married to the more trait-related indices like the G.I. This approach receives support from *Brook and Shaw (1989)*.

The present writer reviewed *Katz' (1978)* findings on the Angle's Classification, finding that this related well to oral self-image perception. This is the other aspect of malocclusion not easily assessed objectively at this point in time. The D.A.I. supposedly assesses social adequacy of occlusion, whereas indices like the G.I. assess occlusal traits and morphology. *Grewe (1973)* feels that these are the three essentials of an ideal malocclusion assessment. However, he feels that self-image perception with respect to malocclusion is so variable that it may never reach the stage of being objectively rated consistently.

The present writer also felt that from reviewing the literature, that at present a Subjective Assessment of Treatment-Need may balance objective assessments which may not be totally valid for all malocclusion situations. Given that the G.I. really is a documentation of occlusal traits in an objective way, then maybe the three types of measures used for the **Tamworth Malocclusion Surveys** really give an overall coverage of aesthetic and morphological traits, and oral self-image perception, and give a balanced interpretation of treatment-need. The **Tamworth Survey** indices may thus be quite valid in the 1990s despite being devised nearly 30 years ago.

(v) CAN MALOCCLUSIONS BE PREVENTED OR MINIMISED?

The short answer is yes, in many but not all instances. To answer this question properly, one should look at the problem from the point of view of levels of prevention (*Dunning, 1986*). These are:

- a) primary prevention - stopping a problem from developing at all;
- b) secondary prevention (interceptive treatment) of a developing problem;
- c) control (disability limitation) of resultant problems.

a) Primary Prevention

Within the scope of this thesis, primary prevention relates to the prevention of tooth decay, and thus, any sequelae of this process. It is assumed below, in the body of evidence to be presented, that tooth decay can lead to various malocclusion events principally related to arch length loss. Tooth decay is preventable by appropriate diet and oral hygiene procedures, and increasing tooth resistance e.g. by fissure sealing; and the pre-and post-eruptive use of fluorides.

This thesis is principally concerned with the role of fluoride in increasing tooth resistance to caries, and thus, the possibility of reduced malocclusion due to arch length losses caused by caries and its possible sequelae (such as extraction).

Several authors state that the skeletal input in malocclusions cannot be prevented by normal primary preventive dentistry means (*Salzmann and Ast, 1955; Walther, 1960*).

On a more theoretical note, and beyond the scope of this thesis, pure prevention could also be related to selective breeding. This might help prevent a large number of skeletal and dental deviations, with respect to sagittal, vertical and transverse problems. Taking this one step further, genetic engineering may also be able to prevent such problems in the future.

b) Secondary Prevention (Interceptive Treatment)

This is principally aimed at halting early, the progress of a pre-existing problem. This involves early diagnosis and prompt treatment.

The interceptive treatment of interest to this thesis is the prompt recognition of caries, and its early, satisfactory repair. This will halt the progress of caries, and prevent the possible sequelae of untreated caries. These collectively, may lead to arch length loss in many cases. Evidence to be presented below, shows arch length loss can lead to malocclusion.

c) Control

Control is disability limitation. Once an orthodontic problem has climaxed, conservative treatment principles are preferred and might be:

- i) the least radical;
- ii) least complex;
- iii) use the least number of irreversible procedures.

This thesis is not concerned with conservative treatment (or control).

From this point, this thesis will concentrate only on the maintenance, or minimised loss of tooth structure.

Tooth structure can be maintained, or minimally lost by employing

purely preventive and interceptive principles, namely:

- prevention of early loss of deciduous and permanent teeth.
- prevention of arch length loss due to the placement of faulty, inadequately contoured restorations.
- early diagnosis of caries and early placement of restorative agents such as glass ionomer cement, and preventive agents such as fissure sealants.
- good oral hygiene and diet.
- administration of systemic and topical fluorides and the benefits these entail.

These benefits of fluoride may extend not only to the increased resistance to caries but may also extend to the alteration of fissure depth and width which may predispose to less caries. (see PART I). Additionally there may be a reduction in permanent tooth size. The mechanism whereby this may prevent or reduce malocclusion will be discussed more in the next section *"The Role of Fluoride in Preventing Malocclusion"*.

The principles mentioned above are all related to dental caries prevention and its early detection and treatment. Early detection of susceptible fissures would hopefully result in early placement of fissure sealants with their attendant high success rate in caries prevention (*Silverstone, 1978; Elderton, 1987*). Early caries detection means early restoration so that drift by other teeth does not occur, which may have resulted in an effective arch length decrease. Presumably this early restoration will be well-placed and properly contoured so that no (further) drifting of teeth can occur.

In the beginning, the carious process itself may have been prevented if the primary and secondary dentition had received, and was still receiving the benefits of good oral hygiene and diet. Combine these with the effects of fluoride, incorporated pre-eruptively and post-eruptively into the deciduous and permanent enamel, this being continually reinforced via fluoride in topical forms such as dentrifice, rinse and/or professionally applied, or even the topical effect of fluoridated water.

If this has been ineffective and caries still occurs, and progresses heavily, all attempts should be made to save primary molars and canines via pulp therapy if indicated. In severe cases even retention of decoronated deciduous teeth is indicated in the absence of overt apico-alveolar pathology (*Powell, 1985; Godfrey, 1990*).

From this point discussion will only centre on:

a) Maintaining Proper Tooth Contacts

Several authors stress the need to consider the maintenance of proper tooth contacts in both primary, mixed and permanent dentitions (*Moyers, 1958; Graber, 1966; Salzmann, 1974; Northway and Wainwright, 1980*). This subject can be considered as comprising:

i) Reduction of Caries and its Prompt Repair

In the first instance the reduction of caries has obvious preventive implications through the use of fissure sealants (*Elderton, 1987*), but also in the context of this thesis, from the use of fluorides both pre-and post-eruptively in their various forms and vehicles. The effectiveness of fluoride in decay reduction in both deciduous and permanent

teeth was reviewed in PART I.

Murray (1987) feels the natural vertical attrition of deciduous teeth plays some role in natural arch length decrease, once attrition has progressed beyond the level of the original proximal contacts. Caries, of course, can act to reduce tooth dimensions and this is of special importance interproximally. Interproximal sites are the main areas of caries activity in the deciduous dentition. They are also the sites of greatest potential fluoride benefit.

Moyers (1958); Graber (1966) and Salzman (1974) all claim that unrestored caries in the deciduous and early permanent dentitions can lead to significant arch length decreases which can lead to crowding and even, potentially, mild distocclusion due to maxillary first permanent molar drift.

Moyers (1958) felt caries on the distal of second deciduous molars could result in almost immediate arch length loss if the first permanent molar was erupting. He cited *Jarvis*, who found in his thesis (*Uni. of Toronto*) that unrestored caries was the most common condition leading to arch length loss.

Taken to its extreme, of course, caries may lead to such tooth destruction and endodontic problems that extraction may be indicated. This may then lead to an amplified situation of arch length loss. The sequelae of early deciduous and permanent tooth loss will be covered later.

Moyers (1958) and *Graber (1966)* both conclude that the best prophylactic in orthodontics is a well-placed, fully contoured, and one could add, timely, restoration in a

primary molar.

ii) Placement of Correctly Contoured Restorations

This naturally follows on from the above views of *Moyers (1958)* and *Graber (1966)*.

In order to prevent arch length loss, or to halt arch length loss that has already occurred, it is obvious that a well-contoured restoration in the proximal area is necessary.

Graber (1966), in particular, states that great care should be taken with contact points. If the contour of the contact is too rounded he feels there is a possibility of a slipped contact, with attendant further arch length loss.

As well, he feels unnecessary pressure in the contact point of deciduous or permanent posterior teeth may predispose to teeth more anteriorly in the arch slipping contacts. Graber also notes the increased possibility of other anterior teeth slipping contacts where an anterior is restored with too great a contact pressure such as in an over-contoured gold inlay. This idea receives support from *Southard et al (1990)* after researching the "*anterior component of Biting Force*".

Naturally, the lack of a contact in a restoration is equally to be avoided in his opinion.

Northway and Wainwright (1980) found that space loss definitely does occur with loss of tooth substance due to caries. They found an average arch length loss of 0.5 to 1 mm in cases of severe caries in primary molars. However,

they state that these losses are far less than the potential arch length losses possible following the early extraction of a primary molar.

Powell (1985) mentions that where marginal ridge breakdown occurs in the primary or early mixed dentition; that the potential for space loss exists. In *Powell's thesis (p 41)* he found that space loss did occur in individual cases of marginal ridge breakdown. He felt other factors apart from the loss of tooth substance dictate the outcome since space loss doesn't occur in all cases. He found it varies with the site; the time and duration of the loss; the inclination of the first permanent molars; cusp heights; the interdigitation of the first permanent molars; and pre-existing crowding.

In his assessment of arch length loss due to his '*minimal treatment (interproximal caries slicing) program*' he found, in general, a clinically insignificant amount of arch length loss. However, some cases showed up to 1.7 mm arch length loss in the maxilla and 2.1 mm in the mandible. In many cases this may equate only to the limit of the "*leeway space*".

Thus the contentions of the earlier authors (*Moyers, Graber and Salzman*) are sustained. However, their clinical significance in most cases is questioned by Powell.

The present reviewer, however, questions Powell's use of the term "*clinical significance*"; since in some mouths even the loss of 1 mm of space can result in clinically significant

crowding and the slipping of tooth contacts. Additionally, the loss of space equal to the leeway space may prevent the achievement of normal molar relationships and thus contribute to crowding and/or a ½ unit molar deviation.

The present writer concludes that the maintenance of proper tooth contacts, or the early repair of damaged (cariou) contacts is easily achievable using conventional preventive dentistry means, such as dietary control, good oral hygiene, the use of fissure sealants, and regular examinations.

Most importantly, the use of systemic and topical fluorides, especially, is recommended from the bulk of opinion in PART I, as the major and easiest means of achieving caries protection.

b) Preventing the Effects of Early Loss of Deciduous and Permanent Teeth

i) Effects of Early Deciduous Tooth Loss

The effects that have been found by various authors are listed below:

1. Space closure (arch length loss) - this effect has aroused the most interest since it may affect eruption of permanent teeth, midlines, overbite and arch size.
2. Eruption of permanent successors may be delayed or hastened.
3. Impactions may be predisposed.
4. Crowding may be predisposed or increased.
5. Rotations and tipping of adjacent erupted and unerupted teeth may occur.
6. There may be a predisposition to altered dental relationships such as crossbites.
7. In some cases a decrease in maxillary size may occur.
8. Some functional and aesthetic impairment may result.

These effects have been found to vary with:

- degree of crowding already present and the time of premature loss;
- position of the premature loss, i.e. the jaw involved and tooth involved;
- time of extraction with respect to the dental age of the patient;

- number of teeth lost;
- eruption sequence of the teeth;
- skeletal, muscle and growth pattern of the patient;
- occlusion of the patient. This relates to dental relationships such as Angle's malocclusions and even to cuspal interlocking.

Some of the evidence to support these contentions is presented below.

Owen (1971) reviewed most literature on this topic up until that time. He found many contrary views but concluded from the general consensus of studies that space closure may result in a permanent arch length decrease. There may be an increase in overbite associated with early permanent tooth loss. Crowding is generally increased as is the likelihood of impactions of permanent successors. Additionally, arch asymmetries are possible especially with unilateral tooth loss. He found these effects varied with those factors mentioned in the section above.

Overall his findings for space closure are as below:

Incidence of Closure

This increases with time. 96% of deciduous extraction spaces present longer than 12 months show some closure. Maxillary second deciduous molar spaces show the higher and earliest incidence of closure; some lower primary

-

extraction spaces showed no closure.

Rate of Closure

Maxillary tooth spaces in general close quicker than mandibular spaces. Rates of closure in the maxilla slow after the first 6 - 12 months following deciduous tooth loss.

Amount of Closure

Maxillary second deciduous molar spaces show the greatest amount of closure. They are followed by mandibular second deciduous molar spaces.

Maxillary and mandibular first deciduous molar spaces show about equal amounts of closure. Generally, the longer the time available, the greater the closure. This is especially so when extractions occur before the first permanent molar erupts.

Direction of Closure

Maxillary spaces close from behind. Mandibular spaces generally show greater closure from the distal movement of anterior teeth with minimal closure from behind.

Various results since Owen's article are presented below.

Ronnerman and Thilander (1977) found early loss of the second molar resulted in greater space closure than early loss of the first primary molar. Additionally, they found 28% of their cases required active orthodontic treatment where there was early unilateral second primary molar loss in the maxilla. For early mandibular loss of the

second primary molar, the figure requiring active orthodontic treatment was 37%.

Helm and Siersbaek-Nielsen (1973) found that in general, early loss of deciduous teeth increases the risk of crowding. This is also supported by *Malerman (1977)*; *Dearing (1981)* and *Berg (1986)*. They found more maxillary than mandibular crowding following early primary tooth loss. In the maxillary lateral segments experiencing early primary molar loss, crowding was 4 times that of controls. However, in each jaw 3/4 of lateral segments with premature loss of primary teeth, had no crowding.

Overall, crowding was greatest after early loss of the maxillary primary canine, a finding not noticed in other studies by this author. Early lateral segment tooth loss generally allowed some resolution of anterior crowding. Loss of second primary molars generally produced more crowding than first primary molar loss.

Kerr (1980) investigated the effect of early primary tooth loss on eruption. He found the effects of early loss of primary canines and molars variable. In general, he found hastened eruption of successors when maxillary primary molars were lost early. The opposite effect was noted for the mandible. This is especially so when mandibular primary canines are lost early.

Overall, early primary tooth loss hastened the permanent successor's eruption except in the mandibular premolar region. Marked space loss and immaturity of dental

development may result in delayed eruption after very early primary tooth loss.

Miyamoto et al (1976) mention that specific factors, such as age at the time of early primary tooth loss, leeway space, and cusp height, all affect the degree of space closure. They found that increasing numbers of prematurely lost teeth increase the likelihood of later orthodontic treatments. However, they also noted that the malocclusion problem decreased with increasing age of deciduous tooth loss in their study.

In contrast to *Helm and Siersbaek-Nielsen (1973)*, they found early primary canine loss had little impact on major malalignment, unlike first and second primary molar loss. They found little difference in effect between the early loss of first and second primary molars. The loss of primary molars prior to 6 years of age resulted in a 74% chance of major malalignment of the permanent dentition.

Walther (1966) stressed the amount of space deficiency already present at the time of early loss of deciduous teeth. Also, the skeletal pattern was important in determining the effect of early primary tooth loss.

He noted the potential for a worsening of dental Class II and III malocclusions due to lingual collapse of lower and upper anterior segments respectively, if extractions were in this region including the extraction of primary canines and to a lesser extent primary first molars. He

also noted that the lingual collapse of lower anteriors may also result in a secondary collapse of upper anterior alignment. This lingual collapse of lower anterior segments was often related to the inclination of the primary canine. If these teeth had distally inclined crowns prior to extraction of nearby primary first molars, the anterior segment was likely to distally incline.

This early canine loss may also be associated with anterior movement of buccal teeth, midline shifts (if unilateral) and often lower anterior collapse if a lower canine is lost.

Magnusson (1979), from studies of Icelandic children, found that such space lost from early primary tooth loss was regained during the later part of the mixed dentition. By dental Stage 4 (canines and premolars fully erupted) only 18% of children had more than 2.5 mm space lost on the side of the premature primary tooth loss. In Dental Stage 2 (permanent incisors fully erupted) there were 75% of children with greater than 2.5 of space lost.

Where deciduous tooth loss was very early 30% showed more than 2.5 mm space lost. This agrees with Owen's (1971) finding on length of time spaces are present. It was felt the regained space was due to absorption of the leeway space.

Ronnerman (1977) reported age-dependent effects of early

primary tooth loss. Primary molar losses prior to 7 1/2 years yielded more resultant crowding than primary molar loss after 7 1/2 years. In this instance there was little effect on relative space.

Additionally, he found early primary loss promoted early eruption of successors. In the mandible, if the first primary molar was lost before 7 1/2 years, the eruption of the first premolar was retarded. After 7 1/2 years of age, the eruption of the premolar was hastened. This effect held for second premolars too. This is supported by Kerr (1980) above.

After early second primary molar loss, the second permanent molars generally erupted earlier. He also cites McLaughlin et al (1967) as showing 20% impaction of the second premolars after early primary second molar loss.

Ronnerman and Thilander (1978) studied craniofacial morphology associated with early primary molar loss. There was little difference between controls and study subjects except in the length of the maxilla from posterior nasal spine (PNS) to A point (subspinale). This was significantly shorter in the early primary tooth loss group. They stated that Siepel's study (1946) supported this finding. Siepel found diminished mesial growth of the dental arch in children with early loss of permanent teeth.

Pedersen et al (1978) found that early deciduous tooth loss resulted in increased sagittal, vertical and

transverse malocclusion. An increase in distocclusions occurred if early primary molar loss occurred in the maxilla only. Distocclusions were often bilateral if both sides had had early primary molar extraction. 12.8% of cases in this category of study showed either a unilateral or bilateral distocclusion.

The frequency of maxillary overjet did not seem to be influenced by early deciduous tooth loss. Overbite was often increased, but only if the extractions were in both jaws was this significant.

Crossbites were more prevalent in early primary tooth loss cases. This was especially so if both jaws or the maxilla alone were involved.

Early mandibular primary tooth loss resulted in decreased distocclusions but not increased mesiocclusion. Presumably this is due to less forward drift in the mandibular arch as pointed out by *Owen (1971)*. Thus, enough drift may occur to correct a distocclusion but insufficient to go as far as causing a mesiocclusion.

Early deciduous tooth loss also resulted in an increased incidence of midline deviations.

Overall there were increased orthodontic treatment needs if early deciduous tooth loss had occurred, especially in the maxilla.

Dearing (1981) listed sequelae of early primary molar loss as tipping, rotation, successor impaction, midline

drift, over-eruption of teeth in the opposing arch, and consequent impaired function.

Of the patients in his study, 8.8% were subsequently undergoing full orthodontic treatment where the cause was due to early primary molar loss. He finishes by citing *King (1961)*, King commented

"spaces in the arch do close in a sufficient proportion of cases to make this factor the most important preventable cause of malocclusion".

Barnard (1956), in his survey of state-school children in *New South Wales*, found 80% had a malocclusion according to the criteria he used. He felt early tooth loss was responsible for

"a great amount of the malocclusion observed".

ii) Effects of Permanent Tooth Loss in the Mixed Dentition

There is much less written on this subject, in the sense of permanent tooth loss due to caries, which is within the scope of this thesis.

First permanent molars are the teeth at greatest risk. Principally, this risk is related to occlusal caries but also to a lesser extent, smooth surface caries (*Bossert, 1933; Sutcliffe, 1974*).

Several authors (*Sutcliffe, 1974; Ripa et al, 1988*) have noted the ongoing nature of carious attack and destruction, both cross-sectionally and longitudinally in the population with respect to first permanent molars.

Sutcliffe (1974) found only 48% of a random sample of over 9000 Scottish 13 year-olds had all first permanent molars present. There was an increasing decay trend for first molars which was paralleled by an increasing extraction trend.

McEwen and McHugh (1970) found a small overbite increase after the loss of all first permanent molars. Also attendant with the loss of all first molars, was a significant decrease in overjet in Angle's Class I malocclusion cases. This was not found for those occlusions already Class II Division 1. They mention *McEwen and Burke (1969)* as suggesting that these overjet decreases are only in patients where the overjet is due to anterior maxillary crowding. This is relieved and thus, so is the overjet. *Godfrey (1990)* feels that maybe this overbite increase with overjet reduction can be related to a closing rotation of the mandible in some cases.

Thilander and Skagius (1970) state that late extraction of first permanent molars in the maxilla (12 years-old +) gave little closure of space. However, a high frequency of rotation of the maxillary second permanent molars occurred. They found maxillary first permanent molar extraction around 8 - 10 years-old yields a good prognosis orthodontically.

On the other hand, even for early loss of the mandibular first permanent molar, there is still unfavourable space,

rotations and tipping. In only 36% of cases did early mandibular first permanent molar loss yield acceptable results.

Evans (1988) found increased levels of lower second permanent molar impaction could be attributed to less early lower first permanent molar loss; however, early primary molar loss did not necessarily improve the angulation of the lower seven.

In cases where impacted lower second permanent molars were detected early, early extraction of mandibular primary molars didn't improve the impaction angle.

Walther (1966, p 146) thought early loss of the upper first permanent molar would result in good contact of the second molar with the second premolar. It would also reduce mild anterior malalignment. Late loss of the upper first permanent molars often yields unclosed space, mesiopalatal rotations of the second molar and some mesial tipping of second molars.

In regard to early loss of lower first permanent molars, usually the second molar will drift forward into contact with the second premolar. Occasionally, there may be little change in the second molar position; instead the premolars are affected. This may yield a second premolar in contact with the second molar, with a space the size of the first molar between the first and second premolar.

Godfrey (1990) regards this as a frequent occurrence depending upon the axial orientation of the developing

second premolar during its eruption. Sometimes a second premolar may impact under the second molar in the mandible. Alternatively, there may be generalised spacing or a large space between the lower second premolar and second molar.

With late lower first permanent molar loss considered, the erupted, or nearly erupted, second molar's space closure is mainly by tipping and rotation with various degrees of premolar distal movement or tip.

Can these effects just mentioned be prevented? Yes, if due to caries, most easily and cost-effectively by fluoridation of water supplies (*Garcia, 1989; Newbrun, 1989*), but also by the application of good preventive dentistry methods as discussed earlier (*Elderton, 1987*).

Discussion of the specific role that fluoride has to play in helping to prevent these detrimental effects to the occlusion is presented in the next section.

vi) THE ROLE OF FLUORIDE IN PREVENTING MALOCCLUSION

Fluoride in systemic and/or topical forms has the potential to reduce the incidence of caries. By reducing caries the potential for arch length loss from untreated caries is reduced. Likewise, the chances of the placement of unacceptable restorations are reduced.

Following on from this, it is obvious that untreated caries can ultimately lead to the necessity for deciduous and/or permanent tooth extraction. Outlined above were the common sequelae to early loss of deciduous and permanent teeth. These sequelae can be prevented or largely reduced by the use of fluoride in systemic and/or topical forms. The literature supporting the above-contentions was reviewed in PART I, on the effectiveness of fluoride in preventing caries in deciduous and permanent teeth.

Another possible role is that fluoride may act to prevent or reduce caries experience, by its alteration of tooth morphology. Most studies reviewed early in PART I showed that cusp heights and angles were reduced by fluoride at, or above 1 ppm. Additionally, the occlusal fissures were generally found to be wider and shallower. This suggests improved anatomy because teeth would be less prone to caries. Thus the spinoffs to reduced malocclusion, by virtue of maintained arch length, are obvious.

However, another controversial area was discussed where fluoride may alter tooth morphology such that mesiodistal tooth widths may be reduced by the consumption of fluoride at 1 ppm or more. The present writer and *Wallenius (1959)*, both felt that reductions in mesiodistal tooth widths, if they occurred, would favour a reduction in crowding, since tooth size relative to basal bone size would be reduced. The

reviewed literature on this topic seems only to generally support a reduction in cusp height, and cuspal angles along with increased fissure width and decreased fissure depth.

There is varying thought on whether cusp height changes have positive or negative correlations with overbite changes (*Cordato, 1990*).

It is thus possible that fluoride may have an effect on overbite values and possibly, indirectly on overjet values. This is by virtue of its effect on tooth anatomy favouring decreased caries, but also reduced cuspal angles and heights.

Table (II-?) shows a summary of the findings of the available articles on this topic.

Tooth size in the mesiodistal dimension increased in two studies in humans; showed no change in one study with humans; and decreased in four studies in humans. Despite a narrow majority of human studies showing this decrease in mesiodistal dimension, the present reviewer feels that no strong relationship can be said to exist between optimal fluoride consumption and mesiodistal tooth dimensions.

Three human studies found reductions in buccolingual tooth widths, these findings occurred in three of the four cases finding mesiodistal reductions as well. Again, clear lack of a significant relationship between the optimal fluoride consumption and buccolingual tooth width is concluded here too.

More study is needed in this area.

The means by which fluoride is thought to exert its greatest effect on preventing, or minimising malocclusion, is by its topical and systemic

effects on deciduous, and permanent tooth enamel. Studies showing support and lack of support for this contention are presented below.

Author	Sample	Change in Cusps	Change in Fissures	Change in Size	Change in M/D width	Change in B/L width
Malherbe & Ockerse (1944)	humans	lower, flatter	shallow	-	-	-
Owen Dental Res. Toronto (1954)	rats	-	shallower	overall smaller molars at 10ppm	-	-
Wallenius (1959)	humans	-	-	larger	larger	-
Paynter & Grainger (1961)	rats	wider	smaller	-	-	-
Cooper & Ludwig (1965) Cooper (1964)	humans	lower	wider	smaller	smaller	smaller
Kruger (1964)	rats	flatter	wider, shallower	-	-	-
Kruger (1966)	rats	-	wider	-	no change	-
Moller (1967)	humans	lower, rounder	shallower	larger	-	-
Simpson & Castaldi (1969)	humans	-	wider	larger	larger	-
Lovius and Goose (1969)	humans	-	-	1st molars smaller	smaller 1st molar	smaller 1st molar
Mercado and Bibby (1973)	rats	smaller	shallower	smaller	-	-
Aasenden & Peebles (1974)	humans	-	atypically shallow	-	-	-
Grahnen et al (1974)	humans	-	-	no change	no change	-
Brock (1974)	humans	-	-	no change	-	-
Goose & Roberts (1979)	humans	-	-	smaller	smaller	smaller
Omar (1987)	humans	-	-	-	smaller centrals with higher Fd levels; premolars smaller in higher Fd levels	-

(vii) THE PREVALENCE OF MALOCCLUSION IN OPTIMALLY FLUORIDATED AND FLUORIDE-DEFICIENT COMMUNITIES

The prevalence of malocclusion *per se*, has been assessed at various times and in various parts of the world by various authors; *Peel (1973)*; *Jago (1974)*; *Foster (1979)*; *McLain and Proffit (1985)*. All present the assembled data from a number of studies on this topic.

However, the results of most of these studies correlate poorly with each other probably due to:

- racial differences in occlusion types
- local gene-pool variations
- possible local environmental differences such as diet (including fluoride ingestion); local dental programs' existence and emphases
- different indices and criteria used
- interexaminer differences in using indices, but also, examiner opinions of the severity of different malocclusion types may distort results, especially where there is a subjective assessment method used.
- ages of subjects examined. Developing dentitions may show increasing or decreasing malocclusion with age of assessment (*McLain and Proffit, 1985*).

Since this thesis is principally concerned with the effects of fluoride on the prevalence of malocclusion, it was deemed reasonable to deal only with articles that were directly related to the topic, and which used controlled studies, with the same examiners for

assessing control and experimental groups. Therefore, such articles should demonstrate minimal interexaminer variability.

Even if criteria were different between studies, at least for each study, valid comparisons could be made intrastudy to arrive at a statistically significant or insignificant result, with respect to the input of fluoride in reducing malocclusion.

If the results of each of these studies were arrived at validly, then the results should allow valid comparisons between studies, even if different criteria of assessment (indices) were used.

Peel (1973), as part of his thesis, reviewed most of such studies conducted up until 1973. He divided the findings into 2 groups:

- a) those exhibiting *significant reductions* in malocclusion where fluoride was consumed, compared with control groups;
- b) those exhibiting *non-significant reductions* in malocclusions where fluoride was consumed compared with controls.

Studies where fluoride was associated with reduced malocclusions

Plater (1949) found that 12-14 year-olds living in fluoridated (1.2 ppm) *Union Grove*, had a higher percentage of correct mesiodistal arch relationships compared with non-fluoridated *Madison*. Plater concluded that the retention of deciduous teeth and permanent first molars was probably the reason for the 30% reduction in malocclusion incidence in *Union Grove*.

Okayay (1957) found that for 2 towns in *Asia Minor*, *Isparta* (high fluoride level in the water) and *Antalya* (low water fluoride level), a

lower percentage of acquired malocclusions were present for children from *Isparta*. Okayay attributed this difference to the higher level of caries and tooth loss in *Antalya*.

Walther (1960) examined the occlusions of 1000 *English* children from *Colchester* (1.2 - 2.0 ppmF). *Chelmsford* (0-1 ppmF) and *Norwich* (0.2 ppmF). *Colchester* showed significantly higher numbers with Class I dental relationships compared to the other groups. The number of Class II Div. 2 cases were also markedly less in the *Colchester* (1.2 - 2 ppmF) group. Normal overbite was likewise more common in *Colchester*, and increased, complete overbite was least common. Walther implicated early deciduous and first permanent molar tooth loss in buccal crowding, overbite and axial deviations of incisors. He felt ethnic groups and fluoride-intake differences may account for the differences in occlusions between the groups studied.

Ast, Allaway and Draker (1962) assessed occlusion differences between 13 to 14 year-olds from *Newburgh* (fluoridated-optimally) and *Kingston* (non-fluoridated). Results showed a higher number of normal Class I molar relationships, with no treatment required, in *Newburgh*. The authors attributed the higher incidence of malocclusion in *Kingston*, to the higher loss of first permanent molars; the higher rate of untreated caries; and the higher rate of other missing teeth.

The *New York State Department of Health* criteria classified 9.4% of *Newburgh*, and 22.5% of *Kingston* children, as having physically handicapping malocclusions.

Tank and Storvick (1965) studied 1-6 year-olds from *Cornwallis* (optimally-fluoridated), and *Albany* (non-fluoridated). *Albany* showed an increased prevalence of malocclusion (18%) compared with *Cornwallis*

(9%). There was a suggestion of a positive relationship between caries and malocclusion. However, a higher loss of deciduous second molars was not associated with malocclusion in the *Albany* children. The present author feels this is understandable since the age-range studied would limit the amount of malocclusion able to develop in these early ages.

Erickson and Graziano (1966) found acceptable-occlusion levels of 43.5% in fluoridated *High Point*, compared with 28.2% having acceptable occlusions in *Greensboro* (low fluoride level). These differences were statistically significant. Index criteria and examination techniques used, and community differences made assertion of a constant relationship between fluoride and malocclusion difficult.

Murray (1969) examined 766, 15 year-old children from *West Hartlepool* (1.5-2 ppmF) and *York, U.K.* (0.2 ppmF). No significant differences were found for the distribution of Class I, Class II and Class III molar relationships. However, 17.9% of *West Hartlepool* children had normal occlusion compared with 8.7% of *York* children. Also of statistical significance, less *West Hartlepool* children required treatment. *York* children had a higher prevalence of maxillary crowding, supposedly due to arch length loss, as a result of early deciduous tooth loss, or caries (alone). Despite this, Murray concluded that fluoride consumption had no effect on anteroposterior dental relationships.

A further study which fits this group is that of *Salzmann and Ast (1955)*. These authors studied 386 children 6-10 years of age in *Newburgh* (optimally-fluoridated), and 373 children of the same ages in fluoride-deficient *Kingston*. Lateral cephalometric headfilms were also used.

The authors concluded that fluoridation *per se*, had no effect on the skeletal facial pattern. *Kingston* children showed higher percentages of malocclusion than *Newburgh* children (all of whose first permanent molars were present). Additionally, the number of children in *Newburgh* with normal occlusion was nearly twice as high, as the number of children in *Kingston* with normal occlusion.

Studies where fluoride was not associated with a significant malocclusion reduction

Pelton and Elsasser (1953) investigated the fluorine/malocclusion relationship in two *Idaho* towns: *Nampa* (1.5 ppmF in the water supply) and *Coeur d'Alene* (negligible fluoride in the water supply). They examined 850 children using the DMFT index and the dentofacial index (DFI). *Coeur d'Alene* children had 4.5 times the DMFT scores of *Nampa* but the DFI scores were only slightly lower for *Nampa*. They thus concluded that caries was not a highly significant factor in the aetiology of malocclusion. *Peel (1973)* felt that the DFI, basically measuring skeletal or anthropological relationships, may not have been the most appropriate to use when most deviations are mainly dental.

Hill, Blayney and Wolf (1959) concluded in their study that, at that time, no definitive statement could be made regarding fluoridation and its effect on the prevalence of malocclusion.

However, they did report 20% less malocclusion among 6 to 8 year-olds, and 17% less malocclusion among 12-14 year-olds who had consumed fluoridated water compared with prefluoridation controls. In 14-year-old children who had consumed fluoridated water all their lives, they found a much higher rate of normal occlusion, and a considerably lower rate of malocclusion, than in children who had consumed non-fluoridated water. Their assessments were based on the Angle's Classification.

Of 141 children 6 - 8 years-old with premature loss of the second deciduous molar, approximately 54% had normal occlusion. Of 84 children at 6 - 7 years of age with loss of the E prior to 6, 7 to 8 years, 59.5% showed normal occlusion. Six years later at 12, 13 or 14 years of age, these same children showed 47% with normal occlusion. None had had orthodontic intervention. Thus *50% or less of children with early E loss develop malocclusion*. No mention is made of the effects of early loss of other deciduous teeth or first permanent molars.

Szwedja (1965) measured the occlusions of 10 - 11 year-old children in *Charlotte* (optimally-fluoridated) and *Mecklenburg County* (unfluoridated). He used the Handicapping Labio-Lingual Deviation Index of *Draker (HLD)*. This index recorded overjet, overbite, openbite, mandibular protrusion, and labiolingual displacements. As assessed by the HLD, there was no statistically significant difference between the 2 groups of children.

Lovius and Goose (1969) investigated malocclusions in two different *Anglesey* towns, *Gwalchmai* (optimally-fluoridated) and *Bodafon* (unfluoridated). Their results suggested that fluoride has no

measurable effect on overjet and overbite.

They did find an increase in the maxillary arch width in the fluoridated subjects. possibly this could be related to early deciduous tooth loss and resultant forward migration of permanent teeth in children from the unfluoridated town of *Bodafon*. Early loss of deciduous teeth was much more common in *Bodafon*.

A study which falls into this same grouping is that of *Davies et al (1964)*. They recorded molar relationship, overjet, overbite, Angle's Class, crossbite, openbite and premature loss of deciduous molars and cuspids. Recordings were of 955 white children 7-9 and 11 - 13 years of age living in *Bloomington* (0.1 ppmF), *Indianapolis* (0.5 - 0.8 ppmF) and *Frankfort* (0.7 - 1.1 ppmF). Chi square tests revealed that the differences in recorded measurements of occlusion in the 3 cities' children could not be related to the fluoride content of the water supply.

THE TAMWORTH MALOCCLUSION SURVEYS

Paralleling the Tamworth Fluoridation Surveys of 1963, 1973 and 1988 (see PART I) were the Tamworth Malocclusion Surveys.

Schoolchildren, who had just completed their dental health checks as part of the Fluoridation Surveys were chosen at random for the Malocclusion Surveys.

The assessment of each subject's occlusion was based on three different methods:

- i) the Angle's Class of the occlusion
- ii) The Subjective Assessment of Treatment-Need of the occlusion
- iii) Their occlusions were assessed also according to the Godfrey Index (GI). This is an objective assessment of various occlusal traits namely: lower anterior crowding; left and right side posterior crowding in the mandible; overbite; overjet; buccal segment anteroposterior relationships of the right and left sides; posterior crossbites of the left and right sides. The scores for these traits were totalled to give an overall GI score.

In 1963 the Malocclusion Survey was conducted by *Godfrey*. The results of this survey form the baseline (controls) for comparison with the later Malocclusion Surveys of 1973 (*Peel*) and 1988 (*Mylchreest*). Additionally, in 1989 it was decided to examine schoolchildren in their early permanent dentitions from *Lismore*. *Lismore* is a non-water-fluoridated town in the same State (New South Wales) as *Tamworth*. It was felt that the results from *Lismore* would act as contemporary

controls. This is especially important, since the aforementioned secular decline in caries (see PART I) became obvious in the late 1970's to early 1980's, several years after *Peel's (1973) Survey of Tamworth schoolchildrens' occlusions*.

This caries decline was chiefly attributed to increasing compliance of children with oral hygiene and the widespread use of fluoride toothpastes. The decline occurred in optimally-fluoridated as well as unfluoridated communities. The mechanisms at work were most likely both topical and systemic. The systemic effect is due to the swallowing of fluoride toothpaste, which is common in children up to 5 years of age according to *Ophaug and Singer (1988)*. Thus, it can be concluded that *Tamworth* children since the late 1970s have probably been consuming above-optimal fluoride levels during at least some of their preschool years. Those children in *Lismore* during the same time period had probably consumed sub-optimal to near-optimal levels of fluoride during part of their preschool years due to fluoride toothpaste ingestion.

It can thus be concluded also, that while contemporary results of *Lismore and Tamworth Malocclusion Surveys* both have input due to the effects of fluoride, those results for *Lismore* are primarily due to fluoride from toothpaste sources, while the results from *Tamworth* are due to both water-fluoridation and fluoride toothpaste effects combined.

PEEL'S (1973) RESULTS FROM THE TAMWORTH MALOCCLUSION SURVEY

Peel (1973) in the discussion of his thesis results, noted a statistically significant variation in the occlusions of *Tamworth* children from 1963 to 1973. He felt this could be due to:

- a) changes in the demographic patterns of *Tamworth* children between 1963 and 1973, but concluded little change had taken place.
- b) intra-and inter-examiner variability. His statistical tests of variability proved this to be minimal, with respect to Angle's Classification and the Godfrey Index. However, he concedes that variability may have existed between Godfrey and himself regarding the Subjective Assessment of Treatment-Need. This component was not tested for variability inter-and intra-examiner. Thus *Peel* felt the validity of this measurement between studies could be questioned.
- c) The effects of local dental programs and their emphases were difficult to quantify. However, he felt that the better quality of teeth since fluoridation with respect to caries, was more likely to lead to them being able to be restored more successfully by dental personnel. Additionally, parents, patients and dental personnel were more liable to want restoration rather than extraction. The general emphasis of the school-based dental programs became more preventive and conservative in nature, due to the diminished caries problem. *Peel* concluded that any effects here may only complement fluoride's effects on

the occlusion.

- d) Fluoridation. He concludes that the majority of the changes in occlusion of children from 1963 to 1973 is attributable to the effects of fluoride consumption.

ANGLE'S CLASSIFICATION (Peel, 1973)

For the Angle's Classification, there was an increase between 1963 and 1973 in the number of children with normal occlusion. This seems to be associated with a decrease in those with Class I malocclusions. Class II malocclusions remained stable in numbers. However, Class III malocclusions increased from 1963 to 1973. Peel felt this may be examiner-related in nature.

SUBJECTIVE ASSESSMENT OF TREATMENT AND NEED (Peel, 1973)

For the measurement of Subjective Assessment of Treatment-Need, severe malocclusions (++) showed negligible change in distribution between 1963 and 1973. Peel suggested little input from fluoridation in such situations as skeletal deep or openbite, severe anteroposterior skeletal problems. Severe tooth size/basal bone size discrepancies would be little changed due to fluoride.

General-practitioner-level-of treatment-malocclusions (+) showed a significant reduction between 1963 and 1973. This accounted for the increased numbers assessed as having normal occlusions. Peel inferred the role of fluoride here in preventing early deciduous and permanent tooth loss.

Also, overall, there was a substantial reduction in the numbers of children who required orthodontic treatment at any level. This reduction was of the order of 25%.

GODFREY INDEX (Peel 1973)

With respect to the Godfrey Index Scores, the following were found:

Mandibular Incisor Crowding: This component showed a statistically significant increase in the 10-11 years age group. Peel felt this increase may reflect the "*benefit*" of fluoride consumption. This related to the fact, that in early loss of deciduous molars and canines, the permanent anteriors may align at the expense of space of permanent canines and second premolars (*Walther, 1966*). Without early loss, (due to the effects of fluoride) the aligning of the lower anteriors could not occur. On the other hand, if the results of *Wallenius (1959)* are given credibility, then this increase in lower anterior crowding could be explained by larger teeth due to fluoride's effect. As well, this effect would be expected in all age groups.

Mandibular Posterior Crowding. There appeared to be a decrease in posterior crowding when children examined in 1973 are compared with 1963 results. This tendency decreased with increasing age. The difference was not significant in those above 10 years of age. This may be due to the fact that these older children would not have been exposed to water fluoridation all their lives, and thus received minimal benefit to deciduous tooth enamel from fluoride. This may also reflect the use of the leeway space to allow unravelling.

Overbite. All groups showed a reduction in overbite in 1973 compared with 1963 controls. This was significant only in the older children.

Peel felt this could be related to variability in the

recording technique at inter or intra-examiner levels. The other effect at play could also have been normal developmental changes with growth. He cites *Björk (1953)* and *Fleming (1961)* as demonstrating a bite-deepening from 9-12 years and a bite opening from 12 - 17 years. Additionally, cusp heights of posterior teeth, and interincisal angles were postulated to play a part here. Peel cited *Prakash and Margolis (1952)* and *Fleming (1961)* as finding a negative correlation between overbite depth and molar height. This author takes it to mean that as molar height decreases overbite increases. However, *Cordato (1990)* cites *Campbell (1925)*; *Neustadt (1935)*; *Mershon (1937)*; *Popovich (1955)*; *Bolton (1958)*; *Sassouni and Nanda (1964)*; *McHorris (1969)*, *Dawes (1986)*; *Slavicek (1988)* and *Cordato (1990)b* as finding positive correlations between cusp height/molar height and overbite depth.

Peel felt that this related more to a "*posterior collapse*" type situation, where vertical dimension of the face may be reduced, and, thus, overbite increased. In fluoridated areas, it is surmised that less of this happens due to the protective effect of fluoride reducing caries, which may lead to the extraction of deciduous and permanent molars.

Peel also felt that in the 1973 sample, the interincisal angle of permanent anteriors would more likely be normal. It has been shown that early loss of deciduous canines can allow the anteriors to retrocline thus increasing the interincisal angle and predisposing to overbite deepening (*Walther, 1966*; *Owen, 1971*). This latter situation would be more the expected situation in the 1963 sample.

Openbite: From 1963 to 1973 an increase in the incidence of openbite was recorded. This was of statistical significance only in the total female group and the 6 - 7 years of age group. Peel cites this as agreeing with the results of *Mills (1966)*.

Overjet: Overjet was substantially less in the 1973 subject. Peel warns that this should be viewed with caution since the 1973 readings were made using a graduated measuring scale. In 1963 this measurement was only done by visual estimation.

Thus, the differences could be due to examiner variability. Peel notes *Szwedja (1963)* and *Lovius and Goose (1969)* as finding no difference between fluoride and non-fluoride groups with respect to overjet.

Reverse overjet: This reading yielded no apparent differences between 1963 and 1973 recordings. This is despite the increased recording of Class III malocclusion in the Angle's Classification. However, this Class III component may be reflected more in the recording of edge-edge in the openbite component.

Buccal Anteroposterior Relationships: There was a trend exhibited here, in the 1973 results, toward a reduction in the scores toward the neutroccclusion position. This reduction occurred from both mesial and distal directions. Peel explained this improvement towards ideal as reflecting the effect of fluoride in reducing decay, and thus, ultimately, early extraction of deciduous and permanent teeth, especially

molars. *Walther (1966); Owen (1971) and Powell (1985)* note the potential for increased mesiocclusion, and especially distocclusion after early E loss (principally).

Statistically significant reductions occurred only in the 14, 15, and 16 years, and total age groups.

A high incidence of cusp-to-cusp relationships were scored. This was in mixed dentition age groups especially, and thus reflects the common flush terminal plane occurrence at this stage. Canine relationships may have better reflected normality/abnormality in these age groups (*Peel, 1973*).

Posterior Crossbites: 1973 subjects showed an overall reduction in both the occurrence and severity of posterior crossbites. This reduction was statistically significant in 6 - 7 year-old and 14,15 and 16 year-old males, and the total age groups. Peel, and this author, are both uncertain as to how to best interpret this finding, with respect to the input of fluoride. Although, *Pedersen et al (1978)* found increased posterior crossbite occurrence following early deciduous molar loss.

Total Godfrey Index Score: A reduction has been noted in all age groups for both males and females in the 1973 study. This reached significance only in the total age group and the 14, 15 and 16 years age group.

Peel pointed out that the overall beneficial effects of fluoride, on the occlusion are not fully expressed until the permanent dentition. Similar observations were made for overbite, overjet and buccal anteroposterior relationships.

The overall occlusions of the *Tamworth* children surveyed in 1973, demonstrated improvement when compared to the 1963 prefluoride group, as assessed by the Godfrey Index. These findings agreed also with those of the 1973 *Angle's Classification* and Subjective Treatment-Need Assessment.

Overall the findings of *Peel (1973)* support the findings of *Plater (1949)*; *Salzmann and Ast (1955)*; *Walther (1960)*; *Ast, Allaway and Draker (1962)* and *Erickson and Graziano (1966)*, that a history of lifetime fluoride consumption can lead to a reduced prevalence of malocclusion.

The next section of this thesis will outline the design and conduct of the 1988 *Tamworth*; and 1989 *Lismore Malocclusion Surveys* which form the Method of this thesis.

(vii) SUMMARY and CONCLUSIONS of PART II

Presented in this section of this thesis were the problems of defining a malocclusion. Where does normality end and a malocclusion state begin? No firm conclusion was reached.

Perhaps we as orthodontists have in the past, and still to some extent, dwell purely on the morphological deviations from ideal? The necessity for assessing the individual's perception of their own occlusion was raised. This should also be coupled with Society's perception of the individual's occlusion. Adding these two elements to the objective recording of individual morphological traits may not only better help us define the malocclusion state, but also help to better establish treatment-need priorities. This applies in both the public and private treatment spheres.

Recent approaches to indexing and assessing malocclusion and treatment-need were presented. A critical analysis was presented of the three methods used to assess malocclusions in the past and present *Tamworth Malocclusion Surveys (1963, 1973, 1988)* and the *Lismore Malocclusion Survey (1989)*.

The possibilities of preventing malocclusion were discussed.

The effects on the occlusion of not being able to curtail dental caries and its sequelae were presented. These effects are largely preventable by controlling caries, especially through good fluoride practices. It is conceded, that not all subjects suffering gross caries and/or early tooth loss will develop malocclusions usually related to such events. However,

a significant number will (up to 40 - 50% according to Hill, Blayney and Wolf, 1959).

Previous studies presented, confirm that certain aspects of malocclusion, were reduced due to the ingestion of fluoride at optimal levels. Generally, the number of individuals with normal occlusions are significantly higher in fluoridated areas.

PART III

METHOD

An effort was made to keep the survey methods nearly identical to that of Godfrey (1963) and Peel (1973).

Selection of Subjects:

Two surveys were carried out by this author. In November 1988, a total of 208 male and female schoolchildren 12 and 15 year-olds from Tamworth were examined in conjunction with the 1988 Tamworth Fluoridation Survey. These children were lifelong residents of Tamworth and had consumed fluoridated water since birth.

In October 1988, 160 male and female schoolchildren 12, 13, 14 and 15 year-olds from Lismore were examined. These children were all students at Kadina High School. They were lifelong residents of the Lismore area. This latter group were surveyed to act as contemporary controls against the Tamworth Group. Lismore is a non-fluoridated town. Tamworth, as already noted, has been fluoridated optimally since 1963.

No attempt was made to select subjects other than randomly. Subjects underwent their occlusion assessment following the main dental caries exam. No attention was paid to order, sex or age group examined at any one time.

In the case of Tamworth, subjects were drawn from Peel High School, Oxley High School, Tamworth High School, East Tamworth Primary School, Hillview Primary School, Calrossy and Rosary Colleges.

Examination Procedure

In the case of *Tamworth*, subjects were chosen at random following their dental examination. Subjects who had had previous orthodontic interventions such as planned extractions, removable or fixed orthodontic appliances were excluded from the survey. It should be noted that this introduces a bias in favour of lowered malocclusion prevalence - but of course it would not be possible to quantitate their malocclusions, once corrected.

Examinations were carried out with the subjects seated in normal school chairs with the examiner working from the 7 - 8 o'clock position. Illumination was provided by an assistant using 2 Hoyt flexible oral illumination lights. These were mainly used for the assessment of the buccal relationships. Natural daylight was adequate for all other measurements.

In the case of *Lismore*, subjects were similarly excluded with respect to prior orthodontic interventions.

Examinations were carried out in the case of *Lismore*, in a dental caravan, using a reclined dental chair with a Faro operating light. The examiner was seated and examined from the 11 - 12 o'clock position.

In both situations, number 5 size mouth mirrors were used for aiding check retraction and were especially useful for visualising buccal relationships.

Overjet measurements were made using toothpicks. These toothpicks were marked with graduations at 1.5 mm and 3mm. A new toothpick was used for each subject and then discarded.

All recording was performed with the aid of a dental therapist writing down the scores after being called by the examiner (R.M.). A specially prepared score chart identical to that of *Godfrey (1963)* and *Peel (1973)* was used (*Fig.III-1 overleaf*).

FIG. III-1

LAYOUT OF SCORING SHEET USED IN
TAMWORTH MALOCCLUSION SURVEYS

<i>Serial No.</i>	<i>Age</i>	<i>Sex</i>	<i>Angle</i>	<i>As</i>	<i>Ac</i>	<i>PCr</i>	<i>PCl</i>	<i>+V</i>	<i>-V</i>	<i>+H</i>	<i>-H</i>	<i>BAP</i> <i>R</i>	<i>BAP</i> <i>L</i>	<i>Cr</i> <i>R</i>	<i>Cr</i> <i>L</i>	<i>TOTAL</i> <i>GI</i>

Additionally, in the case of *Lismore*, the examiner also recorded DMFT scores for each patient seen. Results of this examination were recorded separately.

Prior to the *1988 Tamworth Malocclusion Survey*, the present author worked through a dozen sets of stone models with Godfrey to assess the degree of observer reliability for the Angle's Classification and Godfrey Index. After 2 recordings for each set of models, results were compared and any areas of disagreement were discussed. This enabled areas of conflict in scoring to be resolved. It was thus concluded that the present author's scoring would be reliable for these assessments.

As with *Peel's (1973) Survey*, no calibration was carried out for the Subjective Treatment-Needs Assessment. Due to the lack of criteria of an objective nature to aid the examiner in making a decision, it was felt that As calibration would be of little use.

Information Recorded

1. School: This was designated by the first digit of the serial number.
2. Child's serial number; This was the last 3 digits of the serial number.
3. Child's sex, i.e. M or F.
4. Age of child. This was taken as the chronological age to the child's closest birthday.
5. Angle's Classification of the occlusion.

6. Subjective (qualitative) assessment of the child's treatment needs.
7. Objective (quantitative) assessment of the child's occlusion in terms of the **Godfrey Index**. All components of the index were scored and totals recorded.

Further information is
found below on:

- a) Angle's Classification
- b) Subjective Treatment-Needs Assessment
- c) Godfrey Index assessment.

(a) Angle's Classification

Categories scored:

N = Normal Occlusion

Molars and canines are in Angle's Class I relationship (neutroccclusion). In addition there was minimal overjet, overbite; no crowding, no rotations and no crossbites.

CI = Class I Malocclusion

Molars and canines are in Angle's Class I relationship but there may be excessive overjet and/or overbite or the presence of openbite. Rotations and/or crowding and/or crossbite may be present.

CII¹ - Class II Division 1 Malocclusion

Molars and canines are in Angle's Class II relationship (distocclusion). There is protrusion of maxillary anterior teeth. Values of overbite, crowding, crossbite, & rotations are variable.

CII² - Class II Division 2 Malocclusion

Molars and canines are in Angle's Class II relationship (distocclusion). There is a deep anterior overbite, minimal overjet. Maxillary central incisors are retroclined. Upper lateral incisors or canines may be proclined. Crowding, rotations and crossbite are variable.

CIII - Class III Malocclusion

Molars and canines are in Angle's Class III relationship (Mesiocclusion) Overbite, overjet, crowding, rotations and crossbite are variable.

Note that: - unilateral distocclusion and mesiocclusion are subdivision malocclusions and would not qualify for N or CI;

- the relationship of molars and canines is $>\frac{1}{2}$ cusp discrepancy to a disto- or mesiocclusion in order not to be in the N or CI categories.

(B) Subjective (Quantitative) Assessment of Occlusion in Terms of Treatment-Needs (As):

Godfrey (1963) defined these divisions as:

N (Normal)

No malocclusion present. No treatment needed

- (minor malocclusion)

No orthodontic treatment required

+ (slight to moderate malocclusion)

Simple, straightforward treatment required. General practitioner level of competence required.

++ (Relatively Complex Malocclusion)

Complex treatment-needs. Specialist level of competence required for treatment. This level included those that may be observed initially but would require later specialist treatment.

The basic philosophy of general practitioner (+) level treatment is that is is centred around an intact, uncrowded or minimally crowded mandibular arch.

The assignment of subjects to the different divisions was perceived to be highly subjective and may have altered somewhat as the survey progressed.

Quantitative (Objective) Assessment of Occlusion

This formed the Godfrey Index (occasionally called the Tamworth Index). This index was first used in *Tamworth in 1963*.

Discussion of the Tamworth Malocclusion Survey is presented earlier in this thesis (Pg. 147).

Scoring of the Godfrey Index is according to the following categories and weightings:

A_c (lower anterior crowding in the canine to canine region)

0 = no crowding

1 = crowding to the extent of $\frac{1}{2}$ a lower central incisor width

2 = crowding $> \frac{1}{2} \leq 1$ full width of a lower central incisor

3 = crowding > 1 central incisor width

This was estimated visually and recorded in the A_c column of the score sheet (see Fig. III-1)

PC_r and PC_L (Lower arch posterior crowding from molar to canine region)

Both sides were scored separately. Half scores were used for each division so as not to overstress the importance of posterior crowding c.f. anterior crowding.

Mixed dentitions showing any loss of mesiodistal dimension were recorded as $\frac{1}{2}$.

The assessment was estimated visually

0 = no crowding

$\frac{1}{2}$ = space loss or crowding $\leq \frac{1}{2}$ a bicuspid width

1 = space loss or crowding $> \frac{1}{2}$ 1 bicuspid width

$1\frac{1}{2}$ = space loss or crowding > 1 bicuspid width

+V or -V (Vertical overbite or openbite)

+ V - overbite
-V = openbite

+V (Overbite) was scored as follows

0 = incisal 1/3 of lower incisors covered
1 = middle 1/3 of lower incisors covered
2 = gingival 1/3 of lower incisors covered
3 = beyond the gingival margin of the lower incisors covered.

-V (Openbite) was scored as follows

1 = edge to edge relationships of the upper and lower permanent incisors
2 = Anterior openbite beyond an edge-to-edge relationship

+H or - H (Horizontal Overjet)

+H = maxillary protrusion (relative or actual)
- H = mandibular protrusion (relative or actual)

A score was derived by measuring with the aid of a toothpick (as mentioned earlier) which had graduations at 1.5mm and 3mm. Measures were taken from the lower incisors' labial surface to the upper incisors' labial surface.

+H (maxillary protrusion)

0 = overjet between 0 and 1.5 mm
1 = overjet >1.5mm ≤ 3mm
2 = over > 3mm
3 = lower incisal edges contacting or directed towards the palatal mucosa lingual to the gingival margins of the upper incisors.

-H (mandibular protrusion)

1 = 1 or more upper incisors lingual to the lower incisors but maintenance of anterior incisal contact

2 = upper incisors lingual to the lower incisors and no anterior contact

BAP , BAP (Buccal anteroposterior relationships)
R L Right and Left sides were scored separately

This was observed in centric occlusion. The mirror and adequate lighting enabled good visualisation of the premolar and molar regions without parallax error.

Scores were prefixed with either an "M" or a "D".

M = designated a mesial relationship of the lower molar on that side c.f. Class I molar relationship

D = designated a distal relationship of the lower molar on that side c.f. Class I molar relationship.

0 = normal cusp/groove relationship

1 = between cusp-groove and cusp to cusp relationship

2 = ≥ cusp to cusp relationship mesially or distally

3 = cusp to groove relationship mesially or distally

CR_R and CR_L (posterior crossbite)

This assessed transverse relationships in the molar/premolar segments. Right and left sides were scored separately.

If there was a crossbite due to buccoversion of the maxillary teeth, then the score was prefixed with "B".

1B = lingual cusps of the upper teeth contacting the buccal cusps of the lower teeth.

2B = lingual cusps of the upper teeth completely buccal to the buccal cusps of the lower teeth.

Where no buccoversion was present scoring was:

0 = normal buccolingual relationships

1 = buccal cusps of the upper teeth contacting buccal cusps of the lower teeth.

2 = buccal cusps of the upper teeth completely lingual to the buccal cusps of the lower teeth.

No diagnosis and scoring of functional mandibular shifts were made. When doubt existed over a score the lesser score was always assigned.

Once the Godfrey Index Score was allotted to each trait, the Total Godfrey Index Score was then calculated for each subject and recorded.

PART IV

100

As previously mentioned, in *Tamworth* in 1988, 208 12 and 15 year-old school children were examined using the Angle's Classification, a Subjective Assessment of their treatment need, and the Godfrey Index. In 1989, 120 *Lismore* schoolchildren aged from 12 to 15 years were examined using the same criteria. The *Lismore* group members were to serve as contemporary controls. *Lismore* is non-water-fluoridated, whereas *Tamworth* has been water-fluoridated since 1963.

Results from the 1963, 1973 and 1988 *Tamworth* and 1989 *Lismore* Malocclusion Surveys are presented below. A similar format to that of *Peel* is presented.

TABLE (IV-1)

Numbers seen by age and sex in *Tamworth, 1963 - 1988 and Lismore 1989.*

TAMWORTH										LISMORE				
1963					1973				1988				1989	
M		F			M		F		M		F		M	F
Age	12	15	12	15	12	15	12	15	12	15	12	15	12 - 15	12 - 15
	68	34	63	21	63	62	71	60	42	45	57	64	57	63
	102		84		125		131		87		121		120	
			186				256				208			

ANGLE'S CLASSIFICATION

Refer to the method section for definitions of each category of the Angle's Classification.

The distribution of subjects surveyed from *Tamworth 1963 - 1988* and *Lismore 1989* is presented in TABLE IV-2 below. Note that for *Lismore*, the age groups in each sex were combined.

TABLE (IV-2)

Angle's Classification (A ^c) Results for Tamworth and Lismore														
	1963				1973				1988				1989	
	Male		Female		Male		Female		Male		Female		Male	Female
	12	15	12	15	12	15	12	15	12	15	12	15	12-15	12-15
Normal	1	1	3	1	8	8	11	11	5	9	2	9	8	6
Class I	44	27	47	13	35	36	42	33	20	20	34	30	37	46
Class II Div 1	16	4	5	6	12	9	11	8	10	8	15	11	9	8
Class II Div 2	6	2	5	1	4	3	1	4	4	5	4	10	2	1
Class III	1	0	3	0	4	6	6	4	3	3	2	4	1	2
TOTALS	68	34	63	21	63	62	71	60	42	45	57	64	57	63
	102		84		125		131		87		121		120	
	186				256				208					

TABLE (IV-3) presents the percentage distribution of the Angle's
Classes according to age for males for *Tanworth 1963 to 1988*.

TABLE (IV-3)

Angle's Classification Male: Tanworth 1963 - 1988 % Distribution																
	Normal			Class I			Class II D1			Class II D2			Class III			TOTAL
	'63	'73	'88	'63	'73	'88	'63	'73	'88	'63	'73	'88	'63	'73	'88	
12 years-old	1.47	12.7	11.9	64.7	55.6	47.6	23.5	19	23.8	8.8	6	9.52	1.47	6	7.14	100
15 years-old	2.94	12.9	20	79.4	58	44.5	11.8	14.5	17.8	5.9	4.8	11.1	0	9.7	6.7	100

Figure (IV-1) represents the contents of TABLE IV-3 in graphical form to enable the reader to better visualise the changes from 1963-1988 in Tamworth.

FIGURE (IV-1)

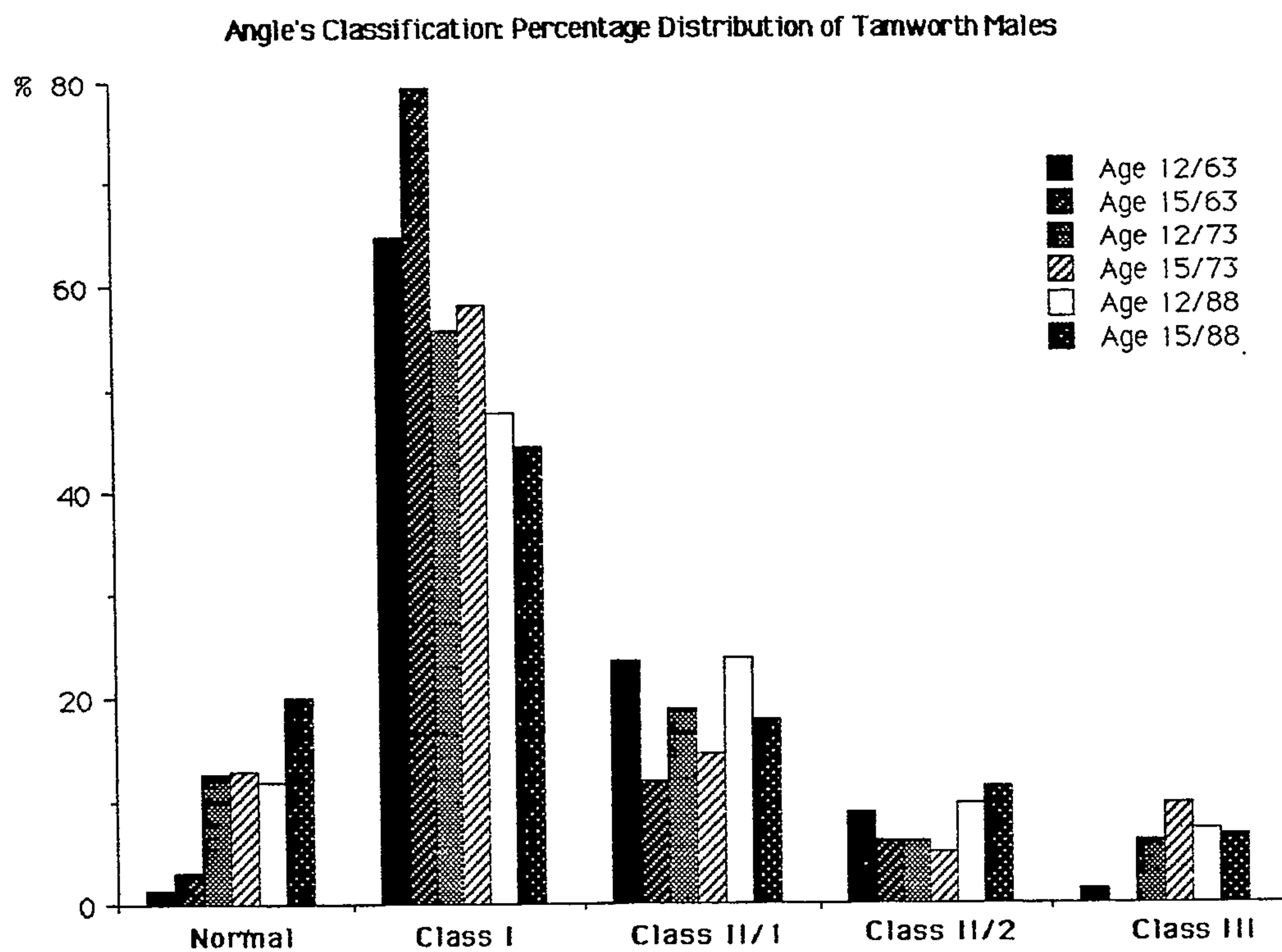


TABLE (IV-4) presents the percentage distribution of the Angle's Classes according to age for females for *Tamworth 1963 - 1988*.

TABLE (IV-4)

Angle's Classification Female : Tamworth 1963 - 1988 % Distribution																
	Normal			Class I			Class II D1			Class II D2			Class III			TOTAL
	'63	'73	'88	'63	'73	'88	'63	'73	'88	'63	'73	'88	'63	'73	'88	
12 years-old	4.76	15.5	3.5	74.6	59	59.65	7.93	15.5	26.3	7.93	1.4	7	4.76	8.5	3.5	100
15 years-old	4.76	18.3	14	61.9	55	46.9	28.6	13.3	17.2	4.76	6.7	15.62	0	6.7	6.25	100

FIGURE (IV-2) represents the contents of Table (IV-4) in graphical form.

FIGURE (IV-2)

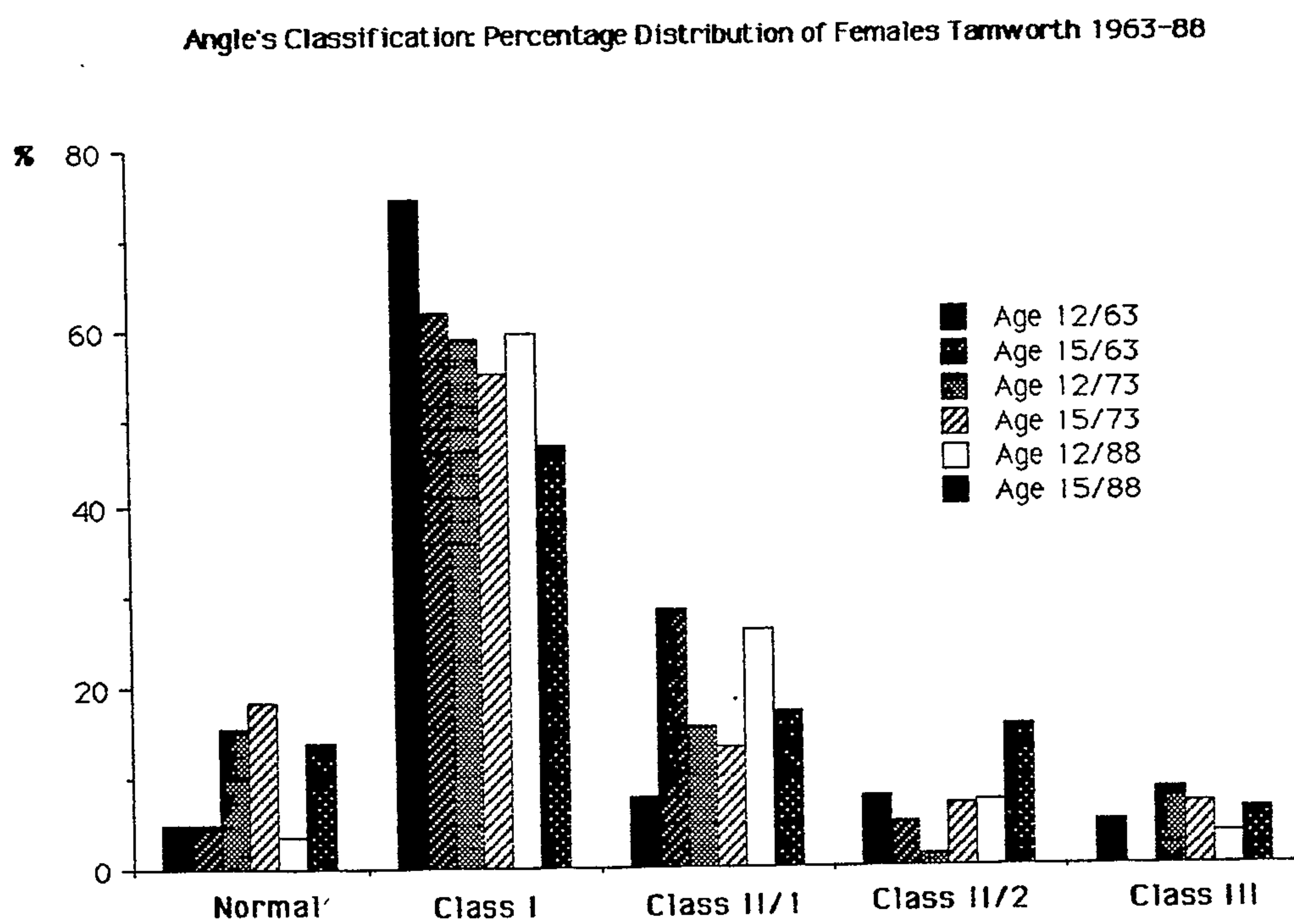


TABLE (IV-5) presents a comparison of the contemporary controls, *Lismore 1989 vs. Tamworth 1988* for the distribution of the Angle's Classes.

TABLE (IV-5)

Angle's Classes - Combined sexes/age groups Comparison of Contemporary Groups				
	TAMWORTH '88		LISMORE '89	
Normal	25	(12%)	14	(12%)
Class I	104	(50%)	83	(69%)
Class II Div 1	44	(21%)	17	(14%)
Class II Div 2	23	(11%)	3	(2.5%)
Class III	12	(6%)	3	(2.5%)
TOTAL	208	(100%)	120	(100%)

FIGURE (IV-3) represents the percentage distribution of the Angle's Classification for the combined Tamworth 1963-88, and Lismore 1989 groups (TABLE IV-6) in graphical form.

FIGURE (IV-3)

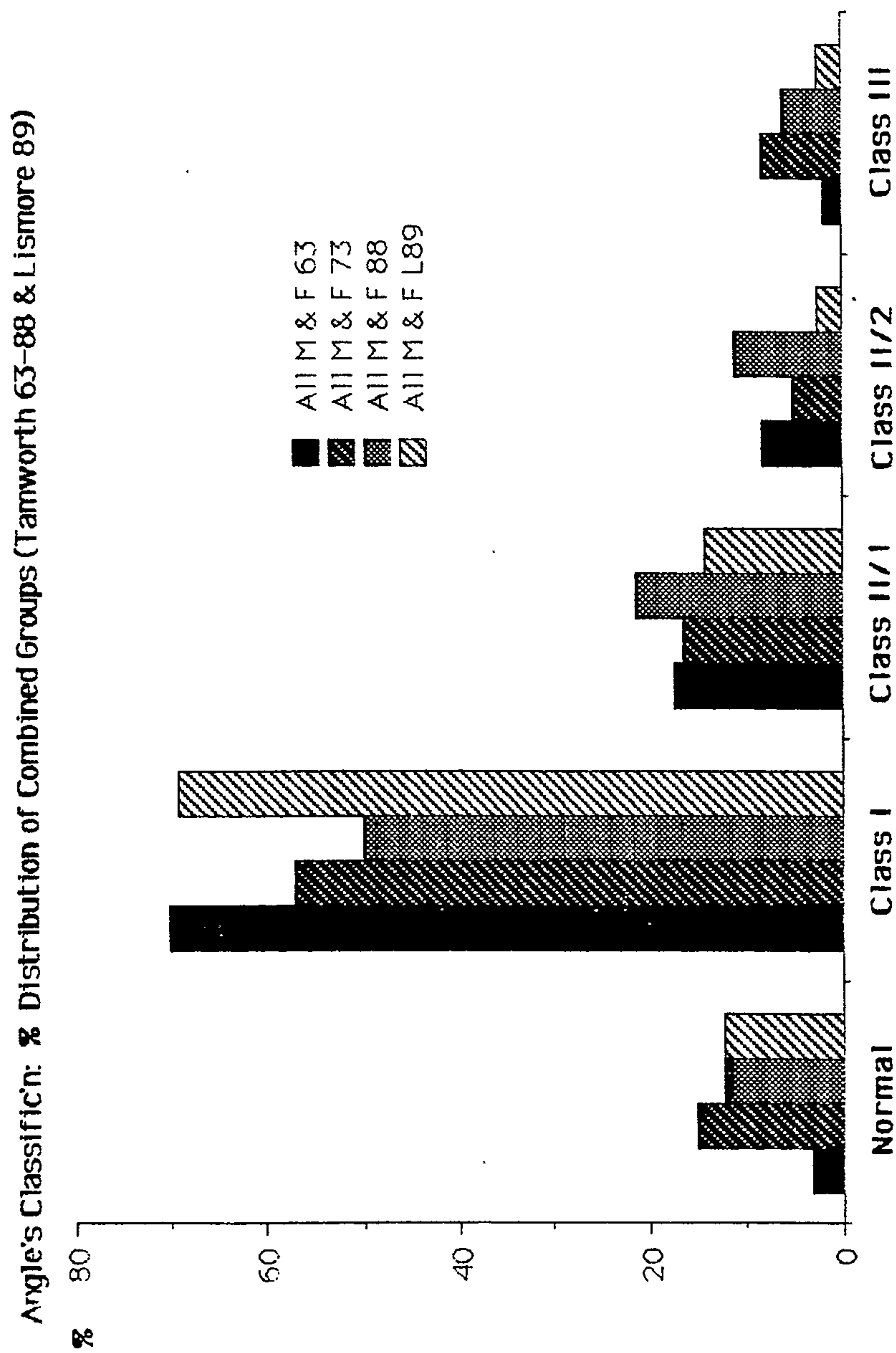


TABLE (IV-6) presents the percentage distribution of the Angle's Classes for the combined ages and sexes for *Tamworth 1963 - 1988* and for *Lismore 1989*.

TABLE (IV-6)

Angle's Classification Male and Female % Distribution All ages combined																				
	Normal				Class I				Class II D1				Class II D2				Class III			
	'63	'73	'88	'89	'63	'73	'88	'89	'63	'73	'88	'89	'63	'73	'88	'89	'63	'73	'88	'89
	Lismore				Lismore				Lismore				Lismore				Lismore			
All ages combined	3	15	12	12	70	57	50	69	17	16	21	14	8	5	11	2.5	2	8	6	2.5

TABLE IV-7 represents the means of each Godfrey Index trait (including the Godfrey Index total and Subjective Assessment of Treatment Need) for each category of the Angle's Classification scored for *Tamworth* for 1988. The ages and sexes are combined.

TABLE IV-7

Tamworth 1988: Means for each category of the Angle's Classification (Total Group 208) Overall Group											
Angle's Class	λ_s	λ_c	PC _L	PC _R	(+V) and V(-x)	(+H) and H(-x)	BAP _L	BAP _R	CrL	CrR	GI TOTAL
Normal	0	0.24	0.02	0.02	0.24	0.36	0.2	0.32	0	0	1.4
Class I	1.7885	0.8077	0.03365	0.05769	0.7596	1.1635	0.3558	0.3558	0.125	0.03846	3.7548
Class II D1	2.3636	0.75	0.0688	0.125	1.1818	2.0682	1.75	1.5682	0.22727	0.22727	8.1477
Class II D2	2.4783	0.6957	0	0.06522	1.6522	1.3913	2	1.3478	0.04348	0	7.1957
Class III	2.5	0.8333	0.08333	0.4167	-0.3333	0.000	1.4167	1.4167	0.41667	0.5	6.0417

Table (IV-8) represents similar data for *Lismore (1989)* for the overall group. Note also that the D, M, F and DMFT scores were obtained for each Lismore subject.

TABLE (IV-8)

Means for each category of the Angle's Classification: Overall group - Lismore 1989 (Total 120)							
	As mean	Ac mean	PC _L mean	PC _r mean	(+V) V (-V) mean	(+I) H (-I) mean	BAP-R mean
Normal	0.0000	0.5714	0.00000	0.00000	0.6429	0.7857	0.3571
Class I	1.8434	0.9277	0.14458	0.07831	0.7831	1.4458	0.2892
Class II D1	2.2353	0.8235	0.11765	0.02941	0.8824	2.1765	2.1765
Class II D2	2.3333	0.3333	0.00000	0.00000	2.3333	2.3333	2.3333
Class III	2.0000	1.3333	0.16667	0.00000	-0.3333	0.6667	2.000
All	1.7000	0.8667	0.12083	0.05833	0.7917	1.4750	0.6583

Means for each category of the Angle's Classification: Overall group - Lismore 1989 (Total 120)								
	BAP-L mean	CR-L mean	CR-R mean	GI mean	D mean	M mean	F mean	DMFT mean
Normal	0.1429	0.00000	0.00000	2.5000	0.5000	0.00000	0.9286	1.4286
Class I	0.3373	0.13253	0.10843	4.3675	0.6220	0.13253	1.2892	2.0361
Class II D1	1.5882	0.00000	0.00000	8.1471	0.7059	0.05882	0.9412	1.7059
Class II D2	2.6667	0.00000	0.00000	10.0000	0.6667	0.00000	0.3333	1.0000
Class III	1.3333	0.33333	0.33333	6.5000	1.0000	0.00000	0.6667	1.6667
All	0.5750	0.10000	0.08333	4.8792	0.6303	0.100000	1.1583	1.8833

Tamworth Intragroup Statistical Analyses

The results from *Tamworth* were first compared with each other for age, sex and year effects.

The analysis used was a contingency Table analysis using the chi square test which employed orthogonal decomposition.

The following abbreviations are used here and will be used in subsequent results.

df = degrees of freedom

X² = chi squared result

ns = not statistically significant

ss = statistically significant

p = denotes the probability that the result occurred by chance

p < 0.05 = denotes a probability of a chance result of less than 1 in 20.

1. Males: 1963 vs 1988			
observation	df	X ²	
Age	4	4.5	n.s.
Year	4	20.3	ss. (p<0.01)
(a) N vs Rest	1	12.1	s.s. (p<0.01)
(b) CI, CII ¹ , CII ² vs CIII	1	5.6	s.s. (p<0.05)
Age x Year	4	1.9	n.s.

Comment:

(a) Orthogonal decomposition of the year effect revealed that for males in *Tamworth* in 1988 there was a statistically

significant increase in the number of Normal occlusions compared with the 1963 sample.

(b) Also, there was a statistically significant increase in the number of Class III malocclusions recorded in 1988 compared with 1963.

2. Females: 1963 vs 1988			
observation	df	X ²	
Age	4	8.13	n.s.
a) N vs Rest	1	4.2	s.s. (p<0.05)
Year	4	7.23	n.s.
b) CI vs. CII ¹	1	4.1	s.s. (p<0.05)
Age x year	4	8.89	n.s.

Comment:

- a) No overall age effect but N vs. Rest was significant at 5% level. There was a statistically significant increase in the number of Normal occlusions from 12 to 15 years of age.
- b) There were significantly more Class II Div 1 malocclusions recorded in 1988 than in 1963. This seemed to be at the expense of Class I malocclusions.

3. Males: 1973 vs 1988			
observation	df	X ²	
age	4	1.4	n.s.
year	4	3.6	n.s.
Age x Year	4	1.1	n.s.

Comment: There was no significant effect found.

4. Females: 1973 vs 1988			
observation	df	X ²	
Age	4	7.99	n.s.
(a) CI, CII ¹ vs CII ²	1	5.8	s.s (p<0.05)
Year	4	10.5	s.s. (p<0.05)
(b) CI, CII ¹ vs CII ²	1	4.2	s.s. (p<0.05)
Age x Year	4	2.1	n.s.

Comment:

- a) There were significantly more females with Class II Div 2 malocclusions in the 15 year-old age group in both years.
- b) Also there were significantly more Class II Div 2 malocclusions recorded in *1988* compared with *1973* in *Tamworth*. This appeared to be related mostly to the 15 year-old group.

5. 1963 vs 1973 vs 1988 - for pooled sexes and age groups		
observation	df	X ²
Age	4	7.06 n.s.
a) N vs Rest	1	4.9 s.s. (p<0.05)
Year	8	36.02 s.s. (p<0.01)
b) 1963 vs 1973, 1988 for N vs Rest	1	14.99 s.s. (p<0.01)
c) 1963 vs 1973, 1988 for CI, CII ¹ , CII ² v CIII	1	7.9 s.s. (p<0.01)
d) 1973 vs 1988 for CI, CII ¹ vs CII ²	1	5.9 s.s. (p<0.05)
Age x Year	8	6.56 n.s.

Comment:

- a) The 15 year-old age group showed the greatest number of Normal occlusions.
- b) 1973 and 1988 showed a statistically significant increase in the number of Normal occlusions compared with 1963.
- c) Significantly more Class III malocclusions were recorded in 1973 and 1988 compared with 1963.
- d) In 1988 there was a statistically significant increase in the number of Class II Div 2 malocclusions recorded compared with 1973.

Lismore 1989 - Intragroup Statistical Analysis

Statistical Analysis revealed no age or sex effect of any significance, intragroup.

Tamworth 1963 - 1988 vs Lismore 1989 - Statistical Analysis

Tamworth 1963, 1973 and 1988 were all compared with *Lismore 1989*. To better show the differences between all groups with time.

Additionally, *Kirby (1990)* found calculation errors in *Peel's (1973)* results. She felt it was best to reanalyse the total Angle's Classification.

1. Lismore 1989 vs Tamworth 1963		
observation	df	X ²
Age	4	0.62 n.s.
Lismore '89 vs Tamworth '63	4	11.6 s.s. (p<0.025)
(a) Normal vs Rest	1	8.5 s.s. (p<0.005)
Age x Lismore '89 vs Tamworth '63	4	3.4 n.s.

Comment:

- (a) There were significantly more Normal occlusions in *Lismore 1989* than in *Tamworth 1963*.

2. Lismore 1989 vs Tamworth 1973		
observation	df	X ²
Age	4	2.4 n.s.
Lismore '89 vs Tamworth '73	4	7.4 n.s.
(a) CI, CII ¹ , CII ² vs CIII	1	4.4 s.s. (p<0.05)
Age x Lismore '89 vs Tamworth '73	4	0.2 n.s.

Comment:

- (a) There was a significantly higher proportion of Class III malocclusions in *Tamworth* in 1973 than in *Lismore* in 1989.

3. Lismore 1989 vs Tamworth 1988		
observation	df	X ²
Age	4	8.4 n.s.
Lismore '89 vs Tamworth '88	4	15.7 s.s. (p<0.01)
a) CI vs CII ¹	1	5.4 s.s. (p<0.025)
b) CI, CII ¹ vs CII ²	1	8.4 s.s. (p<0.005)
Age x Lismore '89 vs Tamworth '88	4	1.9 n.s.

Comment:

- a) There was a significantly greater proportion of Class I malocclusions in *Lismore* 1989 than in *Tamworth* in 1988, while Normal occlusions were of approximately equal proportions in each town.
- b) There was a significantly greater proportion of Class II Div 2 malocclusions in *Tamworth* in 1988 than in *Lismore* in 1989.

SUBJECTIVE ASSESSMENT OF TREATMENT NEED (As)

Refer to the METHOD section for the key to symbols used in this assessment.

The Results of this assessment with respect to *Tamworth 1963 - 1988* and *Lismore 1989* are presented below in TABLE (IV-9)

TABLE (IV-9)

Subjective Assessment of Treatment-Need (As) - Tanworth and Lismore														
	TANWORTH										LISMORE			
	1963				1973				1988		1989			
	M		F		M		F		M		F		M	F
	12	15	12	15	12	15	12	15	12	15	12	15	12 - 15	12 - 15
	Age													
N	1	1	3	1	8	8	11	11	5	9	2	9	8	6
-	12	5	19	1	20	20	24	24	13	20	21	26	20	28
+	33	23	29	13	19	17	24	12	1	0	7	4	9	9
++	22	5	12	6	16	17	12	13	23	16	27	25	20	20
	68	34	63	21	63	62	71	60	42	45	57	64	57	63
	102		84		125		131		87		121		120	
	186				256				208					

The percentage distribution of the A_s groupings for *Tamworth* 1963 - 1988 for 12 and 15 year-old males is presented in Table (IV-10) below.

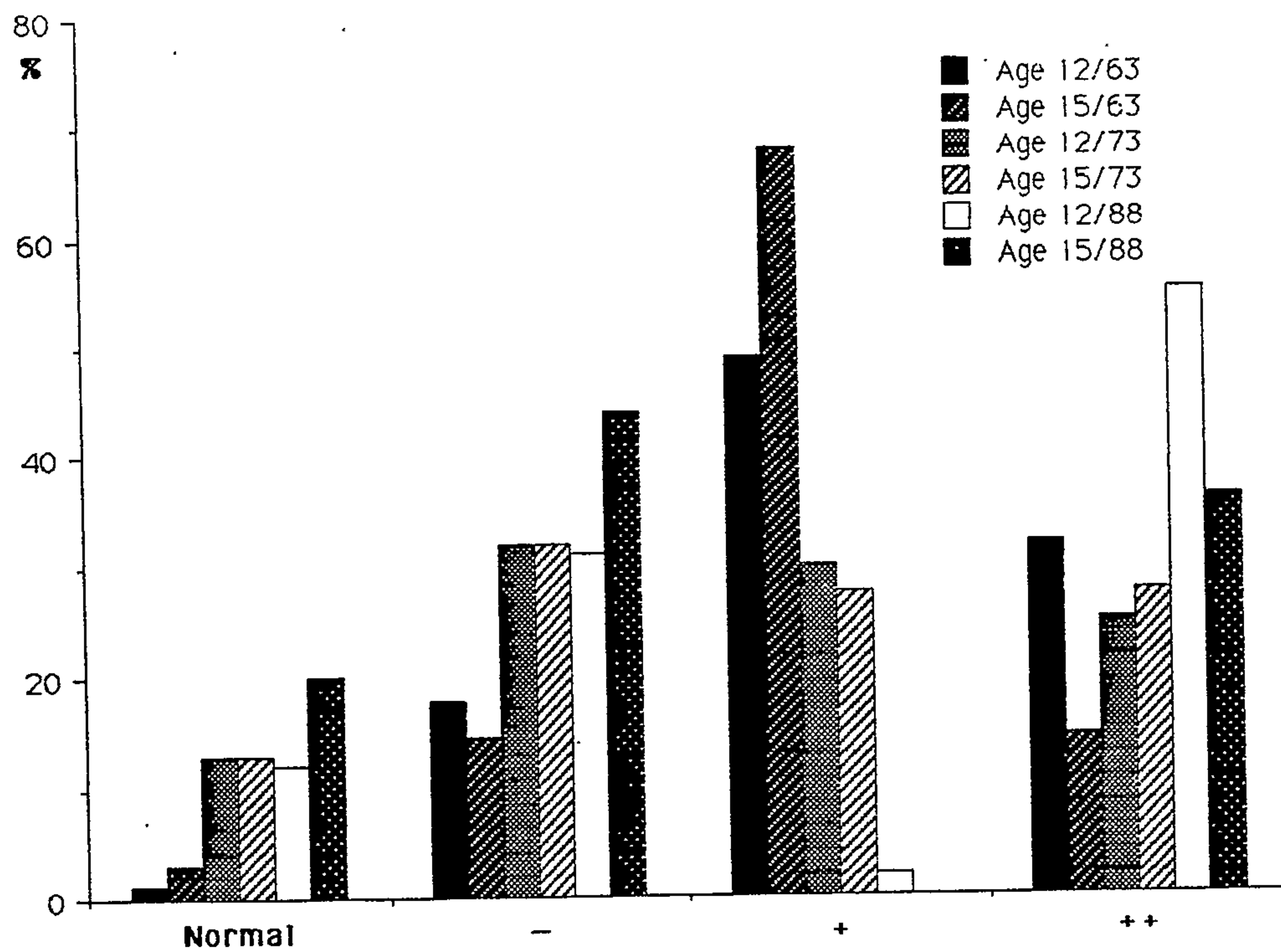
TABLE (IV-10)

Subjectively Assessed Treatment-Need (A _s): Male % Distribution <i>Tamworth</i> 1963 - 1988												
Age	N			-			+			++		
	'63	'73	'88	'63	'73	'88	'63	'73	'88	'63	'73	'88
12 y-o	1%	13%	12%	18%	32%	31%	49%	30%	2%	32%	25%	55%
15 y-o	3%	13%	20%	14.5%	32%	44%	68%	27.5%	0%	14.5%	27.5%	36%

The results of Table (IV-10) are presented graphically in Figure (IV-4) below.

FIGURE (IV-4)

Subjective Assessment of Treatment Need: Percentage Distribution of Males Tamworth 1963-88



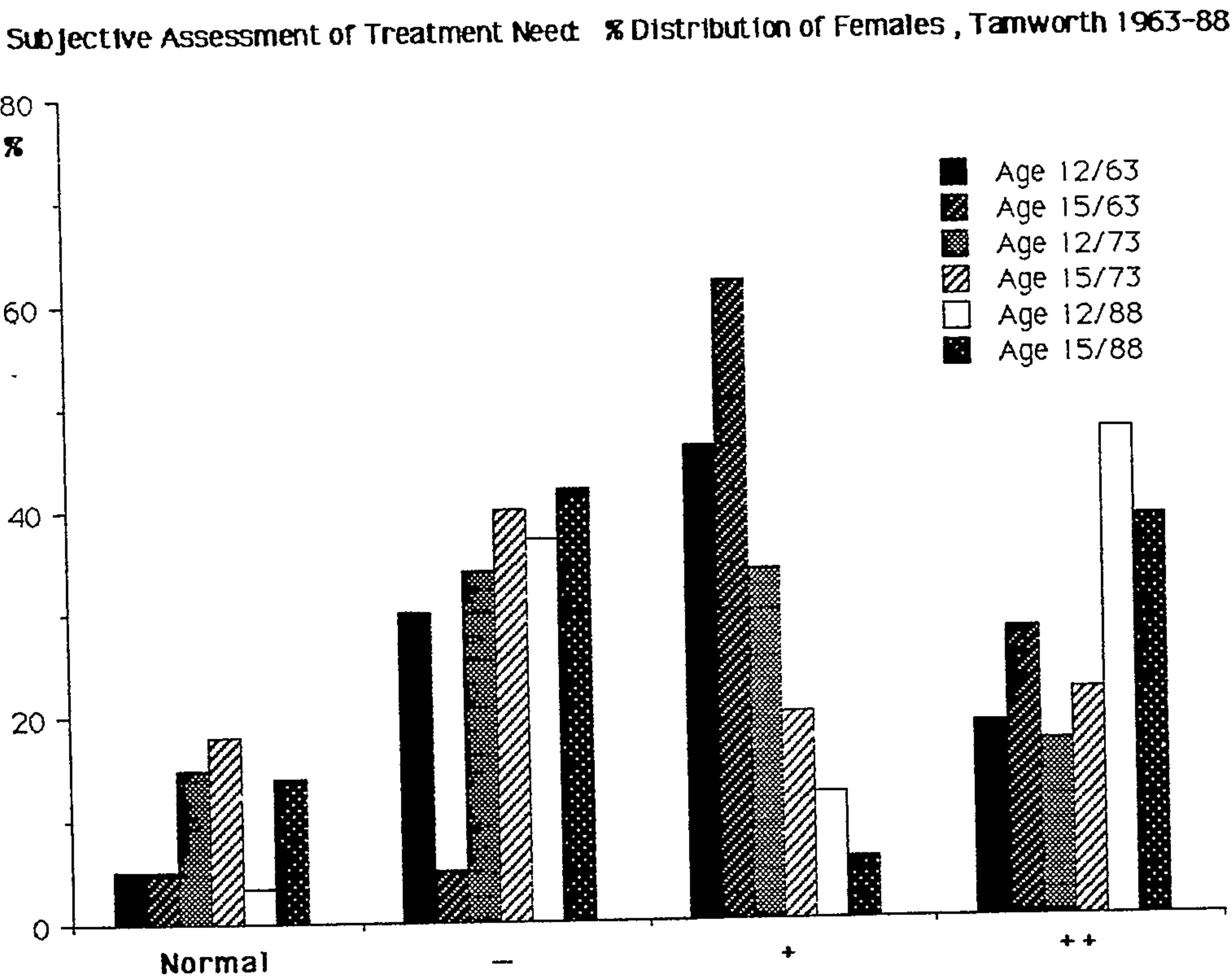
The percentage distribution of the A_s groupings for *Tamworth 1963 - 1988* for 12 and 15 year-old females is presented in Table (IV-11) below.

TABLE (IV-11)

Subjectively Assessed Treatment-Need (As): Female												
% Distribution												
Tanworth 1963 - 1988												
Age	N			-			+			++		
	'63	'73	'88	'63	'73	'88	'63	'73	'88	'63	'73	'88
12 y-o	5%	15%	3.5%	30%	34%	37%	46%	34%	12.3%	19%	17%	47.4%
15 y-o	5%	18%	14%	5%	40%	41%	62%	20%	6%	28%	22%	39%

The results of Table (IV-11) are presented graphically in Figure (IV-5) below.

FIGURE (IV-5)



The As groupings actual and percentage distributions of *Lismore 1989* and *Tamworth 1988* are presented below in Table (IV-12) These figures are for the overall age/sex groups of both towns.

TABLE (IV-12)

Subjective Assessment of Treatment-need: Combined sexes/ages		
	Tamworth '88	Lismore '89
N	25 (12%)	14 (12%)
-	80 (38%)	48 (40%)
+	12 (6%)	18 (15%)
++	91 (44%)	40 (33%)
TOTAL	268 (100%)	120 (100%)

Table (IV-13) below presents the A_s groupings of the combined sex and age groups for *Tamworth* 1963, 1973, 1988 and *Lismore* 1989.

TABLE (IV-13)

SUBJECTIVELY ASSESSED TREATMENT-NEED (A_s) FEMALES
COMBINED SEXES AND AGES:
PERCENTAGE DISTRIBUTION FOR TAMWORTH (1963-88) AND LISMORE (1989)

Subjectively Assessed Treatment-Need (A _s): Combined sexes and ages & Distribution															
I				-				+				++			
'63	'73	'88	L. '89	'63	'73	'88	L. '89	'63	'73	'88	L. '89	'63	'73	'88	L. '89
3%	15%	12%	12%	20%	34%	38%	40%	53%	28%	6%	15%	24%	23%	44%	33%

Figure (IV-6) represents the data of Table (IV-13) in graphical form.

FIGURE (IV-6)

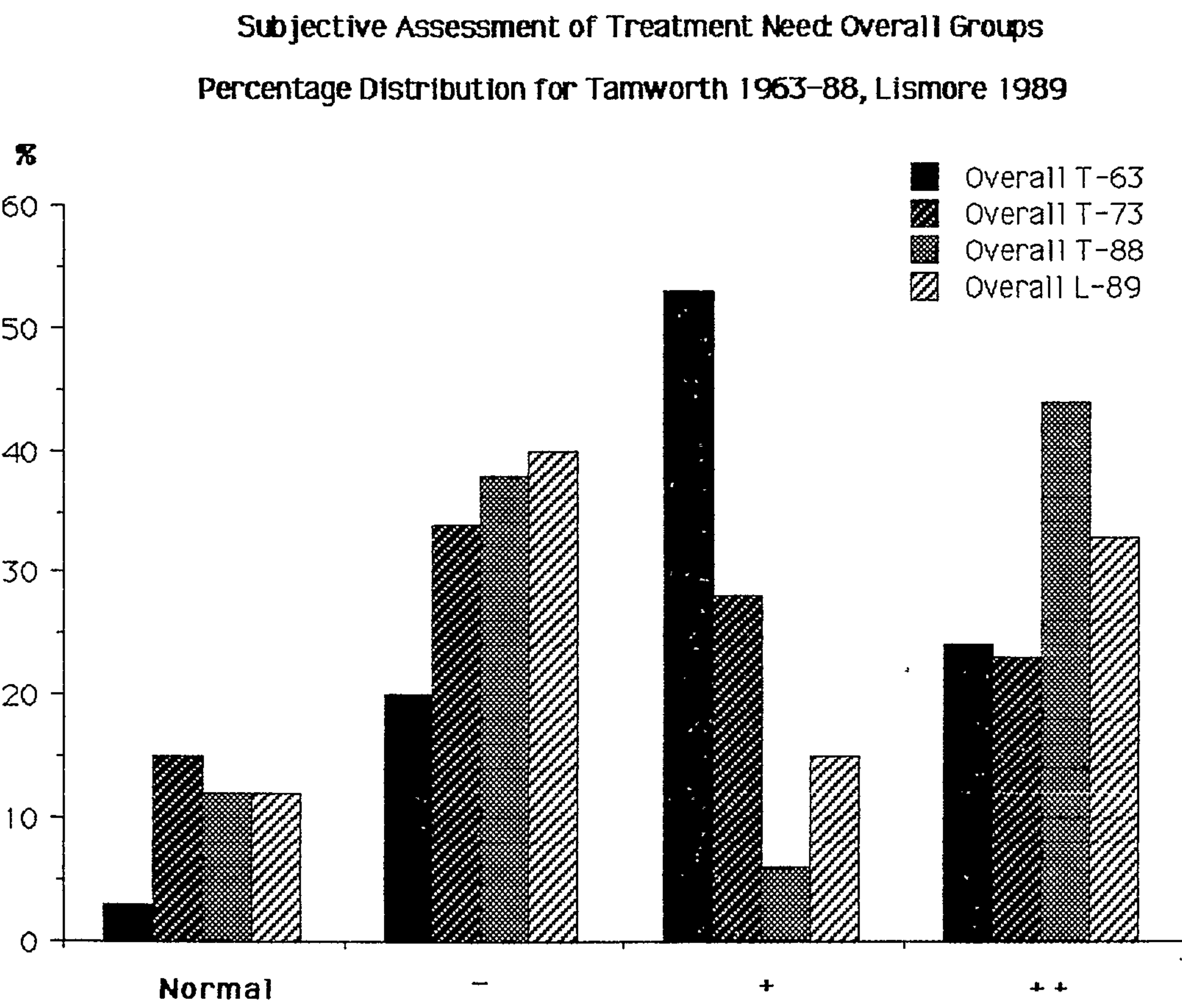


Table (IV-14) represents the mean score of each Godfrey Index trait for each of the groupings of A_s for the overall *Tamworth Group* of 1988.

TABLE (IV-14)

MEAN GI SCORES FOR EACH CATEGORY OF
SUBJECTIVE ASSESSMENT OF TREATMENT-NEED
(A_s - TAMWORTH (1988))

	A _c Mean	P _c L Mean	PC _r Mean	V Mean	H Mean	BAP _r Mean
N	0.2400	0.02000	0.02000	0.2400	0.3600	0.2000
-	0.6125	0.03125	0.05000	0.7375	1.1000	0.6000
+	0.5833	0.00000	0.12500	1.3333	1.5833	1.1667
++	0.9560	0.05495	0.08791	0.9890	1.5055	1.2637

	BAP _L Mean	CR _L Mean	CR _r Mean	GI Mean
N	0.3200	0.00000	0.00000	1.4000
-	0.5500	0.10000	0.00000	3.7812
+	1.3333	0.25000	0.25000	6.6250
++	1.0330	0.19780	0.18681	6.6484

Table (IV-15) represents the mean score of each Godfrey Index trait for each of the groupings of A_s for the overall *Lismore* group of 1989.

TABLE (IV-15)

Lismore - Means for each category of A _s for each GI Trait and DMF and DMFT						
	A _c Mean	P _c L Mean	P _C r Mean	V Mean	H Mean	BAP _r Mean
N	0.5714	0.00000	0.00000	0.6429	0.7857	0.3571
-	0.7708	0.07292	0.03125	0.8542	1.5417	0.5208
+	1.0000	0.08333	0.08333	0.9444	1.4444	0.7222
++	1.0250	0.23750	0.10000	0.7000	1.6500	0.9000

TABLE (IV-15) (continued)

Lismore A _c - Means for each category of A _s for each GI Trait and DMF and DMFT						
	BAP _L Mean	CR _L Mean	CR _r Mean	GI Mean	D Mean	M Mean
N	0.1429	0.00000	0.00000	2.5000	0.5000	0.0000
-	0.5000	0.04167	0.04167	4.3750	0.5745	0.0000
+	0.5000	0.05556	0.00000	5.1667	0.5000	0.0000
++	0.8500	0.22500	0.20000	6.1875	0.8000	0.30000

	F Mean	DMFT Mean
N	0.9286	1.4286
-	0.9792	1.5417
+	2.0000	2.5000
++	1.0750	2.1750

Tamworth Statistical Analysis - Intragroup

The results from *Tamworth* were first compared with each other for age, sex and year effects. The analysis used was a contingency Table analysis employing chi square and orthogonal decomposition. Again *Peel's (1973)* results were reanalysed.

1. Tamworth: 1973 vs 1988 for Males		
observation	df	X ²
Age	3	1.9 n.s.
Year	3	28.5 s.s. (p<0.005)
(a) + vs ++	1	26.7 s.s. (p<0.005)
Age x Year	3	2.9 n.s.

Comment:

- (a) Orthogonal decomposition of the significant year effect revealed that there were significantly more ++ than + malocclusions recorded in 1988 in *Tamworth* than in *Tamworth 1973*. The examiner (RM) partially attributes this to his personal difference in interpretation of examining criteria. The examiner feels that at that time he thought very few occlusions could be competently handled by a general practitioner.

2. Tamworth: 1973 vs 1988 for Females		
observation	df	X ²
Age	3	6.5 n.s.
Year	3	26.1 s.s. (p<0.005)
(a) + vs ++	1	22.8 s.s. (p<0.005)
Age x Year	3	2.0 n.s.

Comment:

- (a) Again, orthogonal decomposition of the year effect revealed that significantly more ++ and + scores were recorded for females in 1973 than in 1988 in *Tamworth*. The same comments regarding examiner feelings apply.

3. Tamworth: 1963 vs 1988 for Males		
observation	df	X ²
Age	3	6.9 n.s.
Year	3	68.6 s.s. (p<0.005)
(a) + vs ++	1	40.5 s.s. (p<0.005)
(b) N, - vs +, ++	1	25.8 s.s. (p<0.005)
Age x Year	3	4.2 n.s.

Comment:

- (a) Orthogonal decomposition of the year effect again reveals that significantly more ++ than + scores were recorded for *Tamworth* in 1988 than in 1963. Again, this may be attributable to examiner preferences.
- (b) Additionally, there was a significant increase in the number of males with N and - malocclusions in *Tamworth* in

1988 than in *Tamworth* in 1963. This also has some importance for the Treatment Need vs. No Treatment Need category of the Subjective Assessment of Treatment Need to be discussed later.

4. Tamworth: 1963 vs 1988 for females		
observation	df	X ²
Age	3	6.1 n.s.
Year	3	43.5 s.s. (p<0.005)
(a) + vs ++	1	35.7 s.s. (p<0.005)
(b) N, - vs +, ++	1	7.7 s.s. (p<0.001)
Age x Year	3	5.1 n.s.

Comment:

- (a) Orthogonal decomposition of the year effect again reveals significantly more ++ than + scores for *Tamworth* in 1988 than in 1963 for females. Examiner preferences may again explain this occurrence.
- (b) Additionally, there was a significant increase in the number of females with N and - scores over +, ++ scores in *Tamworth* 1988 compared with *Tamworth* 1963.

5. Tamworth: 1963 vs 1973 vs 1988 (No age effects, so combined ages)		
observation	df	X ²
Year	6	123.18 s.s. (p<0.005)
(a) For 1973 vs 1988, for + vs ++	1	37.6 s.s. (p<0.005)
(b) For 1963 vs 1973, 1988 for + vs ++	1	43.2 s.s. (p<0.005)
(c) For 1963 vs 1973, 1988 for N,- vs +, ++	1	38.7 s.s. (p<0.005)
Sex	3	2.8 n.s.
Year x Sex	6	6.7 n.s.

Comment:

- (a) There were significantly more ++ than + scores recorded in *Tamworth 1988 than in 1973.*
- (b) There were significantly more ++ than + recorded in *Tamworth 1973 and 1988* than in *Tamworth 1963.*
- (c) There were significantly more N and - scores recorded in *Tamworth 1973 and 1988* than in *Tamworth 1963.*

There were no statistically significant differences between the numbers of N and - scores recorded in *Tamworth 1973 and 1988.*

Lismore Intragroup Statistical Analysis

Separate calculations revealed no age or sex effect intragroup.

TAMWORTH 1963-1988 vs Lismore 1989 - Statistical Analysis

1. Lismore 1989 vs Tamworth 1963

observation	df	X ²
Age	3	3.1 n.s.
Lismore 1989 vs Tamworth 1963	3	48.1 s.s (p<0.005)
a) + vs ++	1	20.47 s.s. (p<0.005)
b) N,- vs +,++	1	26.37 s.s (p<0.005)
c) Age x town	3	8.3 s.s (p<0.05)

Comment:

- There were significantly more ++ than + scores in *Lismore 1989* compared with *Tamworth 1963*.
- There were significantly more N,- scores recorded in *Lismore 1989* compared with *Tamworth, 1963*.
- 15 year-olds in *Tamworth 1963* comprised a significantly lower proportion of N,- scores compared with *Lismore 1989*.

2. Lismore 1989 vs Tamworth 1973		
observation	df	X ²
Age	3	2.6 n.s.
Lismore 1989 vs Tamworth 1973	3	10.8 s.s. (p<0.025)
(a) + vs ++	1	9.36 s.s. (p<0.005)
Age x town	3	5.2 n.s.

Comment:

- a) Orthogonal decomposition revealed that again for ++ vs +, there were significantly more ++ scores registered in *Lismore 1989* than in *Tamworth 1973*. This may reflect examiner preferences (RM).

3. Lismore 1989 vs Tamworth 1988		
observation	df	X ²
Age	3	9.4 n.s. nearly significant at (p<0.05)
a) N,- vs +,++	1	8.9 s.s. (p<0.05)
Lismore '89 vs Tamworth '88	3	9.2 n.s.
b) + vs ++	1	9.1 s.s. (p<0.005)
Age x town	1	2.1 n.s.

Comment:

- a) 15 year-olds comprised a greater proportion of the N,- scores in *Tamworth 1988* and *Lismore 1989* compared with 12 year-olds.
- b) Again, orthogonal decomposition reveals that for + vs ++, significantly more ++ than + scores were recorded in *Tamworth 1988* compared with *Lismore* in 1989. The

present writer feels that his criteria for + were too strict and that despite some easing of personal ideals with respect to the differences between + and ++ levels of treatment need between 1988 and 1989, a statistically significant difference was still to exist for + vs ++ scores in any comparison concerning *Tamworth 1988*. Another possible explanation for this effect will be found in the Discussion section.

Overall, there was no significant difference between the 2 towns for the proportions of normal (N) occlusions and (-) minor occlusions compared with the more treatment-related malocclusions (+, ++), when comparing contemporary groups.

TREATMENT VS NO-TREATMENT-NEED

This result is arrived at by using the findings of the Subjective Assessment of Treatment-Need (As). A division is made between the As categories of N,- and +,++. The Treatment-Need category includes subjects with + or ++ scores, whereas the No-Treatment-Need category includes subjects with the N and - scores.

Table (IV-16) presents the findings of this result category for *Tamworth 1963 - 1988* and *Lismore 1989* for the respective age and sex groups.

TABLE (IV-16)

SUBJECTIVE ASSESSMENT OF TREATMENT-NEED - TREATMENT-NEEDED VS NO-TREATMENT NEEDED (N,- vs +,++)														
1963				1973				1988				Lismore 1989		
	Male		Female		Male		Female		Male		Female		Male	Female
	12 yo	15yo	12yo	15yo	12yo	15yo	12yo	15yo	12yo	15yo	12yo	15yo	12-15	12-14
Treatment- Needed (+,++)	55	28	41	19	35	34	36	25	24	16	34	29	29	29
No-Treatment- Needed (n,-	13	6	22	2	28	28	35	35	18	29	23	35	28	34
TOTAL	68	34	63	21	63	62	71	60	42	45	57	64	57	63
	102		84		125		131		87		121			
	186				256				208				120	

Table IV-17) presents the findings of "Treatment vs No-Treatment" for males 12 and 15 years-old for *Tamworth 1963 - 1988* as a percentage distribution.

TABLE (IV-17)

Subjectively Assessed Treatment Need: Male TAMWORTH 1963 - 1988 - PERCENTAGE DISTRIBUTION						
Age	No Treatment-Needed N and - combined			Treatment-Needed + and ++ combined		
	'63	'73	'88	'63	'73	'88
12 yrs	19%	44%	43%	81%	56%	57%
15 yrs	18%	45%	64%	82%	55%	36%

Figure (IV-7) represents the findings of Table (IV-17) graphically as a percentage distribution.

FIGURE (IV-7)

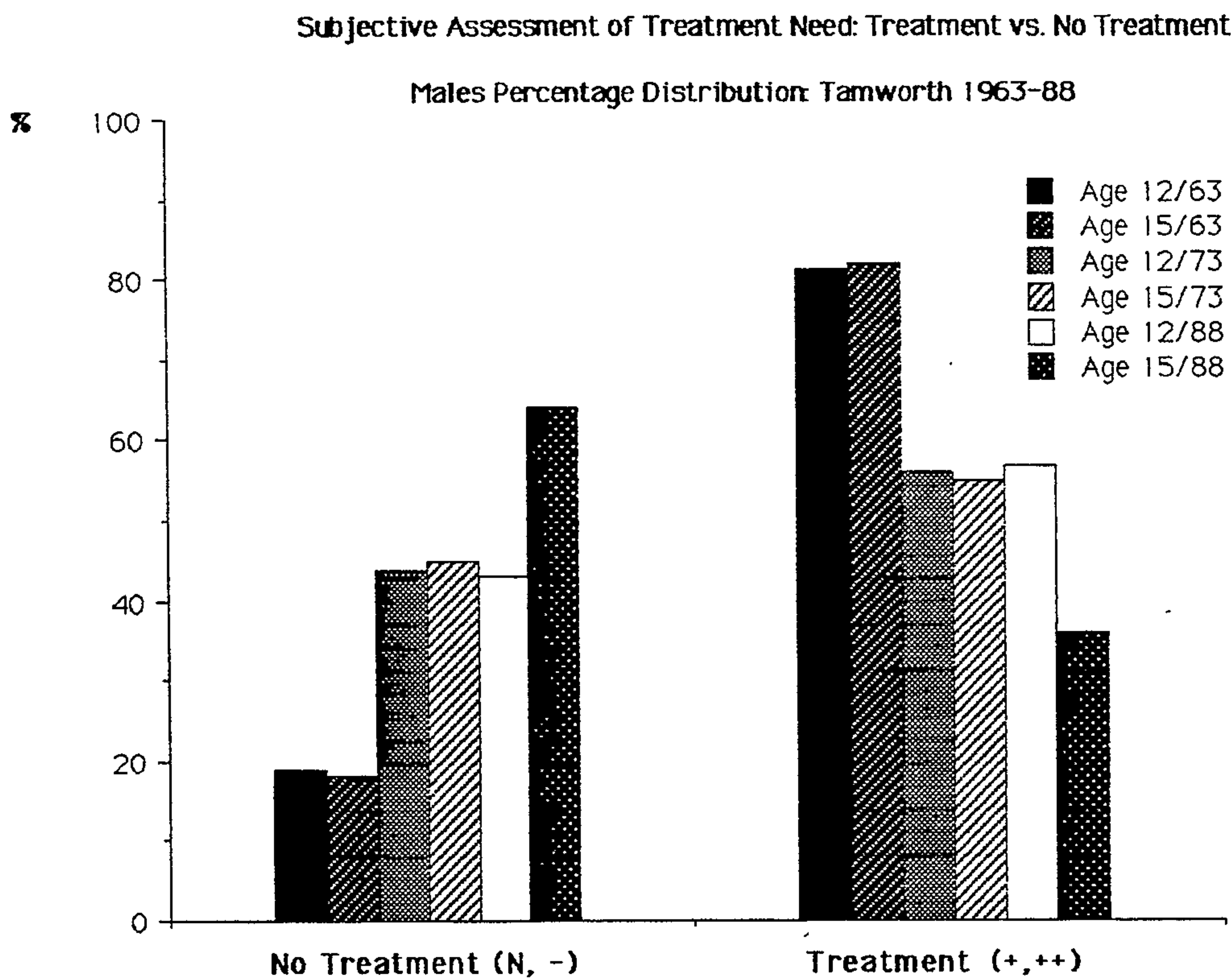


Table (IV-18) presents the findings of "Treatment vs No-Treatment" for females 12 and 15 years-old for *Tamworth 1963 - 1988* as a percentage distribution.

Table (IV-18)

Subjectively Assessed Treatment-Need: Female TAMWORTH 1963 - 1988 - PERCENTAGE DISTRIBUTION						
Age	No Treatment Needed <i>N</i> and - combined			Treatment Needed <i>+</i> and <i>++</i> combined		
	'63	'73	'88	'63	'73	'88
12 yrs	35%	49%	40%	65%	51%	60%
15 yrs	10%	58%	55%	90%	42%	45%

Figure (IV-8) represents the findings of Table (IV-18) graphically as a percentage distribution.

FIGURE (IV-8)

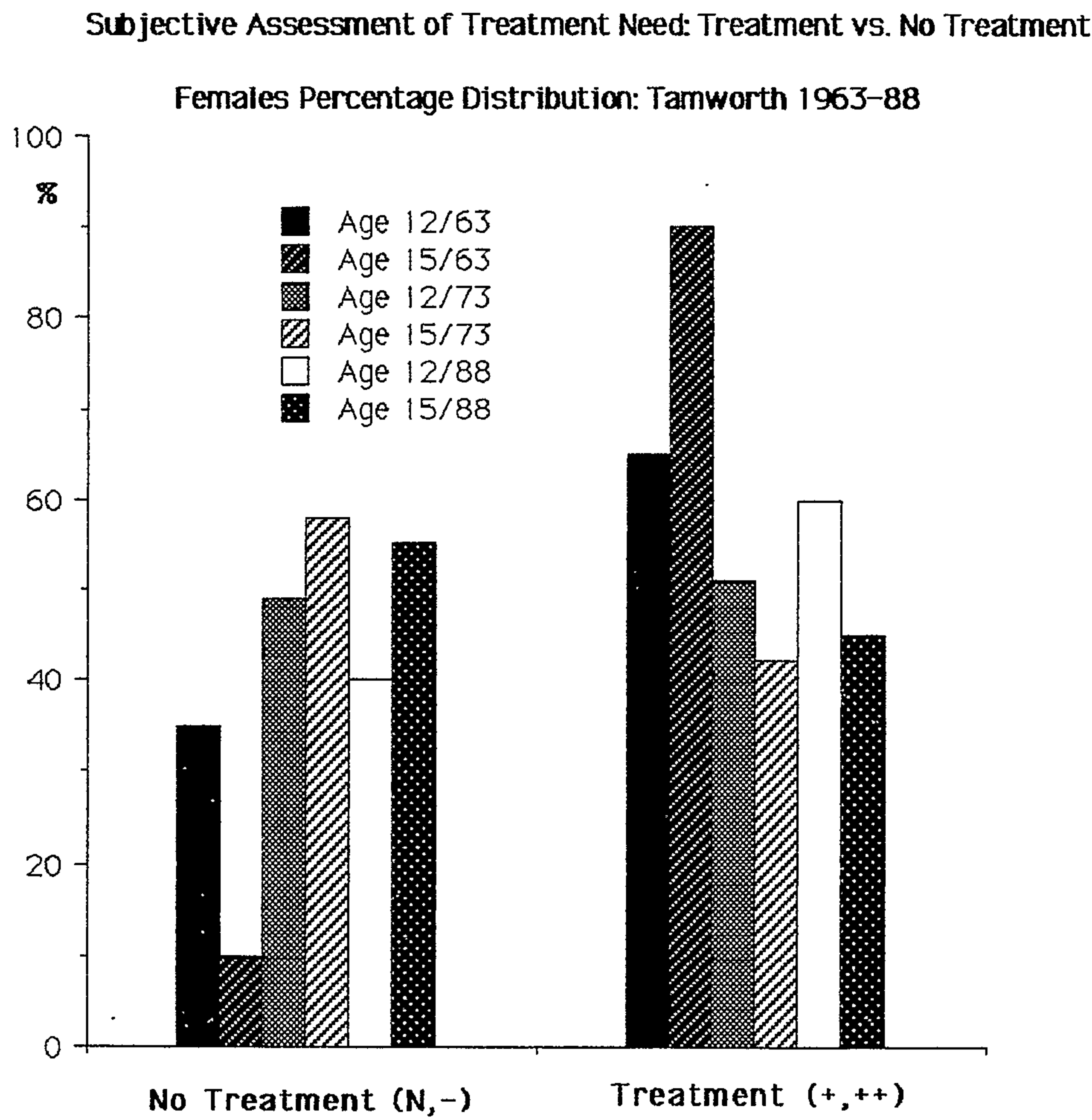


Table (IV-19) below serves as a comparison of contemporary groups - *Lismore 1989* and *Tamworth 1988* with respect to Treatment Need vs No-Treatment-Need.

Table (IV-19)

COMPARISON OF CONTEMPORARY GROUPS - COMBINED SEXES/AGES				
SUBJECTIVE ASSESSMENT OF TREATMENT NEED (A ₂)				
NO TREATMENT-NEEDED (N,-) vs TREATMENT-NEED (+,++)				
(TOTAL GROUPS AND PERCENTAGE DISTRIBUTION)				
	TANWORTH '88		LISMORE '89	
No Treatment-Need (N,-)	105	(50.5%)	62	(51.5%)
Treatment-Need (+,++)	103	(49.5%)	58	(48.5)
TOTAL	208	(100%)	120	(100%)

Table (IV-20) presents the overall percentage distributions for *Tamworth 1963 - 1988* and *Lismore 1989* for the combined ages and sexes.

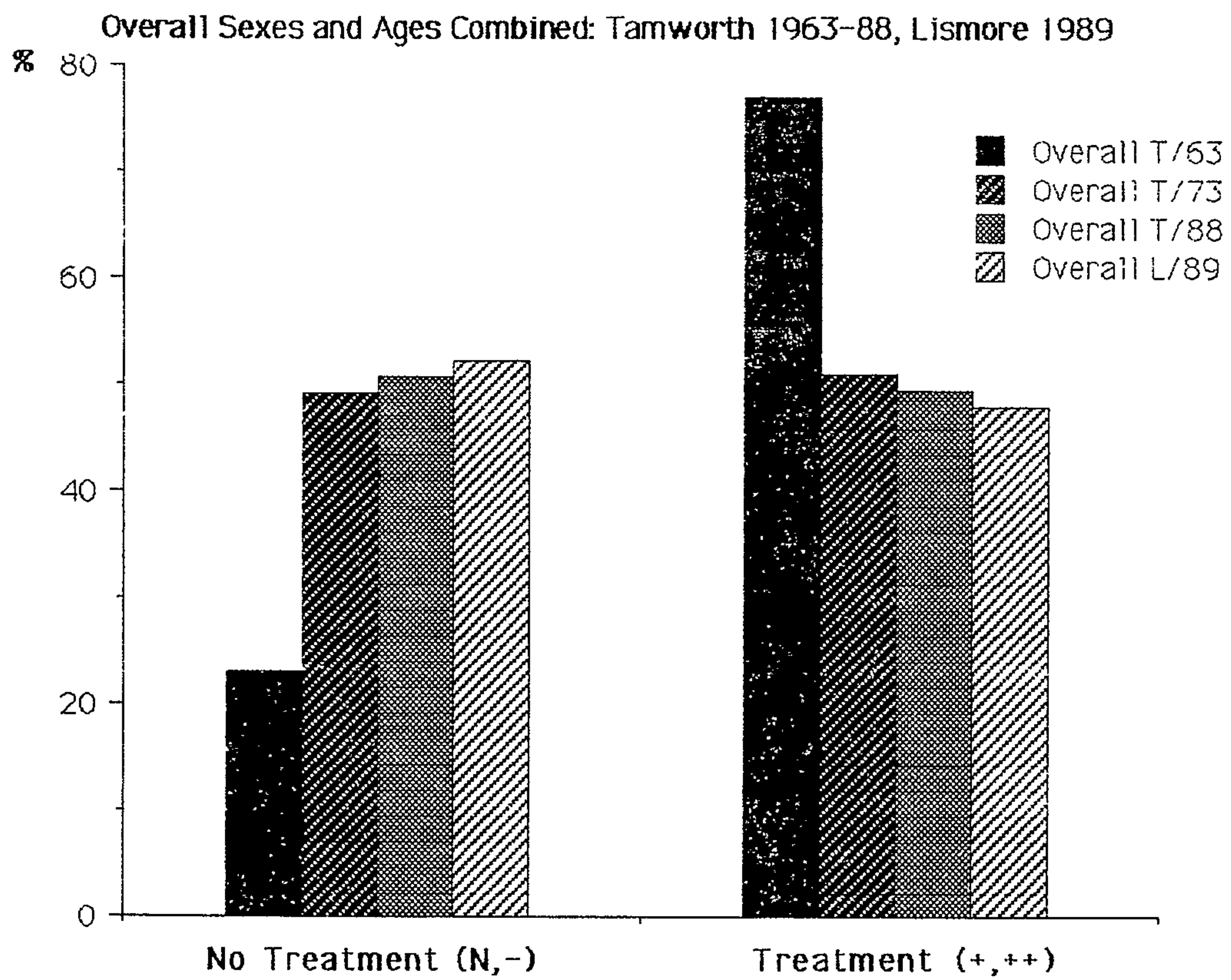
Table (IV-20)

Subjectively Assessed Treatment Need: Combined Ages/sexes							
PERCENTAGE DISTRIBUTION: TAMWORTH 1963 - 1988 and LISMORE, 1989							
No Treatment Needed (N and - combined)				Treatment Needed (+ and ++ combined)			
'63	'73	'88	Lismore '89	'63	'73	'88	Lismore '89
23%	49%	50.5%	52%	77%	51%	49.5%	48%

Figure (IV-9) represents the results of Table (IV-20) graphically as a percentage distribution.

FIGURE (IV-9)

Subjective Assessment of Treatment Need: Treatment vs. No Treatment



Statistical Analysis of Tamworth Groups:
For Treatment-Need (+,++) vs No Treatment-Need (N,-)

TAMWORTH 1973 vs 1988 for Males

From the orthogonal decomposition of the A_s scores previously, the N,- vs +,++ chi squared analysis showed no statistically significant difference.

Tamworth 1973 vs 1988 for Females

Again from the orthogonal decomposition of the A_s scores previously, the N,- vs +,++ chi square analysis showed no statistically significant difference between 1973 and 1988.

Tamworth 1963 vs 1988 for Males

Orthogonal decomposition of the year effect of the A_s (previously) for this group revealed a statistically significant increase in the N,- scores of 1988 over 1963. The chi squared value was 25.8 at 3 df yielding statistical significance at $p < 0.005$. Significantly more males required treatment in 1963 than in 1988 in *Tamworth*.

Tamworth 1963 vs 1988 for Females

From analysis of the A_s scores, the age effect, on orthogonal decomposition revealed that there were significantly less N,- scores in *Tamworth* in 1963 compared with *Tamworth* in 1988. The chi squared value was 7.7 at 1 d.f. yielding $p < 0.01$. Significantly more females required treatment in 1963 than in 1988 in *Tamworth*.

Tamworth 1963 vs 1973 vs 1988.

Again, analysing the overall groups showed that, on orthogonal decomposition, there were significantly less N,- scores in *Tamworth* in 1963 than in *Tamworth* in 1973 and 1988. The chi squared value was 38.7

at 1 d.f. for a $p < 0.005$. The differences between these scores for 1973 and 1988 were not significant.

Statistical Analysis of Tamworth 1963 - 1988 vs Lismore 1989:
For Treatment-Need vs No-Treatment-Need (N,- vs +,++)

Earlier calculations of the A_s scores had revealed no sex effect. Therefore, this was not taken into account in this section.

Tamworth 1963 vs Lismore 1989

Orthogonal decomposition revealed that there were significantly more N,- scores in *Lismore 1989* than in *Tamworth* in 1963. Additionally, 15 year-olds in *Tamworth* in 1963 formed a significantly lower proportion of N and - scores registered.

The analysis of A_s for this group is reproduced below:

A_s		
observation	df	X^2
Age	3	3.1 n.s.
Lismore '89 vs Tamworth '63	3	4.8 s.s. $p < 0.005$
+ vs ++	1	20.47 s.s. $p < 0.005$
Age x town	3	8.3 s.s. $p < 0.05$
N,- vs +,++	1	26.37 s.s. $p < 0.005$

Tamworth 1973 vs Lismore 1989

Previous A_s analysis revealed that no statistically significant differences were found between these 2 groups with respect to N,- vs +,++ scores. That is, Treatment-Need differences between *Tamworth* 1973 and *Lismore 1989* were not statistically significant.

Tamworth 1988 vs Lismore 1989

Orthogonal decomposition of the A_s age effect for this group revealed a statistically significant result for N,- vs +,++. 15 year-olds showed a higher proportion of N,- scores in both towns. The N,- vs +,++ decomposition revealed a chi squared value of 8.9 at 1 d.f. for $p < 0.005$.

This would seem to indicate that with increasing age, from 12 years to 15 years, there is an improvement in occlusion (or a reduction in malocclusion) with respect to Need-of-Treatment assessed subjectively, in these contemporary samples.

Despite possible inter-examiner differences with respect to scoring + vs ++ occlusions, it is assumed that this subgroup difference has no effect on N,- versus +,++, i.e. Treatment-Needed versus No-Treatment-Needed scoring. It is thus assumed that despite possible examiner differences reflected in the A_s scoring (with respect to + vs ++), that the present Treatment-Needed vs No-Treatment-Needed is valid for *1963 - 1988 Tamworth* and *1989 Lismore*. Additionally, it is felt that since the A_s scores in *1988 Tamworth* and *1989 Lismore* were performed by the same examiner, then the A_s comparisons between these 2 towns are valid. However, the comparisons involving *Tamworth '88* and *Lismore '89*, with either of *Tamworth 1963* or *1973*, could be unreliable for the differentiation between + and ++ malocclusions. The N and - scores, as mentioned above, however, would be expected to be still valid, especially when compared with the +,++ combined.

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Table (IV21-IV34) shows the distribution of scores by actual number and percentages for each of the 2 contemporary groups, *Tamworth 1988* and *Lismore 1989*, for each trait of the Godfrey Index.

Table (IV-21)			
Lower Anterior Crowding (A _c): Combined sexes/ages Actual and % Distributions			
	Tamworth 1988		Lismore 1989
0	77	(37%)	26 (22%)
1	115	(55%)	84 (70%)
2	14	(7%)	10 (8%)
3	2	(1%)	0 (0%)
Total	208	(100%)	120 (100%)

Table (IV-22)			
Posterior Crowding (Left) (P _{C_L}):Combined sexes/ages Actual and % Distributions			
	Tamworth 1988		Lismore 1989
0	195	(93%)	94 (78%)
0.5	12	(6%)	23 (19%)
1	2	(1%)	3 (3%)
Total	208	(100%)	120 (100%)

Table (IV-23)			
Posterior Crowding (Right) (PC _r): Combined sexes/ages Actual and % Distributions			
	Tamworth 1988		Lismore 1989
0	183	(88%)	106 (88%)
0.5	22	(11%)	14 (12%)
1	3	(1%)	0 (0%)
Total	208	(100%)	120 (100%)

Table (IV-24)			
Overbite (V): Combined ages/sexes Actual and % Distributions			
	Tamworth 1988		Lismore 1989
-2	4	(2%)	3 (2.5%)
-1	4	(2%)	3 (2.5%)
0	71	(34%)	38 (32%)
1	85	(41%)	52 (43%)
2	34	(16%)	20 (17%)
3	10	(5%)	4 (3%)
Total	208	(100%)	120 (100%)

Table (IV-25)			
Overjet (H): Combined ages/sexes Actual and % Distributions			
	Tamworth 1988		Lismore 1989
-2	1	(0%)	0 (0%)
-1	2	(1%)	0 (0%)
0	43	(21%)	16 (13%)
1	76	(37%)	37 (31%)
2	77	(37%)	61 (51%)
3	9	(4%)	6 (5%)
Total	208	(100%)	120 (100%)

Table (IV-26)				
Buccal Anteroposterior Deviation:(RHS) (BAP _R):				
Combined ages/sexes				
Actual and % Distributions				
	Tamworth 1988		Lismore 1989	
0	103	(49%)	71	(59%)
1	47	(23%)	27	(22%)
2	39	(19%)	14	(12%)
3	19	(9%)	8	(7%)
Total	208	(100%)	120	(100%)

Table (IV-27)			
Buccal Anteroposterior Deviations (LHS) (BAP _L): Combined ages/sexes Actual and % Distributions			
	Tamworth 1988		Lismore 1989
0	109	(52%)	72 (60%)
1	54	(26%)	34 (28%)
2	27	(13%)	7 (6%)
3	18	(9%)	7 (6%)
Total	208	(100%)	120 (100%)

Table (IV-28)			
Posterior Crossbite (RHS) (CR _R): Combined ages/sexes Actual and % Distributions			
	Tamworth 1988		Lismore 1989
0	192	(92%)	112 (93%)
1	12	(6%)	4 (3.5%)
2	4	(2%)	4 (3.5%)
Total	208	(100%)	120 (100%)

Table (IV-29)			
Posterior Crossbite (LHS) (CR _L): Combined ages/sexes Actual and % Distributions			
	Tamworth 1988		Lismore 1989
0	187	(90%)	114 (95%)
1	13	(6%)	2 (1.5%)
2	8	(4%)	4 (3.5%)
Total	208	(100%)	120 (100%)

Table (IV-30

Godfrey Index Total: Combined ages/sexes
Actual and % Distributions

GI score	Tamworth 1988		Lismore 1989	
0	9	4%	2	1.5%
1	14	7%	4	3%
2	22	10.5%	12	10%
2.5	4	2%	3	2.5%
3	30	14.5%	15	12.5%
3.5	1	0.5%	1	1%
4	27	13%	18	15%
4.5	2	1%	2	1.5%
5	19	9%	18	15%
5.5	2	1%	6	5%
6	18	9%	9	7.5%
6.5	2	1%	6	5%
7	13	6%	8	7%
7.5	4	2%	1	1%
8	10	5%	5	4%
8.5	4	2%	-	-
9	9	4%	4	3%
9.5	3	1.5%	-	-
10	7	3%	2	1.5%
10.5	-	-	1	1%
11	2	1%	1	1%
11.5	1	0.5%	1	1%
12	2	1%	1	1%
12.5	1	0.5%	-	-
13	2	1%	-	-
Total	208	(100%)	120	(100%)

Table (IV-31) shows the distribution of scores for the Godfrey Index (GI) total according to age and sex for Tamworth 1988.

Table (IV-31)				
Godfrey Index Scores for sexes/ages				
Tamworth 1988				
GI score				
	F 15 yr	F 12 yr	M 15 yr	M 12 yr
0	= 5	= 1	= 3	= -
1	= 3	= -	= 5	= 5
2	= 10	= 3	= 6	= 3
2.5	= 4	= 1	= -	= -
3	= 5	= 13	= 8	= 4
3.5	= -	= -	= 1	= -
4	= 6	= 7	= 5	= 8
4.5	= -	= 2	= -	= -
5	= 6	= 5	= 2	= 6
5.5	= -	= -	= 2	= -
6	= 5	= 5	= 2	= 6
6.5	= 2	= -	= -	= -
7	= 5	= 4	= 2	= 3
7.5	= 2	= -	= 1	= 1
8	= 1	= 6	= 2	= 1
8.5	= 2	= 2	= -	= -
9	= 2	= 2	= 1	= 4
9.5	= 2	= -	= 1	= -
10	= 2	= 2	= 2	= 1
10.5	= -	= -	= -	= -
11	= 1	= 1	= -	= -
11.5	= -	= -	= 1	= -
12	= -	= 2	= -	= -
12.5	= -	= -	= 1	= -
13	= 1	= 1	= ?	= -
	Total = 64	Total = 57	Total = 45	Total = 42

Table (IV-32) shows the data for the GI total for *Lismore* according to sex only (ages grouped).

Table (IV-32)		
Godfrey Index Scores :		
Males and Females: Lismore 1989		
GI score		
	Females	Males
	12-14 yr	12-15 yr
0	= 2	= -
1	= 3	= 1
2	= 6	= 6
2.5	= 2	= 1
3	= 8	= 7
3.5	= 1	= -
4	= 8	= 10
4.5	= 2	= -
5	= 9	= 9
5.5	= 3	= 3
6	= 4	= 5
6.5	= 6	= -
7	= 2	= 6
7.5	= -	= -
8	= 3	= 2
8.5	= -	= -
9	= 2	= 2
9.5	= -	= -
10	= -	= 2
10.5	= 1	= -
11	= -	= 1
11.5	= 1	= -
12	= -	= 1
12.5	= -	= -
13	= -	= -
Total = 63		Total = 57
120		

Table (IV-33) shows the mean scores of the combined ages and sexes group from *Tamworth 1988* for each Godfrey Index Trait.

Table (IV-33)				
Tamworth 1988: Godfrey Index				
Means of Lumped ages/sexes (Total group 208)				
	mean	median	s.d.	s.e. mean
Lower anterior crowding	0.7163	1	0.6304	0.0437
Posterior crowding LHS	0.0385	0	0.1506	0.0104
Posterior crowding RHS	0.0673	0	0.1911	0.0132
Overbite (+V and -V)	0.8221	1	0.9591	0.0665
Overjet (+H and -H)	1.2163	1	0.8881	0.0616
Buccal anteroposterior relationship deviation RHS	0.8750	1	1.0185	0.0706
Buccal anteroposterior relationship deviation LHS	0.7788	0	0.9776	0.0678
Posterior crossbite LHS	0.1394	0	0.4448	0.0308
Posterior crossbite RHS	0.0962	0	0.3549	0.0246
Godfrey Index Total	4.913	4	2.968	0.2006

s.d. = standard deviation

s.e. = standard error of the mean

Table (IV-34) shows the mean scores of the combined ages and sexes group, from *Lismore 1989*, for each Godfrey Index Trait.

Table (IV-34)				
Lismore 1989: Godfrey Index				
Means of Lumped ages/sexes (Total group 120)				
	mean	median	s.d.	s.e. mean
Lower anterior crowding	0.8667	1	0.5335	0.0487
Posterior crowding LHS	0.1208	0	0.2425	0.0221
Posterior crowding RHS	0.0583	0	0.1612	0.0147
Overbite (+V and -V)	0.7917	1	0.9517	0.0869
Overjet (+H and -H)	1.4753	2	0.7882	0.0720
Buccal anteroposterior relationship deviation RHS	0.6583	0	0.9303	0.0849
Buccal anteroposterior relationship deviation LHS	0.5750	0	0.8468	0.0773
Posterior crossbite LHS	0.1	0	0.3975	0.0363
Posterior crossbite RHS	0.0833	0	0.3798	0.0347
Godfrey Index Total	4.879	5	2.423	0.221

s.d. = standard deviation

s.e. = standard error of the mean

Tables (IV-35 to IV-50) show the means and standard deviations for the various *Tamworth 1963-88* and *Lismore '89* groups, for each trait.

Table (IV-35)					
Lower Anterior Crowding (Ac)					
Age			Group Size	$\bar{x}$	s.d.
14-15-16 yrs	Male	1963	103	0.573	0.62
14-15-16 yrs	Male	1973	152	0.592	0.568
12 and 15 yrs	Male	1988	87	0.7586	0.6281
12 to 15 yrs	Male	1989(L)	57	0.9474	0.5484
14-15-16 yrs	Female	1963	86	0.465	0.547
14-15-16 yrs	Female	1973	157	0.599	0.529
12 and 15 yrs	Female	1988	121	0.6860	0.6329
12 to 15 yrs	Female	1989(L)	63	0.7937	0.5730
Overall ages and sexes combined		1963	189	0.524	0.58)pooled
		1973	309	0.596	0.55)s.d.= 0.63
		1988	208	0.716	0.63
		1989(L)	120	0.867	0.53

Table (IV-36)

Posterior Crowding (RHS) - (PC_r)

Age			Group Size	$\bar{x}$	s.d.
14-15-16 yrs	Male	1963	103	0.15	0.327
14-15-16 yrs	Male	1973	152	0.151	0.230
12 and 15 yrs	Male	1988	87	0.05172	0.17109
12 to 15 yrs	Male	1989(L)	57	0.06140	0.16557
14-15-16 yrs	Female	1963	86	0.192	0.369
14-15-16 yrs	Female	1973	157	0.169	0.303
12 and 15 yrs	Female	1988	121	0.07851	0.20421
12 to 15 yrs	Female	1989(L)	63	0.05556	0.15840
Overall ages and sexes combined		1963	189	0.169	0.348) pooled
		1973	309	0.160	0.267) s.d. =
	"	1988	208	0.067	0.191) 0.261
	"	1989(L)	120	0.058	0.161)

Table (IV-37)					
Posterior Crowding (LHS) - (PC _L)					
Age			Group Size	$\bar{x}$	s.d.
14-15-16 yrs	Male	1963	103	0.116	0.290
14-15-16 yrs	Male	1973	152	0.171	0.321
12 and 15 yrs	Male	1988	87	0.01724	0.09176
12 to 15 yrs	Male	1989(L)	57	0.08772	0.21387
14-15-16 yrs	Female	1963	86	0.140	0.366
14-15-16 yrs	Female	1973	157	0.188	0.317
12 and 15 yrs	Female	1988	121	0.05372	0.18030
12 to 15 yrs	Female	1989(L)	63	0.15079	0.26389
Overall ages and		1963	189	0.200	0.328) pooled
sexes combined		1973	309	0.180	0.319) s.d. =
"	"	1988	208	0.039	0.150) 0.266
"	"	1989(L)	120	0.121	0.242)

Table (IV-38)					
Openbite (-V)					
Age			Group Size	$\bar{x}$	s.d.
14-15-16 yrs	Male	1963	103	0.087	0.316
14-15-16 yrs	Male	1973	152	0.148	0.437
12 and 15 yrs	Male	1988	87		
12 to 15 yrs	Male	1989(L)	57		
14-15-16 yrs	Female	1963	86	0.116	0.389
14-15-16 yrs	Female	1973	157	0.147	0.421
12 and 15 yrs	Female	1988	121		
12 to 15 yrs	Female	1989(L)	63		
Overall ages and		1963	189	0.100	0.352
sexes combined		1973	309	0.147	0.429
"	"	1988	208	0.062	0.3123
"	"	1989(L)	120	0.075	0.347

Table (IV-39)

Overbite (+V)

Age	Group Size	$\bar{x}$	s.d.
14-15-16 yrs Male 1963	103	1.039	1.057
14-15-16 yrs Male 1973	152	0.678	0.751
12 and 15 yrs Male 1988	87		
12 to 15 yrs Male 1989(L)	57		
14-15-16 yrs Female 1963	86	0.861	0.996
14-15-16 yrs Female 1973	157	0.459	0.712
12 and 15 yrs Female 1988	121		
12 to 15 yrs Female 1989(L)	63		
Overall ages and sexes combined 1963	189	0.958	1.026
" " 1973	309	0.582	0.731
" " 1988	208	0.880	0.851
" " 1989(L)	120	0.8667	0.809

Table (IV-40)

Overjet (+H)

Age	Group Size	$\bar{x}$	s.d.
14-15-16 yrs Male 1963	103	0.874	0.813
14-15-16 yrs Male 1973	152	0.599	0.944
12 and 15 yrs Male 1988	87		
12 to 15 yrs Male 1989(L)	57		
14-15-16 yrs Female 1963	86	0.802	0.749
14-15-16 yrs Female 1973	157	0.522	0.748
12 and 15 yrs Female 1988	121		
12 to 15 yrs Female 1989(L)	63		
Overall ages and sexes combined 1963	189	0.841	0.781
" " 1973	309	0.560	0.846
" " 1988	208	1.226	0.869
" " 1989(L)	120	1.4750	0.7882

Table (IV-41)					
Reverse Overjet (-H)					
Age			Group Size	$\bar{x}$	s.d.
14-15-16 yrs	Male	1963	103	0.039	0.239
14-15-16 yrs	Male	1973	152	0.053	0.224
12 and 15 yrs	Male	1988	87		
12 to 15 yrs	Male	1989(L)	57		
14-15-16 yrs	Female	1963	86	0.035	0.185
14-15-16 yrs	Female	1973	157	0.076	0.29
12 and 15 yrs	Female	1988	121		
12 to 15 yrs	Female	1989(L)	63		
Overall ages and		1963	189	0.037	0.212
sexes combined		1973	309	0.064	0.257
"	"	1988	208	0.039	0.275
"	"	1989(L)	120	0.000	0.000

Table (IV-42)					
Combined M and D Deviations of Buccal Anteroposterior Relationships (RHS) (BAP _r)					
Age			Group Size	$\bar{x}$	s.d.
14-15-16 yrs	Male	1963	103	1.194	1.006
14-15-16 yrs	Male	1973	152	0.921	1.039
12 and 15 yrs	Male	1988	87	0.7931	0.9167
12 to 15 yrs	Male	1989(L)	57	0.7018	0.9442
14-15-16 yrs	Female	1963	86	1.128	1.009
14-15-16 yrs	Female	1973	157	0.809	0.914
12 and 15 yrs	Female	1988	121	0.9339	1.0858
12 to 15 yrs	Female	1989(L)	63	0.6190	0.9233
Overall ages and		1963	189	1.164	1.007) pooled
sexes combined		1973	309	0.864	0.977) s.d. =
"	"	1988	208	0.8750	1.018) .99
"	"	1989(L)	120	0.658	0.930)

Table (IV-43)					
Combined M and D Deviations of Buccal Anteroposterior Relationships (LHS) (BAP _L)					
Age			Group Size	$\bar{x}$	s.d.
14-15-16 yrs	Male	1963	103	0.990	1.089
14-15-16 yrs	Male	1973	152	0.921	1.010
12 and 15 yrs	Male	1988	87	0.7701	0.9487
12 to 15 yrs	Male	1989(L)	57	0.7018	0.9056
14-15-16 yrs	Female	1963	86	1.093	1.102
14-15-16 yrs	Female	1973	157	0.701	0.895
12 and 15 yrs	Female	1988	121	0.7851	1.0017
12 to 15 yrs	Female	1989(L)	63	0.4603	0.7793
Overall ages and		1963	189	1.036	1.095
sexes combined		1973	309	0.809	0.952
"	"	1988	208	0.779	0.978
"	"	1989(L)	120	0.575	0.847

Table (IV-44)					
Distal Buccal Anteroposterior Relationships of the RHS (BAP _R -D)					
Age			Group Size	$\bar{x}$	s.d.
14-15-16 yrs	Male	1963	103	0.874	0.813
14-15-16 yrs	Male	1973	152	0.599	0.944
12 and 15 yrs	Male	1988	87		
12 to 15 yrs	Male	1989(L)	57		
14-15-16 yrs	Female	1963	86	0.767	1.070
14-15-16 yrs	Female	1973	157	0.459	0.836
12 and 15 yrs	Female	1988	121		
12 to 15 yrs	Female	1989(L)	63		
Overall ages and		1963	189	0.0205	0.941
sexes combined		1973	309	0.529	0.89
"	"	1988	208	1.7952	0.7450
"	"	1989(L)	120	1.722	0.815

Table (IV-45)					
Distal Buccal Anteroposterior Relationship of the LHS (BAP _L -D)					
Age			Group Size	$\bar{x}$	s.d.
14-15-16 yrs	Male	1963	103	0.621	1.104
14-15-16 yrs	Male	1973	152	0.632	1.011
12 and 15 yrs	Male	1988	87		
12 to 15 yrs	Male	1989(L)	57		
14-15-16 yrs	Female	1963	86	0.756	1.147
14-15-16 yrs	Female	1973	157	0.369	0.728
12 and 15 yrs	Female	1988	121		
12 to 15 yrs	Female	1989(L)	63		
Overall ages and		1963	189	0.690	1.126
sexes combined		1973	309	0.500	0.87
"	"	1988	208	1.7606	0.8188
"	"	1989(L)	120	1.769	0.863

Table (IV-46)					
Mesial Buccal Anteroposterior relationships of the RHS (BAP _r M)					
Age			Group Size	$\bar{x}$	s.d.
14-15-16 yrs	Male	1963	103	0.301	0.654
14-15-16 yrs	Male	1973	152	0.270	0.587
12 and 15 yrs	Male	1988	87		
12 to 15 yrs	Male	1989(L)	57		
14-15-16 yrs	Female	1963	86	0.361	0.658
14-15-16 yrs	Female	1973	157	0.350	0.678
12 and 15 yrs	Female	1988	121		
12 to 15 yrs	Female	1989(L)	63		
Overall ages and		1963	189	0.331	0.656
sexes combined		1973	309	0.310	0.632
"	"	1988	208	1.5	0.740
"	"	1989(L)	120	1.308	0.480

Table (IV-47)

Mesial Buccal anteroposterior relationships of the LHS
(BAPL-N)

Age	Group Size	$\bar{x}$	s.d.
14-15-16 yrs Male 1963	103	0.369	0.657
14-15-16 yrs Male 1973	152	0.29	0.605
12 and 15 yrs Male 1988	87		
12 to 15 yrs Male 1989(L)	57		
14-15-16 yrs Female 1963	86	0.337	0.644
14-15-16 yrs Female 1973	157	0.331	0.720
12 and 15 yrs Female 1988	121		
12 to 15 yrs Female 1989(L)	63		
Overall ages and sexes combined 1963	189	0.353	0.650
" " 1973	309	0.310	0.662
" " 1988	208	1.321	0.548
" " 1989(L)	120	1.0455	0.2132

Table (IV-48)

Posterior Crossbite - RHS: (CR_R)

Age	Group Size	$\bar{x}$	s.d.
14-15-16 yrs Male 1963	103	0.243	0.602
14-15-16 yrs Male 1973	152	0.092	0.388
12 and 15 yrs Male 1988	87	0.06897	0.33387
12 to 15 yrs Male 1989(L)	57	0.07018	0.31958
14-15-16 yrs Female 1963	86	0.163	0.482
14-15-16 yrs Female 1973	157	0.147	0.464
12 and 15 yrs Female 1988	121	0.1157	0.36946
12 to 15 yrs Female 1989(L)	63	0.09524	0.42934
Overall ages and sexes combined 1963	189	0.201	0.542) pooled
" " 1973	309	0.107	0.426) s.d. =
" " 1988	208	0.096	0.355) 0.338
" " 1989(L)	120	0.083	0.38)

Table (IV-49)					
Posterior Crossbite - LHS : (CR _L)					
Age			Group Size	$\bar{x}$	s.d.
14-15-16 yrs	Male	1963	103	0.282	0.633
14-15-16 yrs	Male	1973	152	0.105	0.384
12 and 15 yrs	Male	1988	87	0.06897	0.33387
12 to 15 yrs	Male	1989(L)	57	0.01754	0.13245
14-15-16 yrs	Female	1963	86	0.105	0.377
14-15-16 yrs	Female	1973	157	0.108	0.417
12 and 15 yrs	Female	1988	121	0.19008	0.50521
12 to 15 yrs	Female	1989(L)	63	0.17460	0.52486
Overall ages and sexes 1963			189	0.207	0.505
sexes combined					
		1973	309	0.120	0.40
		1988	208	0.139	0.445
		1989(L)	120	0.10	0.3975

Table (IV-50)					
Godfrey Index Total (GI Total): (CR _L)					
Age			Group Size	$\bar{x}$	s.d.
14-15-16 yrs	Male	1963	103	5.49	3.091
14-15-16 yrs	Male	1973	152	4.428	2.544
12 and 15 yrs	Male	1988	87	4.6092	2.8687
12 to 15 yrs	Male	1989(L)	57	5.1667	2.4410
14-15-16 yrs	Female	1963	86	5.099	2.960
14-15-16 yrs	Female	1973	157	3.924	2.924
12 and 15 yrs	Female	1988	121	5.1322	3.0302
12 to 15 yrs	Female	1989(L)	63	4.6190	2.3958
Overall ages and sexes 1963			189	5.30	3.025) pooled
sexes combined					) s.d.
		1973	309	4.18	2.734) = 8.03
		1988	208	4.91	2.97)
		1989(L)	120	4.88	2.423)

Statistical Analysis of the Godfrey Index

TAMWORTH 1988

INTRAGROUP - ANOVA

Lower Anterior Crowding Ac

ANOVA revealed no significant age or sex effect.

Mandibular Posterior Crowding (LHS) (PCL)

No significant effect was found.

Mandibular Posterior Crowding (RHS) (PCR)

No significant effect was found.

Overbite (+V, -V combined)

ANOVA revealed a statistically significant age effect at $p < 0.006$.

Comment: There was a statistically significant decrease in overbite scores from 12 years to 15 years.

Overjet (+H, -H combined)

a) ANOVA revealed a sex effect which was statistically significant at $p < 0.016$.

b) Also revealed was a statistically significant age effect at $p < 0.035$.

Comment:

a) Females had a significantly higher mean overjet score than males in *Tamworth* in 1988.

b) Overjet scores decreased significantly from 12 years to 15 years in *Tamworth* in 1988. This was especially marked in females.

Buccal Anteroposterior Relationship deviation (RHS) (BAP_R)

No significant effect was found.

Buccal Anteroposterior Relationship Deviation (LHS) (BAP_L)

No significant affect was found.

Posterior Crossbite (LHS) (CR_L)

ANOVA found a sex effect which was statistically significant at $p < 0.046$.

Comment: The sex effect reveals that females had a higher average posterior crossbite score than males in *Tamworth* in 1988.

Posterior Crossbite (RHS) (CR_R)

No significant effect was found, however, for crossbite on this side of the mouth.

Godfrey Index Total (GI total)

No significant effect was found here for an age or sex effect intragroup.

LISMORE 1989

INTRAGROUP - ANOVA

Lower Anterior Crowding (Ac)

No significant age or sex effect was found.

Mandibular Posterior Crowding (LHS) (PC_L)

An age effect was found which was statistically significant at $p < 0.058$.

Comment: There was a significant decrease in posterior crowding scores with increasing age in *Lismore '89* for the sample

group. $p < 0.058$ is a higher probability than usually accepted, $p < 0.05$ is the most accepted level for validity.

Mandibular Posterior Crowding (RHS) (PC_R)

However, no significant effect was found for this trait on this side of the mouth.

Overbite (+V, -V)

No significant effect was found.

Overjet (+H, -H)

A statistically significant age effect was found intragroup. This was significant at a level of $p < 0.013$.

Comment: There was a significant decrease in overjet scores with increasing age of the subject for the *Lismore 1989* sample.

Buccal Anteroposterior Relationship Deviation (RHS) (BAP_R)

No significant age or sex effect was found for this trait.

Buccal Anteroposterior Relationship Deviation (LHS) (BAP_L)

A statistically significant sex effect was found at the $p < 0.06$ level.

Comment: Males had a significantly higher mean score for buccal sagittal relationship deviation than females in *Lismore* in 1989. Again, this level of probability is slightly higher than the generally accepted maximum of $p < 0.05$.

Posterior Crossbite (LHS) (CR_L)

A statistically significant sex effect was found at the $p < 0.047$.

Comment: Females had a significantly greater mean score for posterior crossbite compared with males in *Lismore* in 1989.

Posterior Crossbite (RHS) (CR_R)

On this side of the mouth, however, no significant effect was found for posterior crossbite.

Godfrey Index Total (GI total)

No significant age or sex effect was found for this category.

INTERGROUP ANALYSIS:

Tamworth 1963 vs Tamworth 1973 vs
Tamworth 1988 vs Lismore 1989

Statistical Analysis of these groups was undertaken using the calculated pooled variance for each trait.

To compare any 2 means for significance between them, they are first subtracted from each other.

They are then compared to the least significant difference value (l.s.d.), for a particular probability level.

For $p < 0.05$ the factor used is 1.96(*)

for $p < 0.01$ the factor is 2.57;

for $p < 0.001$ the factor is 3.29.

Values for l.s.d. were calculated for a $p < 0.05$ for the Intergroup Analysis used here.

$$\text{l.s.d.} = 1.96(*) \times \sqrt{\text{pooled variance} \times \left(\frac{1}{\text{group size}} + \frac{1}{\text{other group size}} \right)}$$

If the difference between the means for the particular groups is greater than the l.s.d, then the means are significantly different.

Ac Lower Anterior Crowding Combined ages/sex				
	Tamworth '63	Tamworth '73	Tamworth '88	Lismore '89
$\bar{x}$	0.524	0.596	0.716	0.867

pooled variance = 0.394

All measures were significantly different at the $p < 0.05$ level.

Comment: Significant differences in lower anterior crowding existed between all groups. *Lismore 1989* had the greatest mean crowding score, followed in descending order of crowding, by *Tamworth 1988*, *Tamworth 1973* and *Tamworth 1963*.

PCl Posterior Crowding LHS Combined ages/sexes				
	Tamworth '63	Tamworth '73	Tamworth '88	Lismore '89
$\bar{x}$	0.2000	0.180	0.039	0.121

pooled variance = 0.071

a) There was no statistically significant difference between the scores for *Tamworth 1963* vs *Tamworth 1973*.

b) There was a statistically significant difference between *Tamworth 1963, 1973* vs *Tamworth 1988*. This was at the $p < 0.05$ level.

c) There was a statistically significant difference between *Tamworth 1963, 1973* vs *Lismore 1989*. This was at the $p < 0.05$ level.

d) There was a statistically significant difference between *Tamworth 1988* vs *Lismore 1989*. This was at the $p < 0.05$ level.

Comment: No statistically significant difference existed between the posterior crowding on the LHS for *Tamworth 1963* and *1973*. However, the *Tamworth 1988* group had significantly less posterior crowding on the LHS than the *1963* and *1973* *Tamworth* groups. *Lismore 1989* had significantly more

posterior crowding (LHS) than *Tamworth 1988*. *Lismore 1989*, however, had significantly less posterior crowding (LHS) than either *Tamworth 1963* or *1973*.

PC _R Posterior Crowding RHS Combined ages/sexes				
	Tamworth '63	Tamworth '73	Tamworth '88	Lismore '89
$\bar{x}$	0.169	0.160	0.067	0.058

pooled variance = 0.068

- a) There was no statistically significant difference between the scores for *Tamworth 1963* vs *Tamworth 1973*.
- b) There was no statistically significant difference between the score for *Tamworth 1988* vs *Lismore 1989*.
- c) However, there was a statistically significant difference between *Tamworth 1963, 1973* vs *Tamworth 1988, Lismore 1989*. This was at the $p<0.05$ level.

Comment: There was no statistically significant difference between posterior crowding (RHS) between *Tamworth 1963* and *Tamworth 1973*. Neither was there a statistically significant difference between posterior crowding (RHS) scores for *Tamworth 1988* and *Lismore 1989*.

However, both *Tamworth 1988* and *Lismore 1989* had significantly less posterior crowding (RHS) than *Tamworth 1963* and *1973*.

+V Overbite Combined ages/sexes				
	Tamworth '63	Tamworth '73	Tamworth '88	Lismore '89
$\bar{x}$	0.958	0.582	0.880	0.8667

pooled variance = 0.720

a) There was no statistically significant difference between +V mean scores for *Tamworth 1963, 1988* and *Lismore 1989*, ($p < 0.05$).

b) There is however, a statistically significant difference between +V scores for *Tamworth 1973* vs *Tamworth 1963, 1988* and *Lismore 1989*, ($p < 0.05$).

Comments: Overbite scores decreased significantly from 1963 to 1973. There was then a significant increase in overbite from 1973 to 1988. The 1988 level of overbite was not significantly different to the 1963 level. *Lismore 1989* and *Tamworth 1988* had very similar overbite scores.

-V Openbite Combined ages/sexes				
	Tamworth '63	Tamworth '73	Tamworth '88	Lismore '89
$\bar{x}$	0.100	0.147	0.062	0.075

pooled variance = 0.139

a) There was no statistically significant difference between -V scores for any of the groups, ($p < 0.05$).

b) There was no significant difference between -V scores for the contemporary groups *Tamworth 1988* and *Lismore 1989*.

Comments: -V scores increased from 1963 to 1973, the decreased from 1973 to 1988. the 1988 level was lower than the 1963 level. As noted, none of these changes were statistically significant.

+H Overjet Combined ages/sexes				
	Tamworth '63	Tamworth '73	Tamworth '88	Lismore '89
$\bar{x}$	0.841	0.560	1.226	1.475

pooled variance = 0.585

- a) There was a statistically significant difference between all groups at p<0.05.
- b) +H was highest in *Lismore 1989* and lowest in *Tamworth* in *1973.*
- c) There was a significant decrease in overjet from 1963 to 1973. There was then a significant increase in overjet from 1973 to 1988. There was significantly more overjet recorded in 1988 than in 1963.
- d) Comparison of contemporary groups showed a significantly greater overjet for *Lismore 1989* than *Tamworth 1988.*

-H Reverse Overjet Combined ages/sex				
	Tamworth '63	Tamworth '73	Tamworth '88	Lismore '89
$\bar{x}$	0.037	0.064	0.039	0.000

pooled variance = 0.064

- a) There was no statistically significant difference between any of the *Tamworth* groups for -H scores. This was at the $p < 0.05$ level.
- b) All *Tamworth* groups had significantly more -H (reverse overjet) than *Lismore* which scored zero.

BAP _R Buccal segment anteroposterior relationship deviation (RHS) Combined ages/sexes				
	Tamworth '63	Tamworth '73	Tamworth '88	Lismore '89
$\bar{x}$	1.164	0.864	0.875	0.658

pooled variance = 0.981

- a) There was no statistically significant difference between *Tamworth 1973* vs *Tamworth 1988*.
- b) There was a statistically significant difference between *Tamworth 1973, 1988* vs *Tamworth 1963*. This was at the $p < 0.05$ level.
- c) There was a statistically significant difference between the scores for *Lismore 1989* vs *Tamworth 1963*. This was at the $p < 0.05$ level.
- d) There was a statistically significant difference between the scores for *Tamworth 1973, 1988* vs *Lismore 1989*. This was at the $p < 0.05$ level.

Comment: The deviations either mesially or distally from the ideal Class I position were not significantly different between *Tamworth 1973* and *1988*. However, *Tamworth 1963* had a

significantly greater deviation from the ideal Class I position than *Tamworth 1973* or *1988*. Likewise, the *Lismore 1989* group had significantly less deviation from the ideal Class I position than the *Tamworth 1963* group. Additionally, *Lismore 1989* had significantly less deviation from the ideal Class I position than did either *Tamworth 1973* or *1988*.

Therefore, in ascending order of deviation from the ideal Class I position, *Lismore 1989* is the least followed by *Tamworth 1973* and *1988* together, and highest deviation is *Tamworth 1963*.

BAP _L Buccal segment anteroposterior relationship deviations (LHS) Combined ages/sexes				
	Tamworth '63	Tamworth '73	Tamworth '88	Lismore '89
$\bar{x}$	1.036	0.809	0.779	0.575

pooled variance = 0.963

- a) There was no statistically significant difference between the scores for *Tamworth 1973* vs *Tamworth 1988*.
- b) There was a statistically significant difference between *Tamworth 1973, 1988* vs *Tamworth 1963*. This was at the $p < 0.05$ level.
- c) There was a statistically significant difference between *Lismore 1989* vs *Tamworth 1963*. This was at the $p < 0.05$ level.

- d) There was a statistically significant difference between the scores for *Tamworth 1973, 1988* vs *Lismore 1989*. This was at the $p < 0.05$ level.

Comment: This result follows the same pattern as BAP_R above. *Lismore 1989*, has the least deviation from ideal Class I either mesially or distally.

Tamworth 1973 and *1988* are not significantly different with respect to each other. However, *Tamworth 1973* and *1988* have significantly less buccal segment deviation than *Tamworth 1963*, but *Tamworth 1973* and *1988* have significantly more buccal segment deviation than *Lismore 1989*.

BAP _R -D Distal deviations of buccal sagittal relationships on (RHS) Combined ages/sexes				
	Tamworth '63	Tamworth '73	Tamworth '88	Lismore '89
$\bar{x}$	0.8205	0.529	1.7952	1.722

pooled variance = 0.733

- a) For BAP_R -D scores, there was no significant difference between *Lismore 1989* and *Tamworth 1988*, ($p < 0.05$).
- b) There was a significant decrease in mean BAP_R -D scores from *Tamworth 1963* to *1973*, ($p < 0.05$).
- c) There was then a markedly significant increase in BAP_R -D scores from *1973* to *1988* in *Tamworth*, and *Lismore 1989* was also markedly higher than *Tamworth 1973*, ($p < 0.05$).
- d) Both *Tamworth 1988* and *Lismore 1989* had significantly higher BAP_R -D scores than *Tamworth 1963*, ($p < 0.05$).

BAP _L -D Distal deviations of buccal sagittal relationships on (LHS) Combined ages/sexes				
	Tamworth '63	Tamworth '73	Tamworth '88	Lismore '89
$\bar{x}$	0.690	0.500	1.7606	1.769

pooled variance = 0.852

- a) The contemporary groups *Lismore 1989* and *Tamworth 1988* showed no significant difference for BAP_L-D scores.
- b) A similar phenomenon was noted as for BAP_R-D scores. There was a significant decrease in BAP_L D scores from *Tamworth 1963* to *Tamworth 1973*. There was then a highly significant increase in BAP_L-D scores from *1973* to *1988* (and also to *Lismore 1989*).
- c) *Lismore 1989* and *Tamworth 1988*, both showed significantly higher BAP_L-D scores than *Tamworth 1963*.

BAP _R -M Mesial deviations in buccal sagittal relationships on (RHS) Combined ages/sexes				
	Tamworth '63	Tamworth '73	Tamworth '88	Lismore '89
$\bar{x}$	0.331	0.31	1.5	1.308

pooled variance 0.419

- a) There was no significant difference between BAP_R-M scores for *Tamworth 1963* and *1973*.
- b) There was a very significant increase in BAP_R-M scores from *Tamworth 1963* and *1973*, to *Tamworth 1988*.

c) *Lismore 1989* had significantly higher BAP_R-M scores than *Tamworth 1963* and *1973*.

d) The BAP_R-M scores for *Tamworth 1988* were significantly higher than *Lismore 1989*.

BAP _L -M Mesial deviations in buccal sagittal relationships on (LHS) Combined ages/sexes				
	Tamworth '63	Tamworth '73	Tamworth '88	Lismore '89
$\bar{x}$	0.353	0.310	1.321	1.0455

pooled variance = 0.344

a) A similar pattern to BAP_R-M scores was seen here. There was no significant difference between BAP_L-M scores for *Tamworth 1963* and *1973*.

b) *Tamworth 1988* had significantly higher mean BAP_L-M score than either *Tamworth 1963* or *1973*.

c) *Tamworth 1988* also had a significantly higher mean BAP_L-M score than *Lismore 1989*.

d) *Lismore 1989* also had a significantly higher mean BAP_L-M score than either *Tamworth 1963* or *1973*.

CR _L Posterior Crossbite (LHS) Combined ages/sexes				
	Tamworth '63	Tamworth '73	Tamworth '88	Lismore '89
$\bar{x}$	0.207	0.10	0.139	0.120

pooled variance = 0.168

- a) There was no statistically significant difference between *Tamworth 1973* vs *Lismore 1989*.
- b) There was a statistically significant difference between the scores for *Tamworth 1973, 1988* and *Lismore 1989* vs *Tamworth 1963*. This was at the $p < 0.05$ level.
- c) There was a statistically significant difference between the scores for *Tamworth 1973, Lismore 1989* vs *Tamworth 1988*. This was at the $p < 0.05$ level.

Comment: *Tamworth 1963* had the highest score for posterior crossbite (LHS). While *Tamworth 1973* had the lowest score for posterior crossbite.

The posterior crossbite scores for *Tamworth 1973* and *Lismore 1989* were not significantly different. However, these scores were significantly less than *Tamworth 1963*.

Tamworth 1988 had significantly less posterior crossbite (LHS) than *Tamworth 1963*, but *Tamworth 1988* had significantly more posterior crossbite (LHS) than *Tamworth 1973* and *Lismore 1989*.

CR _R Posterior Crossbite (RHS) Combined ages/sexes				
	Tamworth '63	Tamworth '73	Tamworth '88	Lismore '89
$\bar{x}$	0.201	0.107	0.096	0.083

pooled variance = 0.151

- a) There was no statistically significant difference between *Tamworth 1973* vs *Tamworth 1988* vs *Lismore 1989*.

- b) There was a statistically significant difference between the scores for *Tamworth 1973, 1988 and Lismore 1989* vs *Tamworth 1963*. This was at the $p < 0.05$ level.

Comment: Posterior crossbite on the RHS was significantly greater in *Tamworth 1963* than in *Tamworth 1973, 1988 and Lismore 1989*.

There was no statistically significant difference for posterior crossbite (RHS) between *Tamworth 1973, 1988 and Lismore 1989*.

GI total - Godfrey Index Total score Combined ages/sexes				
	Tamworth '63	Tamworth '73	Tamworth '88	Lismore '89
$\bar{x}$	5.30	4.18	4.91	4.88

pooled variance = 8.03

- a) There was no statistically significant difference between *Tamworth 1963* vs *Tamworth 1988* vs *Lismore 1989*.

- b) There was a statistically significant difference between *Tamworth 1963, 1988 and Lismore 1989* vs *Tamworth 1973*. This was at the $p < 0.05$ level.

Comment: *Tamworth 1973* had the lowest mean GI total score. This was significantly less than *Tamworth 1963, 1988 and Lismore 1989*. Neither *Tamworth 1963, 1973* nor *Lismore 1989* had significantly different GI total scores.

ORTHODONTIC TREATMENT

It was mentioned earlier in this thesis, that those subjects who had previously had orthodontic intervention or were currently undergoing orthodontic treatment, were excluded from the assessments in the *Malocclusion Surveys*.

However, it was also the brief of this author to obtain figures for the number of children who had been treated or were presently being treated for orthodontic problems.

TAMWORTH 1988

The number examined in the *Malocclusion Survey* was 208. Forty-four were excluded from the examination because of orthodontic intervention past or present. Therefore, from a total group $(208 + 44) = 252$, 17.5% had or were having orthodontic treatment.

LISMORE 1989

The number examined in the *Malocclusion Survey* was 120. Forty were excluded from the examination because of orthodontic intervention, past or present. Therefore, from a total group $(120 + 40) = 160$, 25% had or were having orthodontic treatment.

This information was applicable to 12 and 15 year-old males and females for *Tamworth*, and 12-15 year old males and females in *Lismore*. The breakdown of males vs females having, or who had had treatment was females 62% vs. males 37.5%

Fourteen year-old girls were the highest single group,

comprising 32.5% of the sample. This information was only available for *Lismore*.

The *Surveys of 1963 and 1973* did not record the numbers with respect to orthodontic treatment so no comparisons can be made with the earlier *Surveys*.

The *Australian National Oral Health Survey, 1987-88*, results for *Victoria and New South Wales* show that for the 10-19 year-old age group, 14.6% of *Victorian* and 10.75% of the *N.S.W.* sample had been treated orthodontically or were undergoing treatment.

CORRELATION DATA

Previous *Malocclusion Survey Results* from *Tamworth* in 1963 and 1973 did not incorporate correlation data.

However, it was felt that interesting information could be gained from the *Rank Correlation Analysis of Tamworth '88* results and *Lismore '89* results. This might yield interesting information in comparing the 2 contemporary groups.

Lismore presented a special opportunity for this because not only were malocclusion scores made for each trait in each patient, but the individual D, M, F and DMFT scores were also available. It was felt that the direct correlation of D,M,F and DMFT scores with individual trait data may reveal interesting results.

It was felt correlations of the *Angle's Classification* with other traits would be invalid, however, all other trait measures could be correlated with each other validly since they measured a degree of malocclusion or deviation from ideal.

TAMWORTH

For *Tamworth*, 206 degrees of freedom are present, and for $p < 0.001$ the t value must be greater than 3.29 for statistical significance.

For $p < 0.01$ the t value is 1.96 at 206 d.f. and for $p < 0.05$ the t value is 2.58 at 206 d.f. Measures that correlations were attempted on were; the A_s (Subjective Assessment of Treatment-Need), and all Godfrey Index traits as well as the Godfrey Index total.

The correlation between lower anterior crowding and BAP_L had the lowest t value (1.96298), but this was still statistically significant

at $p < 0.05$. The correlation between lower anterior crowding and BAP_R was the other low correlation with a t value of 2.82317. This was still statistically significant at $p < 0.01$.

All other cross correlations were highly significant at $p < 0.001$.

The A_s correlated highly with the Godfrey Index total score. The A_s 's strongest correlations were with the Godfrey Index and right side posterior crossbites. The A_s weakest correlation was with overbite (V) and BAP_L .

The strongest correlations were found for

PC_L and CR_R	(t value 21.0031);
CR_L and CR_R	(t value 20.3925);
PC_L with PC_R	(t value 19.8153).
PC_L with CR_L	(t value 19.4922);
PC_R with CR_R	(t value 16.973)
and PC_R with CR_L	(t value 15.0769).

The GI total correlated most with the A_s , H (overjet); BAP_L and BAP_R . The GI total correlated least with A_c (lower anterior crowding). Note that all GI total correlations were statistically significant at $p < 0.001$.

LISMORE

For *Lismore* for 118 degrees of freedom

for a $p < 0.001$ the t value is	3.37
for a $p < 0.01$ the t value is	2.62
for a $p < 0.05$ the t value is	1.98

The A_s and all Godfrey Index traits and the GI total were all cross-correlated as for *Tamworth 1988*. However, the D,M,F and DMFT scores were also cross-related with the A_s and Godfrey Index traits and GI total.

This presented an ideal opportunity to assess the impact of caries traits and thus caries' effect on the various traits. Thus some of the effects of caries on malocclusion can be assessed.

Correlations with no statistical significance were:

A _s with V	(1.25598 t value;
A _s with DMFT	(1.93007 t value)
V with D	(1.11833 t value);
V with F	(1.54883) "
V with DMFT	(0.73614) "
H with D	(1.57199) "
H with F	(0.70269) "
H with DMFT	(-0.47386) "
GI total with F	(0.29110) "
GI total with DMFT	(-0.19151) "
GI TOTAL WITH D	(1.91696). "

Correlations significant at $p < 0.05$ were:

A _s with F	(2.02075 t value;
A _c with V	(2.07348) "
A _c with BAP _R	(2.08924) "
A _c with BAP _R	(2.08924) "
A _c with F	(2.47099) "
A _c with DMFT	(2.54662) "
V with BAP _R	(2.28270) "
BAP _R with F	(2.16430) "
BAP _R with DMFT	(2.11942) "
BAP _L with F	(1.99899) "
BAP _L with DMFT	(2.04577).

Correlations significant at $p < 0.01$ were:

A _c with H	(2.62434) t value
PC _L with BAP _R	(2.99405) t "
V with BAP _L	(3.23722) t "

All other correlations were highly significant at $p < 0.001$.

The A_s and GI correlations were highly significant at $p < 0.001$.

The A_s strongest correlations at $p < 0.001$ were with M (missing teeth); CR_L and CR_R (posterior crossbites). The weakest correlation of A_s was with V (overbite), F (filled permanent teeth) and DMFT. The Godfrey Index Total correlated strongest with BAP_L, BAP_R and H (overjet) at

$p < 0.001$. Its weakest correlation was with F and DMFT, where there was no significance.

Overall the strongest correlation at $p < 0.001$ was CR_R with CR_L (posterior crossbites on each side of the mouth). Further CR_R and CR_L were also highly correlated with M (missing permanent teeth). Also, as would be expected, F correlated highly with DMFT. F was the highest scoring subject of the DMFT index for *Lismore*.

For *Tamworth 1988* and *Lismore 1989* the following tables have not been included in this thesis but are available from the author:

- 1) *Rank Correlation data.*
- 2) *Age and sex vs Godfrey Index trait means and standard deviations.*

PART V

DISCUSSION

Part I of this thesis demonstrated how fluoride works both in systemic form and in topical form. As well, it may act together in the individual in both forms at once.

In water-fluoridated towns, it was shown that both systemic and topical effects occur due to fluoride. The systemic effect occurs by way of ingested fluoride being incorporated into the enamel of newly forming teeth. Additionally, fluoridated water in its passage past erupted teeth prior to swallowing may exert a topical effect. Another topical effect associated with fluoridated water ingestion is the secretion of fluoride in saliva and gingival crevice fluid. The fluoride levels are low in these two vehicles.

In non-water-fluoridated towns, it has been highlighted, that since the late 1970s, individuals in these towns receive both systemic and topical fluoride effects as well. The principal vehicle of delivery is fluoride toothpaste. However, foods processed in water-fluoridated areas, and vegetables and fruits grown in fluoride-rich soils are also implicated as vehicles for this fluoride experience in such areas.

In the late 1970s, fluoride toothpastes became the largest part of the toothpaste market (approximately 95%) (*Anderson et al, 1982; Latcham et al, 1982*). In fact this has been deduced to be the largest single factor responsible for the worldwide secular decline in dental caries which also occurred from the later 1970s (*F.D.I., 1985*).

It is now assumed that children living in non-fluoridated areas

receive a systemic effect from the use of fluoride toothpaste. This is due to the swallowing of large amounts of fluoride toothpaste up to 6 years of age. Immature swallowing patterns in young children, combined with very early use of fluoride toothpaste and brushing, and brushing instruction by the parent are mostly held responsible for this (*Mellberg et al, 1985; Ophaug and Singer, 1988*). As well, the usual topical effect of this fluoride toothpaste is also at play.

It is concluded that children in non-water-fluoridated towns now also receive a significant amount of systemic fluoride in the ideal age range for the pre-eruptive incorporation of fluoride into the enamel of a large number of teeth of the permanent and deciduous dentitions.

Recent research now clearly demonstrates that the post-eruptive fluoride experience of the teeth can often be more important with respect to caries resistance than the pre-eruptive fluoride experience of the tooth (*Beltran and Burt, 1988*).

In fact some studies previously mentioned that there was an insignificant difference between the enamel fluoride level of subjects in water-fluoridated and nearby non-water-fluoridated areas (*Aasenden, 1974; Schamschula et al, 1982; Mellberg et al, 1985*).

It was concluded that the post-eruptive fluoride experience of teeth in the fluoridated areas contributed to the difference in caries levels between such communities (*Beltran and Burt, 1988*).

It must also be remembered that the children in water-fluoridated towns from the late *1970s* were also receiving the effects of fluoride toothpaste. It is assumed that these children too, were also swallowing fluoride toothpaste and thus they would have received both

a water-fluoride systemic effect plus a fluoride toothpaste systemic effect. In addition, they would experience a water-fluoride topical effect as well as a fluoride toothpaste topical effect. However, *Mellberg and Ripa (1983)* concluded that there is not just a simple additive effect on DMFT, DMFS values. They feel this effect needs better researching.

In the present study, it seems reasonable to assume that *Tamworth* children from the late *1970s* have been receiving both the systemic effects of ingested fluoride from water and toothpaste vehicles, as well as a topical effect from these sources. Indeed, a perusal of the figures of DMFT and dmft for *Tamworth* demonstrates a further decrease in these scores from *1979*, over the post *1963* figures.

For *Lismore* children, no long-term figures are available but it is assumed that the children in the present study have also been experiencing systemic as well as topical effects from toothpaste fluoride. Remember, that *Lismore* is non-water-fluoridated.

Personal communication with *Craig (1990)* revealed that the amount of fluorosis in *Tamworth 1988* (1 ppmF) was minimal. Fluorosis is regarded as becoming obvious at 2ppm of fluoride in the water. Other researchers have recorded fluorosis at lower levels of fluoride (1-1.2 ppmF) [*Grahnen et al, 1974*]. So it would seem *Tamworth* children are not receiving a further 1ppmF from their ingestion of toothpaste fluoride (and from other sources) during enamel formation.

From this author's assessment of those examined by him in *Lismore 1989*, the fluorosis levels were minimal as well.

It also seems reasonable to assume that DMFT and dmft figures from *Tamworth* since the late *1970s* have been due to the combined effects of

water-fluoridation, fluoride toothpaste use, as well as lesser input from fluoride-containing foods (drinks, processed foods, fruits and vegetables) originating in fluoride areas.

Current DMFT figures for *Lismore 1989* are assumed to be due to input from fluoride toothpaste principally. It is assumed that had DMFT and dmft figures been available for *Lismore* they would have shown a large decrease in DMFT and dmft scores from the late *1970s*.

McKenna (1990) who was an orthodontist in *Lismore* from *1978 to 1985* states that caries levels showed a noticeable decrease over the time he worked there.

Thus, the large differences in caries-experience in primary and permanent teeth between fluoridated and non-fluoridated towns have declined markedly since the later *1970s*. However, in the present study, *Tamworth* children 12 and 15 years-old combined had a DMFT score 40% less than the 12-15 year-old *Lismore Group*. This is actually a wider gap than that mentioned by the *American Dental Association (1989)*. They mentioned an average difference of only 25% between DMF scores between fluoridated and non-fluoridated towns in the *U.S.A.*

A large number of authors (*Walther, 1966; Owen, 1971; Helm and Siersbaek-Nielsen, 1973; Miyamoto et al, 1976; Ronnerman and Thilander, 1977 and 1978; Pedersen et al, 1978; Dearing, 1981*) have shown that early loss of deciduous teeth, especially deciduous molars, and to a lesser extent canines, can result in an increase in malocclusion experience. Additionally, the early and later loss of permanent teeth can also lead to malocclusion (*Walther, 1966; McEwen and McHugh, 1970; Evans, 1988*).

It was previously concluded, that up to 50% of such resulting malocclusions could have been prevented by preventing tooth loss associated with dental caries.

However, *Jago (1974)* felt that there was no clear evidence to establish that malocclusion could be related to caries experience and its sequelae.

Several authors contend that caries by itself, or its untimely, or its poor restoration can allow drift of teeth, and diminish arch length and predispose to crowding (*Moyers, 1958; Graber, 1966; Salzmann, 1974*). However, *Powell (1985)* bases the "*minimal treatment*" technique of deciduous tooth caries control on the fact that statistically insignificant drift and crowding results in most cases where this interproximal caries slicing technique is employed. This flies somewhat in the face of the previous authors. However, several of Powell's cases had significant drift and thus may have had a malocclusion result eventually.

The amount of dental arch space loss in most cases appeared to be no more than accepted mixed dentition leeway space which is a type of "safety factor" in allowing for natural eruption of permanent canine over premolar teeth without dental arch crowding. Crowding which may be observed is then restricted to incisor segments which if already crowded would not be expected to improve (*Godfrey, 1990*).

Thus, while the caries and restoration aspect is questioned, there is no questioning of the possible sequelae of missing teeth.

It is thus concluded that the missing tooth (MT, mt) and teeth indicated for extraction (it) components of the DMF and dmf indices are the most important indicators of the likelihood, of a malocclusion

developing. It is felt that the loss of deciduous and permanent teeth in the samples studied, when the subjects were around the ages of 4-7 years-old, reflects the resultant malocclusion best.

12 and 15 year-old subjects in 1963 when 4-7 years of age, would have been in the early to mid 1950s. It is assumed that their dmft scores would have at least been greater than or equal to their 1963 dmft scores.

Peel (1973) commented that some subjects seen in *Tamworth 1973* by him had not experienced whole of life water-fluoride consumption. So, for the 12 and 15 year-olds seen in 1973, there was no fluoride ingestion during the first 2 years of the 12 year-olds lives. For the 15 year-olds, there was no systemic fluoride for the first 5 years.

Findings presented in PART I showed that despite lack of whole of life consumption of fluoride, and thus, lack of maximum opportunity for pre-eruptive enamel incorporation of fluoride, there are still the very important and effective post-eruptive mechanisms of fluoride (*Mellberg and Ripa, 1983; Beltran and Burt, 1988*).

Naturally, those examined in 1988 were lifelong consumers of water fluoride, and in addition, had experienced the superimposed effects of fluoride toothpaste, as previously outlined.

Closer examination of the missing deciduous and missing permanent tooth components of the dmft and DMFT scores as they have changed from 1963 - 1988 reveals the dramatic reduction obvious in the early loss of deciduous teeth especially. For instance:

For 6 year-olds: (mt - missing deciduous teeth due to caries)

- There was an approx. 60% decrease in mt between 1963 and 1973

- There was an approx. 25% decrease in mt between 1973 and 1979

* - There was an approx. 90% decrease in mt between 1979 and 1988

For 5 year-olds:

- There was an approx. 75% decrease in mt between 1963 - 1973.

- There was an approx. same mt value 1973 - 1979.

* - There was an approx. 75% decrease in mt between 1979 and 1988.

* - These figures are probably attributable to the late 1970s secular decline in caries due to widespread fluoride toothpaste usage.

It is obvious that large decreases in early losses of deciduous teeth occurred between 1963 and 1973. These decreases continued to 1988.

Examining permanent tooth loss shows, that in the 4-7 years of age range, permanent tooth loss was minimal from 1963 - 1988. However, for 12 year-olds, 35.5% of children had had one or more permanent teeth extracted due to caries in 1963. By 1973 this had dropped to 11.7%. This was a reduction of approximately 66%. For 15 year-olds in 1963, 49.2% had had one or more permanent teeth extracted due to caries. By 1973 this figure had dropped to 22.8%. This was a reduction of over 50% in that time.

DISCUSSION OF THE 1963-1988 TAMWORTH AND 1989 LISMORE MALOCCLUSION SURVEY RESULTS

As just mentioned much of the understanding of the following results is based on the premise that early loss of deciduous and/or permanent teeth has contributed to many of the malocclusions recorded below.

Discussion of Results of the Angle's Classification

For males, there was no significant difference in the distribution of the Angle's Classes between 1973 and 1988. However comparing males in 1988 with those in 1963 it was found that there were significantly more normal and Class III occlusions in 1988.

For females, there were more Class II Division 2 occlusions recorded in 1988 than in 1973. This is related mostly to the gene pool of the local area. There are many families of Scottish and Irish backgrounds in the Tamworth area. Luffingham and Campbell (1974) report a much higher incidence of Class III malocclusions in these groups in the United Kingdom (see Appendix). The fact that these Class II Div. 2 malocclusions became more frequent with increasing age may relate to the effect of mandibular growth due to an underlying skeletal Class III sagittal relationship, found in many Class II Div. 2 malocclusions. (Moss, A.S.O. Lecture, Sydney, 1984).

This explanation is given more credence when comparisons of Tamworth 1963 and 1988 are made. Here the effects of mandibular growth may

explain some results. In 1988 there were more *normal* occlusions in older age groups. As well, there were more Class II Div. 2 and Class III malocclusions with increasing age. As well, there were more Class II Div 1 malocclusions in 1988, but these decreased with age. Again, the effect of mandibular growth may tend to reduce this malocclusion type with age (*Proffit, 1986, Knutson, 1955*). At 12 years, many girls will be early or prepubertal with much potential mandibular growth. By 15 years, most growth has been expressed (*Proffit, 1986*).

Overall group comparisons between 1963 - 1988 show an increasing number of *normal* occlusions with increasing age. Additionally, there were significantly more *normal* occlusions in 1973 and 1988 compared with 1963. As well, there were more Class III malocclusions in 1988 and 1973 compared with 1963. While *Tamworth 1988* had more Class II Division 2 malocclusions than *Tamworth 1973* (principally due to the female group).

Increasing numbers of *normal* occlusions with age again reflects mandibular growth, as do increasing numbers of Class II Div. 2 malocclusions in 15 year-old females. However, the actual change in the proportions of the Class III and Class II Div. 2 malocclusions between 1963 and 1988 can't be explained with certainty. Reduced mesial drift of upper first permanent molars following decreased early deciduous molar loss may explain the increase in Class III malocclusions in recordings..

Previously, these Class III malocclusions may have been disguised by mesial permanent molar drift allowing a Class I or Class II molar relationship. They would then have qualified as Class I or Class II malocclusions.

The present writer feels that in many circumstances the Angle's Classification reflects the underlying skeletal pattern more than dental traits such as crowding, crossbites and so on. Thus, an increasing Class III skeletal change may be occurring, or may be being unmasked in the population. *Salzmann and Ast (1955)* and *Cooper and Ludwig (1965)* found fluoride had no effect on facial dimensions. Yet, there are more normal occlusions too. These may be explained in part by dental improvements which may have fluoride input. Analysis shows the increase in *normal* occlusions has been at the expense of Class I malocclusion.

Walther (1966) believes early loss of deciduous canines may allow retroclination of anterior teeth favouring a Class II Div. 2 type incisor pattern. Additionally, any forward drift of upper first permanent molars and deciduous molars may allow a Class II molar relationship to develop (*Walther, 1966, Owen, 1971*) with maxillary crowding as well, in the above example. Thus it is possible to see how the prevention of early deciduous canine loss (by the effect of fluoride) can contribute to less malocclusion and more *normal* occlusions. This would be especially at the expense of Class I malocclusions.

The use of the contemporary control, *Lismore 1989*, highlights gene pool differences. The *Lismore* area has a more mixed Western European and Southern Mediterranean population. *Tamworth 1988* had significantly more *Class III and Class II Div. 2* malocclusions than *Lismore*. However, *Lismore* had significantly more Class I malocclusions.

Additionally, *Lismore* had significantly more *normal* occlusions than

Tamworth 1963, but there was no significant difference between *Lismore 1989 and Tamworth 1973 and 1988* with respect to *normal* occlusions.

Having recorded individual D, M, F and DMFT scores for subjects examined in the *Lismore Malocclusion Survey*, the mean scores for each of these indices can be related to each of the Angle's Classes for *Lismore* (see Fig. IV-).

The highest mean D score was in Class III, while the lowest was in *normal* occlusion. No statistical analysis for significance was carried out here, but one could perhaps use this as support for undertaking orthodontic treatment to reduce decay incidence. On the other hand, one might argue that the low D score was responsible for normal occlusion.

The highest mean M score was for Class I malocclusion followed by Class II Div. 1. The lowest scores were 0 in the other 3 categories. Perhaps this finding could be used to support the earlier contention that missing teeth contribute to malocclusion. However, it should be borne in mind that the actual number of subjects with M scores >0 were 3 out of 120 subjects. Thus any occlusions with M>0 are unfairly weighted by this score.

One could enter into a "*chicken and egg*" debate as to whether the malocclusion contributed to the caries and thus the tooth loss, or whether the tooth loss precipitated the malocclusion.

The mean F score was highest in Class I malocclusions and lowest in Class II Div. 2. With Class I malocclusions being most common it would be expected that they would naturally have the greatest number of restorations.

Likewise the DMFT score was highest in Class I malocclusion and lowest in Class II Div. 2.

An examination of mean scores recorded for each Angle's Class is presented below for *Tamworth 1988* and *Lismore 1989*.

a) As (N = 0, - = 1, + = 2, ++ = 3)

As should be expected, the *Normal* occlusion category scored a mean of 0. This equates to N in the As.

In *Lismore*, Class II Div. 2 had the highest mean of 2.3, while in *Tamworth*, Class II Div. 2 and Class III both approximated a mean of 2.5. These, therefore, approximate to category +⁽²⁾ to ++⁽³⁾.

b) Lower anterior crowding (Ac)

In *Tamworth*, Class III malocclusion showed the largest mean score of 0.83, while in *Lismore*, Class III was also the class with the highest mean score for lower anterior crowding. This score was 1.33 which is significantly greater than for *Tamworth*.

c) Posterior crowding in the mandible (PC_R and PC_L)

Only *Normal* occlusion and Class II Div. 2 showed no posterior crowding in *Lismore*.

PC_L scores for *Lismore* were highest in Class III, closely followed by Class I then Class II Div. 1. In *Tamworth* the highest PC_R score was in Class II Div. 1.

d) Overbite (V [+V, -V])

The greatest overbite in *Lismore* was in Class II Div. 2, whereas anterior openbite was only found in Class III.

In *Tamworth*, the greatest overbite was found in Class II Div.1., closely followed by Class II Div. 2. Again, anterior openbite was only found in Class III. This was of the same order as *Lismore*.

e) Overjet (H [+H,-H])

A mean score of 0 was found only for *Tamworth* Class III. The greatest overjet in *Tamworth* was naturally in Class II Div. 1. However, for *Lismore* Class II Div. 2 scored the highest mean overjet, closely followed by Class II Div. 1. This is an unexpected finding since Class II Div. 1 is normally accepted as having a large overjet. Class II Div. 2 is normally expected to have minimal overjet. However, this author feels that Class II Div. 2 malocclusions may still be classified as such with large overjets. The essence of this classification lies in the classical incisor angulations combined with a greater than $\frac{1}{2}$ unit distocclusion. Could this be then taken to mean that Class II Div. 2 malocclusions in *Lismore* had a greater skeletal II sagittal input. Whereas, it has been inferred earlier that Class II Div. 2 is increasing in *Tamworth* along with Class III. It is speculated that these Class II Div. 2s may have underlying skeletal III bases. The mean overjet score for *Lismore* Class II Div. 2 is 2.3 while for *Tamworth* is 1.4 approx.

f) Buccal segment anteroposterior deviations (BAP_R, BAP_L)

In *Lismore* Class II Div. 2 had the greatest buccal occlusion deviation mean score. This is closely followed by Class II Div. 1.

In *Tamworth*, BAP_R shows the greatest deviation mean score for Class II Div. 2. Closely followed bilaterally by Class II Div. 1 and Class III.

g) Posterior Crossbite (CR_L , CR_R)

As might be expected, Class III had the highest mean score in *Lismore* and *Tamworth*.

In *Lismore, normal*, Class II Div. 1 and Class II Div. 2 all showed zero means.

However, Class II Div. 1 in *Tamworth* showed a mean score about 45% less than Class III. *Normal* class in *Tamworth* was the only class with a zero mean.

h) Godfrey Index Total Score (GI total)

In *Lismore*, Class II Div. 2 had the highest mean score, followed by Class II Div. 1, Class III then Class I. *Normal* scored the lowest as should be expected.

In *Tamworth*, Class II Div. 1 had the highest mean score, followed by Class II Div. 2, Class III. There was then a larger gap to the Class I score than in *Lismore*. Again *Normal* class had the lowest mean. *Lismore Normal* class had a mean nearly twice that of *Tamworth Normal* class..

Class III had a mean score of approximately 6 in both towns. The highest *Lismore* mean score was 10 for Class II Div. 2, while for *Tamworth* it was 8.15 for Class II Div. 1.

Class II Div. 1 had approximately the same mean GI score in *Lismore* and *Tamworth*.

Only *Lismore* data was available for D, M, F and DMFT scores.

D - Decayed Permanent Teeth

There was no significant difference between 0 scores for *Normal*, Class I, Class II Div. 1 and Class II Div. 2. However, Class III had a significantly higher D mean score of 1.0. The other classes ranged from 0.5 to 0.7 mean scores for D.

It would be very difficult to assume a cause/effect relationship with respect to which is the precipitating factor, Class III malocclusion causing higher decay, or earlier decay experience (which may be indicated by the D score), causing the malocclusion.

M - Missing Permanent Teeth.

The *Normal* class had no missing teeth, and neither did Class II Div. 2 nor Class III. Class I had the higher M mean score. This was 50% higher than the M mean score for Class II Div. 1.

F - Filled Permanent Teeth

Class I malocclusion had the highest F mean score of 1.3 (approx.). *Normal* and Class II Div. 1 both had scores of a similar mean value of 0.93 and 0.95 respectively.

Class II Div. 2 scored the lowest F mean score at 0.33 while Class III fell midway between class II Div. 2, and *Normal* and Class II Div. 1, with a F mean score of 0.66.

Discussion of Subjectively Assessed Treatment-Need (As)

Results of this part of the Malocclusion Survey reveal:

- 1) In *Tamworth in 1988* significantly more ++ than + scores were registered compared with *Tamworth 1963 and 1973*.

The difference between ++ scores for *Tamworth 1963 vs. Tamworth 1973* was not significant. However, *Tamworth 1963* had significantly more + scores than *Tamworth 1973*.

- 2) In *Tamworth in 1988* significantly more ++ than + scores were recorded than in *Lismore 1989*.
- 3) In *Lismore 1989*, significantly more ++ than + scores were recorded than for *Tamworth 1963 and 1973*.
- 4) Significantly more N and - scores were recorded in *1973 and 1988 in Tamworth*, and in *1989 in Lismore*, compared with *Tamworth 1963*.
- 5) There was no significant difference for N,- over +,++ scores between *Tamworth 1973, Tamworth 1988 and Lismore 1989*.
- 6) 15 year-olds in *Lismore 1989* had significantly more N,- scores than +,++ scores.
- 7) 15 year-olds in *Lismore 1989 and Tamworth 1988* had significantly more N,- scores than the younger age group.

Points 1) and 2) above may be explained in 2 ways. Firstly, the present writer attributes the larger number of ++ over + scores as demonstrating his individual preferences. At the

time of the Tamworth 1988 Survey, the writer was a very zealous, first year postgraduate. He was heavily involved with diagnosis, treatment planning and mechanics of mostly complex cases in the University of Sydney Orthodontic Clinic.

At this stage of postgraduate training, one becomes somewhat 'blinkered' about the abilities of the general practitioner to handle anything but the simplest cases. This is especially reinforced when one comes straight from a general practice situation, feeling inadequate in one's ability to handle orthodontics skilfully.

Therefore, when such an assessment as the A_s calls for discrimination between G.P. level (+) malocclusions, over specialist level (++) malocclusions, the neophyte orthodontist feels that there is very little that the G.P. can handle. He thus allots a ++ rather than a + score in the vast majority of cases. This is a problem with Subjective Assessments and has been highlighted by Freer et al (1973) and Bowden and Davies (1975). There was no conflict, however, between allotting these + and ++ scores when compared with N and - scores. For example, there was no difficulty between - and + scoring. This agrees with Freer et al (1973) and Bowden and Davies (1975).

After initial perusal of the Tamworth 1988 results for A_s, the writer realised his feelings had obviously affected results.

For the Lismore 1989 Survey an intentional effort was made to be less rigid in personal criteria for + compared with ++ scoring. However, results between Tamworth 1988 and Lismore

1989 still show a significant difference in + compared with ++ scores. Peel (1973) also felt that his scoring in 1973 could not be taken as fully compatible with Godfrey's 1963 scoring since he had not standardised his scoring against Godfrey for the As before the 1973 Tamworth Malocclusion Survey. He had however, checked and found his interexaminer variability with respect to the Angle's Classification and Godfrey Index Traits, was acceptable.

In Part II of this thesis, a critique of the As was made. One conclusion reached there was that Subjective Assessments are acceptable (and some times preferred) if they are based on satisfying certain objective criteria. This writer believes that the As results would be more valid if such a "check list" were compiled to aid As assessments.

The second point to be made regarding ++ and + scoring is that it may actually reflect a change in *Normal* and mild occlusions Vs more severe occlusions. Angle's Classification results indicated more Class II Div. 2 and Class III malocclusions in *Tamworth 1988* compared with *Tamworth 1963, 1973 and Lismore 1989*. *Tamworth 1988's* ++ over + scoring was significantly higher than the other groups' scoring for ++ over +. It is assumed there has been a change in fluoride presence in *Tamworth* from the late 1970s, with the advent of fluoride toothpaste as the major seller. Therefore, have most preventable malocclusions (from a caries aspect) been reduced so that only major skeletal defects dominate the scoring of the ++ category? Many of the more genetic-based malocclusions

may now be dominating the ++ end of the A_s scale. Peel (1973) comments similarly.

Therefore, even though criteria were eased for + over ++ scores for *Lismore 1989*, the significantly greater scoring of ++ over + in *Lismore 1989* compared with *Tamworth 1963 and 1973* may again be explained by this domination of ++ scores by genetically-based malocclusions, especially with skeletal deviations.

The fact that *Lismore 1989* is significantly lower than *Tamworth 1988* for ++ scores over +, may be explained as before by racial and gene-pool differences.

Further interesting information is revealed in the means of each Godfrey Index Trait for each category of A_s (see Tables IV-)

- lower anterior crowding was highest in the ++ group and lowest in the N group. This would be expected, and held true for *Lismore 1989 and Tamworth 1988*. The *Lismore* group had higher mean scores for each category.
- posterior crowding on the right side clearly showed a higher prevalence in the + and ++ categories in *Lismore and Tamworth*. This held for the left side as well where the ++ category in *Lismore* had the highest mean crowding score. The N groups had 0 mean scores in *Lismore* but slightly greater than 0 in *Tamworth*.
- overbite was highest in *Tamworth* in the + group but -, + and ++ were significantly higher than the N group. For *Lismore*, overbite was highest in the + group and lowest in the N

group but the differences were not as marked. This could perhaps be related back to examiner attitudes. This examiner acknowledge that he is not overly concerned with overbite *per se*.

- + and ++ categories showed the highest mean overjet scores. There was little difference in the overjet means between -, +, ++ categories, this was especially so for *Lismore*. *Lismore's* N category overjet mean was more than double that of *Tamworth's*. There is a possibility here that this reflects the tendency for more class III malocclusions and Class II Div. 2 malocclusions to be present in *Tamworth* in 1988. It was previously suggested that *Lismore* may have more skeletal II than *Tamworth*.
- buccal segment anteroposterior deviations were much higher in the + and ++ categories in *Tamworth* and less so in *Lismore*. *Tamworth* had significantly greater BAP scores in the + and ++ categories than *Lismore*. It would be expected that marked molar relationship deviations would attract a higher treatment-need score than minimal deviations. This also reflects the underlying skeletal deviation often accompanying such buccal segment deviations. The Angle's Classification was earlier mentioned as reflecting to a large extent underlying skeletal problems.
- posterior crossbites showed the highest means in + and ++ categories in *Tamworth*, and in the ++ category in *Lismore*. The examiner could thus be said to be ranking crossbite in more severe treatment-need categories.

- In *Tamworth*, there was little difference between Total Godfrey Index scores between the + and ++ categories. This was not so in *Lismore* where the ++ category clearly showed the greatest mean score. *Tamworth's* N group had a mean score of 1.4 while this same group in *Lismore* had a mean score of 2.5.

For *Lismore 1989* the following are considered of special interest since individual D, M, F and DMFT scores were recorded for each subject. Therefore, caries and missing tooth information can be related to orthodontic treatment-need.

- For D scores, the N category had the lowest mean D score, and the highest mean D score was found in the ++ category. Thus, the greatest amount of orthodontic treatment-need was found in mouths with the greatest amount of caries experience in permanent teeth.
- Probably the most important measure is the M score. It has been stressed how important missing teeth are considered to be in the predisposition to malocclusion. The ++ category (orthodontist level of treatment-need) showed the highest mean M score. The other 3 categories had means of 0. Therefore, the malocclusion level with the greatest need of treatment was also the malocclusion level with the greatest score for missing permanent teeth. A direct cause and effect relationship cannot be claimed but can be speculated.

- For F, the highest F score was found in the + category, with the + category next but only just ahead of - and N categories.
- For DMFT, the highest scores were found in the +, and then ++ categories. Again a cause and effect relationship can only be speculated.

Discussion of points 4)-7) of the A_s results concerns N, - categories versus +, ++ categories. Since statistical breakdown showed that this is where the main difference between the other A_s results lie, then it was decided to deal with the discussion of these results in the next section where they are more appropriately handled. That is, in the Subjectively Assessed Treatment-Need subcategory.... Treatment vs No Treatment-Need, that is, N, -(no treatment-needed) vs. +, ++ (treatment-needed).

Discussion:
Treatment-Needed vs. No Treatment-Needed

No treatment-needed = combined N and - scores from As.

Treatment-needed = combined + and ++ scores from As.

No significant differences were found for N,- vs. +,++ distributions between *Tamworth 1973, 1988 and Lismore 1989*. However, all showed significantly greater proportions of N and - scores (no treatment-need) than *Tamworth 1963*.

From the above results, it is obvious that treatment-need levels for *Tamworth* have not changed significantly since 1973 despite increasing amounts of fluoride in the diet from ingested toothpaste and processed foods. From *1963 to 1973 in Tamworth*, however, there was a significant improvement in the numbers of subjects requiring treatment. *Peel 1973* felt that by deduction this could be attributed in the majority to the various effects of water-flouridation commenced in *1963*. Remember that fluoride toothpastes were a very small proportion of sales in *1973* and before (*F.D.I. Joint Working Group, 1985*).

However, inspection of the contemporary control group reveals no significant difference between *Tamworth 1973 - 1988 and Lismore 1989* with respect to treatment-need. Two possibilities exist, firstly, that despite not having had water-fluoridation, *Lismore* has always had significantly better malocclusion levels than *Tamworth*. This writer feels that this is unlikely. Secondly, *Lismore* probably had malocclusion levels similar to *Tamworth 1963* up until the *mid to late 1970s*.

Unfortunately, no *Lismore* figures on malocclusion are available *pre 1989*, but it is assumed that over the last 10 - 12 years, *Lismore* has

seen a marked reduction in malocclusion. This has brought *Lismore 1989* treatment-need to the level of *Tamworth 1973* and contemporary *Tamworth 1988*.

A personal communication with *McKenna (1990)* was an orthodontist in *Lismore* from *1978 to 1985*, revealed that he felt there was a noticeable (but not extreme) improvement in malocclusion prevalence that could be related to early loss of deciduous or permanent teeth.

Thus, despite the increased availability of fluoride both systemically and topically in *Tamworth* from the *late 1970s*, at the time of the fluoride toothpaste boom, there has not been a concomitant improvement of significance in treatment-need from *1973 - 1988 in Tamworth*.

Couple this with the fact that DMFT scores for *Lismore 1989* are 40% worse than *Tamworth 1988*, then one must assume that a certain threshold level of fluoride exposure exists which is enough to confer enough caries-protection to prevent most deciduous and permanent tooth loss through decay. Previous discussion stresses that tooth loss appears to be the major factor at work in predisposing to a malocclusion that is preventable.

At this point it is worth noting the Australian National Oral Health Survey figures for *New South Wales (1987 - 1988)* and *Victoria (1987 - 1988)*. The categories for "malocclusion treatment" are:

- no treatment-needed - this writer equates this to N
- doubtful treatment-need - this writer equates this to -
- treatment-needed - this writer equates this to +
- urgent treatment-needed - this writer equates this to ++

- previously treated-)
under treatment)

- these two categories will be
deleted at this point in
discussion but will be compared
later with similar information
obtained for *Lismore 1989 and*
Tamworth 1988. Peel (1973) and
Godfrey (1963) did not produce
figures for these categories.

Reworkings of "malocclusion treatment" percentage results from the
Australian National Oral Health Survey are presented below, along with
comparisons with *Tamworth 1963 - 1988 and Lismore 1988*. The N.S.W.
data obtained divides the relevant age groups into 10 - 14 year-olds
and 15-19 year-olds. This splits the *Tamworth and Lismore* groups.
Therefore they were combined to give an overall 10-19 year-old group.
This is the group with which *Tamworth and Lismore* comparisons will be
made.

TABLE (V-1)

A.N.O.H.S. 1987-88			TAMWORTH			LISMORE
	N.S.W.	VICTORIA	1963	1973	1988	1989
N	63%	62%	3%	15%	12%	12%
-	12%	14%	20%	34%	38%	40%
+	22%	23%	53%	28%	6%	15%
++	3%	1%	24%	23%	44%	33%

Combining these groups to suit the category under dicussion, i.e. N,-
(no treatment-needed) vs. +,++ (treatment-needed), the figures are
seen below in TABLE (V-2).

TABLE (V-2)

A.N.O.H.S. 1987-88			TANWORTH			LISMORE
	N.S.W.	VICTORIA	1963	1973	1988	1989
No Treatment Needed	75%	76%	23%	49%	50%	52%
Treatment-Needed	25%	24%	77%	51%	50%	48%

The comparison of the results above is only valid if:

- 1) it is accepted that the N,-, + and ++ scores equate to the particular groups mentioned in the A.N.O.H. Survey.
- 2) one accepts that there will be little inter-examiner variability between collected samples. The present writer has no way of telling if this is true. He suspects that the A.N.O.H.S. data are somewhat invalid by themselves since they come from separate states and no doubt different examiners performed the examinations. However, they are calibrated according to objective criteria. No mention is made of where the subjects came from geographically, or their racial backgrounds.

So from this point of view, there is probably more variation than results actually show for the A.N.O.H.S. Survey. This writer feels that Table (V-2) probably conveys safer information than TABLE (V-1) since the self-acknowledged examiner preference of this author with respect to + vs ++ scoring is negated by combining them in TABLE (V-2).

Examination of the results in TABLE (V-2) definitely shows a large

discrepancy between results for the contemporary groups. Therefore it is assumed that this author's comparison may be invalid due to the reasons just outlined above.

However, TABLE (V-2) quickly shows the massive improvement in those not requiring treatment between Tamworth in 1963 and later groups. This is an improvement of over 50% and has been attributed mainly to the effect of fluoride by both Peel (1973) and the present writer.

Results also show that 15 year-olds in 1963 were significantly less represented in the "No Treatment-Needed" group than they were in the Tamworth 1973, 1988 and Lismore 1989 groups. The interpretation this author puts on this result relies on the knowledge that DMFT scores (from Part I) demonstrate an increasing D, M, F and DMFT score with increasing subject age. Thus, in 1963, when caries was rife, the 15 year-olds would have had more chance of first molar extraction, and more chance for the orthodontic sequelae of caries-related extractions to be expressed. However, once fluoridation commenced 15 year-olds although they may not have received lifelong fluoride systemically received some topical protection as outlined in Part I. (Hardwick et al, 1982; Beltran and Burt, 1988). This is assumed to have been adequate to cross the threshold previously proposed, and place more of them in a "No Treatment-Needed" category by 1973.

Statistical analysis also revealed that in both Lismore 1989 and Tamworth 1988, there was an age effect. This meant that there was an increase in the proportion of N and - scores over + and ++ scores with increasing age. That is, 15 year-olds in both towns had less treatment-need than 12 year-olds.

This author believes that this reflects the impact of growth. Results

for the Angle's Classification showed more Class II Div. 2 and Class III malocclusions with age in *Tamworth 1988*. This was attributed to the effect of mandibular growth.

This would increase Class III malocclusions and maybe those Class II Div. 2 malocclusions with an underlying skeletal III base. Likewise, mandibular growth may reduce overjet and buccal segment distocclusions during the pubertal growth period. So, observation at 12 and 15 years of age may be quite appropriate to highlight this effect. As well, final adjustments of molar relationship and lower incisor crowding across the population may not be fully completed by 12 years of age if the leeway space is still present or deciduous molars are still present. Thus, 15 year-olds should demonstrate the end result of growth and leeway utilisation.

DISCUSSION OF RESULTS FOR THE GODFREY INDEX (GI)

Ac - Lower Anterior Crowding

There was an increasing trend for lower anterior crowding from *Tamworth 1963 to Tamworth 1973 to Tamworth 1988 to Lismore 1989*. The mean values were significantly different. Between each group at the $P < 0.05$ level.

The action of fluoride ingestion effecting a reduction in tooth size cannot be given any support from the results of lower incisor crowding, from *1963 to 1988*. Less crowding would be expected if this mechanism was at work.

The increasing amount of lower anterior crowding is most probably related to less early posterior deciduous tooth loss. With less posterior loss, anterior crowding is less able to be relieved by distal drift. Distal drift is the main mechanism at work in the mandibular arch (*Walther, 1966; Owen, 1971*).

This decreased early deciduous tooth loss is most likely related to the action of fluoride.

PC_L - Posterior Mandibular Crowding LHS.

There was no significant difference between PC_L scores for *Tamworth 1963 and 1973*. However, there was significantly less posterior crowding on the left side for the *Tamworth 1988* group when compared with either *Tamworth 1963 or 1973*.

Lismore 1989, as well, had significantly less posterior crowding on the left side compared with either *Tamworth 1963 or 1973*.

However, *Lismore* had significantly more posterior crowding on the left side than *Tamworth 1988*.

Whether the difference between *Lismore 1989* and *Tamworth 1988* is a gene pool difference, or linked to the higher fluoride intake level of *Tamworth* subjects, is unknown.

Additionally, why *Tamworth 1963 vs. 1973* is not significantly different, when the As and Angle's Classification assessments show significant differences between *1963 and 1973* cannot be explained by this writer?

It is assumed that the decrease in PC_L scores from *1973 to 1988* is due to less early loss of lower deciduous molars. With early loss, 6s drift forward, or more commonly lower anteriors fall back and reduce space for erupting premolars (*Walther, 1966; Owen, 1971*). The action of fluoride is assumed to have effected this result.

PC_R - Posterior Mandibular Crowding RHS

Statistical Analysis revealed that there was no significant difference between *Tamworth 1963 and 1973* groups for this trait.

Neither, was there a significant difference between *Tamworth 1988* and the contemporary control group *Lismore 1989*.

However, there was a significant difference (at the $P < 0.05$ level) between *Lismore, 1989, Tamworth 1988 vs. Tamworth 1963, 1973*. There was significantly less posterior crowding on the right hand side in both *Tamworth 1988 and Lismore 1989* than in *Tamworth 1963 and 1973*.

There was over 60% greater posterior crowding in *Tamworth 1963, 1973* than in *Lismore 1989 and Tamworth 1988*.

Again, despite significant improvements from 1963 to 1973 for the number of *Normal* scores for both the Angle's Classification and the Subjectively Assessed Treatment-Need Assessment, there was no significant difference for PC_R scores.

Meanwhile the two contemporary groups showed no intergroup difference for PC_R scores, but significantly less posterior crowding than the earlier groups (*Tamworth 1963, 1973*). This difference is assumed to be due to the effect of fluoride consumption preventing caries and thus ultimately, early deciduous tooth loss, especially loss of deciduous molars.

Openbite (-V)

Statistical Analysis revealed no significant differences between openbite scores for all the groups under study.

One might have expected more openbite in the 1988 *Tamworth Group* since the Angle's Classification results showed significantly more Class III malocclusions in 1973 and 1988 than for the 1963 group. Openbite tendency is considered part of the Class III malocclusion syndrome in many cases (*Proffit, 1986*). However, it may be that the Class III malocclusions in *Tamworth*, have underlying skeletal bases of a more forward growth rotation type. Thus there is a tendency for less openbite. This is possibly supported by the significant increase in Class II Div. 2 malocclusions also recorded. It was postulated earlier, that these may also have underlying Skeletal III sagittal patterns. Therefore if the two are related, the forward growth rotation Class III skeletal pattern may be more the dominant type since it is also associated with the deep-bite Class II Div. 2 malocclusion.

Overbite (+V)

Statistical analysis revealed no significant difference for overbite scores between *Tamworth 1963, 1988 and Lismore 1989*.

However these above groups all had significantly greater overbite scores than *Tamworth 1973*.

Why overbite may have decreased from *Tamworth 1963 to Tamworth 1973*, then increased significantly in *Tamworth 1988*, is unknown?

In *Tamworth 1963*, deep overbite may have been precipitated by early deciduous canine or molar loss (in particular) allowing anterior teeth to collapse lingually, deepening the bite. Presumably, the significant improvement between overbite scores in *1963 and 1973* is due to less loss of the deciduous teeth mentioned. Possibly it may even be due to less loss of 6s allowing posterior bite collapse (*Walther, 1966; McEwen and McHugh, 1970*).

However, why there has been a significant overbite increase again, from *1973 to 1988* is difficult to explain? The possibility of inter-examiner variability at work here is raised.

Contemporary groups *Lismore 1989 and Tamworth 1988* were not significantly different for overbite scores.

Reverse Overjet (-H)

Statistical Analysis revealed no significant difference for -H scores between any of the *Tamworth* groups (ie. *1963, 1973, 1988*). However, as has already been mentioned the Angle's Classification results showed significantly more Class III malocclusions in *1973 and 1988* in *Tamworth* compared with *Tamworth 1963*. Therefore, one might expect a greater reverse overjet mean score in *1973 and 1988*. In fact, the

Tamworth 1988 group had a lower mean score than the 1973 group, but as mentioned, there was no significant difference at the $p < 0.005$ level. The results of Ronnerman and Thilander (1978) indicate that early deciduous tooth loss may lead to a decreased sagittal length of the maxilla. In these cases, reverse overjet may have been higher in the 1963 group.

The other point to remember, too, is that it was suggested that these Class III cases in Tamworth 1988 may be predominantly forward growth rotation-types. Therefore, one might expect more reverse overjet in Tamworth 1988, if this were the case.

Lismore 1989 registered no reverse overjet subjects and therefore was significantly different from the Tamworth 1963, 1973 and 1988 groups. Gene pool and racial differences, as previously mentioned, are thought to account for this occurrence.

Overjet (+H)

Overjet decreased significantly from 1963 to 1973 in Tamworth. However, there was then a significant increase in overjet from 1973 to 1988. The 1988 level being also significantly greater than that of Tamworth 1963. Additionally, Lismore 1989, like Tamworth 1988, also had significantly more overjet than either Tamworth 1963 and 1973. Lismore as well, had significantly more overjet than Tamworth 1988. Mean overjet levels for Tamworth 1988 were over 50% higher than Tamworth 1963 and around 60% + higher than Tamworth 1973. A similar trend was found here as for overbite (+V) in the Tamworth groups. This was, that overbite significantly decreased from 1963 to 1973. There was a significant increase in overbite from 1973 to 1988. For overbite, this return of higher overbite scores after 1973 was to a level not

significantly different to the 1963 overbite level. However, for overjet, the 1988 level was significantly higher than the 1963 level.

This parallel relationship would seem to confirm that overbite and overjet are intimately related. This has been found in numerous studies (Haynes, 1974; Cordato, 1990).

This author has no explanation for the significant increase in overjet from 1973 to 1988 in Tamworth.

Buccal Segment Sagittal Relationship Deviations - BAP_R

There was a significant decrease in overall deviations from the ideal molar relationship from 1963 to 1973.

From these results, it is assumed that early deciduous tooth loss reduced from 1963 to 1973. Thus, undesirable drift of permanent molars and canines was reduced from 1963 to 1973. Therefore, molar and canine relationships were more likely to be closer to the ideal position.

BAP_R scores showed no significant change between 1973 and 1988. This also agrees with results for the Angle's Classification (except for Class II Div. 2 malocclusion); the Subjectively Assessed Treatment-Need; and the Treatment vs. No Treatment-Need categories.

Comparison of contemporary groups, *Lismore 1989 and Tamworth 1988* revealed that *Lismore* had significantly less deviation in buccal relationship (right side) than *Tamworth*. In the Angle's Classification *Lismore* had more Class I malocclusions which supports this. However, *Lismore* had a significantly higher mean overjet score than *Tamworth*.

From the above findings and earlier findings it would seem that

Lismore has a high percentage of increased overjet Class I malocclusions.

BAP_R Distocclusion

BAP_R Distocclusions decreased significantly from 1963 to 1973, then increased significantly from 1973 to 1988. The 1988 mean deviations were over 50% higher than the 1963 mean. This was statistically significant.

The present writer has no explanation for the marked increase in distal deviations from 1973 to 1988. It may be linked with overbite and overjet changes as well.

The explanation for the significant decrease in BAP_R D scores from 1963 to 1973 lies in the reduced number of maxillary deciduous molars lost. Early loss of these teeth allows mesial drift of the upper 6s into a more Class II molar relationship (*Walther, 1966; Owen, 1971*).

It is presumed that the action of fluoridated water has reduced maxillary deciduous molar loss between 1963 and 1973. An increase in deciduous molar loss has not caused the increased distocclusion since 1973. Missing teeth figures do not support this.

There was no s.s. difference between the BAP_R D scores for *Lismore 1989 and Tamworth 1988*.

BAP_R Mesiocclusion

A significant increase in mesiocclusion on the right side has occurred since 1973. *Tamworth 1963 and 1973* means show no s.s. difference. The 1988 mesiocclusion mean is approximately 5 times higher than the 1963 and 1973 means.

This author feels that this increase in mesiocclusion from 1973 to

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1988 again reflects a reduced loss of deciduous molars in the maxilla. In subjects with an underlying Skeletal III pattern, early maxillary deciduous molar loss may have led to forward drift of the 6s into a Class I or even Class II molar relationship. In 1988, with very low levels of missing deciduous teeth, the upper 6s now remain in their Class III relationship and, thus now register as mesiocclusions. Angle's Classification results from 1973 and 1988 also support this.

This mechanism again supports the action of fluoride in reducing early deciduous tooth loss. Being pedantic, one could say fluoride may actually create a Class III malocclusion.

Tamworth 1988 had a significantly higher mesiocclusion mean on the right side than Lismore 1989. This is attributed to racial and gene pool differences.

BAP_L - Buccal Segment Sagittal Relationships Deviations - Left Side

A similar pattern of buccal segment deviations to the right side was noted on the left side in Tamworth between 1963-1973-1988.

BAP_L scores reduced significantly from 1963 to 1973. There was no s.s. difference between BAP_L scores for 1973 and 1988 groups.

Lismore 1989 had significantly lower deviations from the ideal relationships than Tamworth 1988.

The same comments that were made for BAP_R apply here.

BAP_L Distocclusion

A similar pattern exists for distocclusions on the left side as for the right side. The same comments made for BAP_R Distocclusion apply here.

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Distocclusion means decreased significantly from *1963 to 1973*. There was then a marked and highly significant increase in distocclusion mean score from *1973 to 1988*. The *1988* mean was approximately 2.5 times greater than the *1963* mean. This difference was statistically significant.

The contemporary groups, *Lismore 1989 and Tamworth 1988* were not significantly different for left side distocclusion means.

BAP_L Mesiocclusion

Mesiocclusion scores on the left side mirror those of the right side. The same comments made about BAP_R mesiocclusion scores apply on this side.

There was no significant difference between *Tamworth 1963 and 1973*. However, there was a significant increase in left side mesiocclusion mean score from *1973 to 1988*. The *1988* mean is approximately 3.5 times higher than the *1963 and 1973* means. This difference is statistically significant.

Again, as for BAP_R mesiocclusions, *Tamworth 1988* had a significantly higher mean BAP_L score than *Lismore 1989*. One question arises when considering overall BAP and BAP distocclusion vs BAP mesiocclusions. Why did BAP, and BAP distocclusions reduce significantly between *1963 and 1973*, while BAP mesiocclusions did not show significant increases until *1988*? This author is unable to explain this phenomenon.

CrL - Posterior Crossbite - LHS

Statistical Analysis revealed a significant decrease in posterior crossbite scores from *Tamworth 1963 to 1973*. From *1973 to 1988* there was a slight increase in posterior crossbite (LHS) but this was not

statistically significant. However the *Tamworth 1988* crossbite scores were significantly lower than *Tamworth 1963*. *Lismore 1989* crossbite scores were lower than all *Tamworth* groups, but only significantly lower than *Tamworth 1963 and 1988*. The reason for a significant difference between contemporary groups may again lie in the racial/gene pool differences. *Tamworth* has a higher proportion of Class III Angle's malocclusions and it is to be expected that these subjects would be the main contributors to posterior crossbite (LHS) scoring.

Pedersen et al (1978) note that early loss of deciduous molars and/or first permanent molars, in the maxilla alone, or the maxilla and mandible can lead to an increase in posterior crossbites. The action of fluoride is felt to have been responsible for the significantly reduced level of posterior crossbites (bilaterally) from *1963 to 1973*.

CrR - Posterior Crossbite - RHS

Statistical analysis showed a significant decrease in CrR scores from *1963 to 1973 for Tamworth*. However, from *1973 through to 1988* there has been a further decrease in posterior crossbite (RHS) but this has not been statistically significant.

The contemporary groups *Lismore 1989 and Tamworth 1988* did not show any significant difference for posterior crossbite (RHS). *Lismore 1989* also showed significantly less crossbite than *Tamworth 1963*. In fact, *Lismore* showed the lowest CrR mean score.

Contemporary mean scores for CrR were approximately $\frac{1}{2}$ those of *Tamworth 1963*. Again, the reason for the (insignificant) difference between *Lismore 1989 and Tamworth 1988* may be due to the higher Class III Angle's scores for *Tamworth* as outlined for CrL.

Godfrey Index Total (GI total)

Statistical analysis showed that there was a significant decrease in mean GI scores from *Tamworth 1963* to *1973*. There was then a significant increase from the *1973* level to the *1988* level for the GI scores in *Tamworth*. However, the *Tamworth 1988* mean GI score was less than that of *Tamworth 1963* but not significantly so.

The comparison of contemporary groups, *Lismore 1989 and Tamworth 1988*, revealed very little intergroup mean GI score difference. This difference was of no statistical significance. To reiterate, there was no statistically significant difference between the GI scores for *Tamworth 1963, Tamworth 1988 and Lismore 1989*. The differences between the *Lismore 1989 and Tamworth 1988* groups was very small. However, the mean GI scores for *Tamworth 1988 and Lismore 1989* were noticeably lower than the *Tamworth 1963* group. As well, *Tamworth 1963, 1988 and Lismore 1989* all had significantly higher mean GI scores than *Tamworth 1973*.

Tamworth: As previously noted, several trait means showed a significant decrease from 1963 to 1973, then a significant rise to 1988. These traits exhibiting this trend are no doubt responsible for such a trend in mean GI scores from 1973 to 1988.

Traits which showed significant differences following the abovementioned trend were:

- 1) +V (Overbite)) declined *Tamworth* 1963 to 1973
) and then rose
2) +H (Overjet)) *Tamworth* 1973 to 1988.

Tamworth GI traits showing continuing decrease or a levelling off after reduction with time were:

- 1) CR_r (posterior crossbite-RHS) continued decline from 1963 - 1988. But no significant change after 1973
- 2) CR_L (posterior crossbite - LHS) declined from 1963 - 1973 and then insignificant increase again in 1988.
- 3) BAP_R (RHS buccal segment sagittal deviation) - showed a similar reaction to CR_L
- 4) BAP_L (LHS buccal segment sagittal deviation) - continued decline from 1963 - 1988 but no significant change after 1973.

Lower crowding showed a significant increase from 1963 to 1973, and then again, a significant increase to 1988.

Open bite (-V) and Reverse Overjet (-H) showed no significant changes from 1963 - 1988.

However, both sides of the mouth showed significant decreases in posterior crowding only after 1973 (*i.e.* 1988). However levels of posterior crowding on both sides had fallen insignificantly from 1963 to 1973.

The contemporary groups showed significant differences for the following GI traits:

- 1) CR_L - *Lismore* showed significantly less.
- 2) BAP_R *Lismore* showed significantly less deviation from ideal.
- 3) A_c (lower anterior crowding) - *Lismore* showed significantly more.

- 4) PC_L (posterior crowding - LHS). *Lismore* showed significantly more.
- 5) +H (overjet). *Lismore* showed significantly more.
- 6) -H (reverse overjet). *Lismore* showed significantly less.

DISCUSSION OF THE RESULTS OF THE "ORTHODONTIC TREATMENT" CATEGORY

The previous *Tamworth Malocclusion Surveys of 1963 and 1973* took no account of the numbers who had had, or were presently having orthodontic intervention or treatment.

The present writer was asked to obtain figures for the group in *Tamworth 1988*. Subsequently, figures were also obtained for *Lismore 1989*.

Results showed that the percentage of patients fitting the above categories for the 10-19 year age group in the *1987-88 Australian National Oral Health Survey*, was 10.75% of the surveyed group in *N.S.W.* In *Victoria*, it was 14.6% of the surveyed group. Compared with these, *Tamworth* at 17.5% and *Lismore* at 25%, were much higher than expected, especially when compared to *N.S.W.* figures for the 10-19 year-old age group.

The *Lismore* percentage was also noticeably larger than the *Tamworth* percentage. This may be due to the socio-economic spread of the Kadina High School subjects examined. It was acknowledged by the Deputy Principal of the School, and the Regional Principal Dental Officer, that this school had more students from a higher socio-economic bracket than most other schools in *Lismore*. It might be assumed then, that their parents were better able to afford orthodontic care for their children.

Likewise, in *Tamworth*, two of the schools examined had a student body from a higher socioeconomic bracket than the remainder of the schools. These two schools may have weighted the *Tamworth* results for orthodontic treatment as well.

No male versus female breakdown was made for *Tamworth*. However, for *Lismore*, 62.5% of those undergoing, or who had undergone orthodontic treatment were females. While, 14 year-old girls comprised the largest single group who had had, or were undergoing orthodontic treatment. They comprised 32.5% of the entire *Lismore* sample.

DISCUSSIONS OF THE RESULTS OF CORRELATION DATA FOR TAMWORTH 1988 and LISMORE 1989

Tamworth:

Results for *Tamworth* showed that all GI traits, as well as the A_s , positively correlated with each other. Only 2 correlations were not at the $P < 0.001$ level, these were; lower anterior crowding with BAP_L (correlated at $P < 0.05$); and lower anterior crowding with BAP_R (correlated at $P < 0.01$).

It was noted that the A_s correlated least with overbite. This was taken to reflect this author's opinion at the time of recording that overbite *per se* was not an indication for treatment in most cases.

Strong correlations were found for CR_L and CR_R . This is to be expected, if a posterior crossbite existed on one side, there would be a good chance of the other side also having a crossbite or near crossbite. A similar strong correlation was also found for PC_R and PC_L . It is quite likely that if one side has crowding, the other side is likely to also have crowding.

Interestingly, posterior crowding and posterior crossbite were also strongly correlated. This perhaps relates to the mechanism whereby fluoride can prevent deciduous tooth loss and subsequent possible arch-length loss. With arch length loss, upper second premolars especially, may erupt lingually into crossbite. This author imagines, that if this were severe enough then upper first premolars may do this as well. Similarly, upper first permanent molars, may, as they drift forward, and rotate around their lingual roots, actually rotate into crossbite. Naturally, this mechanism can lead to worsened molar relationships more toward Class II.

Another explanation of this effect may be that, especially in *Tamworth*, with increasing Angle's Class III malocclusions (hinting at an underlying skeletal base problem), that the crossbite and posterior crowding correlation is due to a large proportion of small maxillae both sagittally and transversely.

The A_s and GI total correlated highly. This confirms their complementary roles in the overall *Malocclusion Surveys*. Overbite and overjet were again found to be positively related in a strong way. All GI traits and the A_s showed positive correlations, most at $P < 0.001$. No further discussion shall be undertaken of all the correlations done.

Lismore:

The value of the *Lismore* group, was that individual D, M, F and DMFT scores were available for each person. Thus, correlations could be made between DMFT elements and occlusal traits.

Unlike *Tamworth*, some traits did not correlate significantly, the D and F, and DMFT when compared to certain traits were amongst these. However, the M element showed positive correlations always at $P < 0.001$ level. The fact that D and F scores were not correlated, or lowly correlated, may reflect the fact that caries and poor restorations have little contemporary impact on malocclusion. Rather, the role of missing teeth may be the main mechanism still operational in causing malocclusions related to loss of tooth substance and subsequent arch length loss. This may further complement *Powell's* findings in 1985.

M correlations of interest are:

M with A_s . This was at the $P < 0.001$ level. Thus, as the number of missing permanent teeth increased, the subjectively assessed need of treatment increased. Thus, even though *Lismore* had only 3 subjects with an M score > 0 , the relationship between missing permanent teeth and treatment-need is vindicated.

M with A_c . Significant correlation at $P < 0.001$. Again, as M increased so did lower incisor crowding. The author has no explanation for this. It might be expected that with M reflecting caries and eventual extraction of first permanent molars and/or premolars, that lower anterior crowding may be relieved if posteriors were lost. *Walther (1966)* supports this mechanism.

M with PC_L and PC_R. This relationship lends support to the argument for missing teeth allowing drift and arch length loss. This will naturally depend on the site of tooth loss. Therefore, no specifics can be obtained from this relationship. The relationship of earlier d, m, f and dmft scores for these subjects may have been more enlightening.

M with V. Significant at $P < 0.001$. This relationship could be interpreted as agreeing with *Walther (1966)*; and *McEwen and McHugh (1970)*. These authors felt that missing teeth especially loss of 6s could lead to degrees of posterior bite collapse. Also, they felt that anteriors may retrocline and close the bite by this mechanism. As well, subsequent anterior overeruption might exacerbate this.

M with H. Significant at $P < 0.001$. Again, the effect might vary with the site of tooth loss. *Walther (1966)* proposed that there is in fact a reduction in Class I increased overjet malocclusions.

However, one mechanism that may support his correlation is that of a Class II Div. 1 malocclusion with a lip trap of hypertonicity of the lower lip. Here, further retroclination of the lower anteriors may be allowed with lower posterior permanent tooth loss. Again, comparing d, m, f and dmft with H may be of more use.

M with BAP_R and BAP_L. Significant at $P < 0.001$. Again, depending on the site of loss of permanent tooth (teeth), a valid explanation may be made. With loss of an upper first permanent molar, the 7 may not erupt fully into the 6 position thus

leading to a Class III molar relationship with the lower 6, or a Class II relationship with the lower 7.

In the lower arch, the loss of a lower 6 may allow minor forward drift and eruption of the 7, but more likely a posterior drift of the lower 5. Thus a disrupted buccal relationship can occur in both instances where permanent tooth loss occurs. This is supported by *Walther (1966); Godfrey (1990)*.

M with CR_R and CR_L. Significant at $P < 0.001$. *Ronnerman and Thilander (1977)* showed a contraction in maxillary length when early deciduous tooth loss occurred. Several clinicians (*Dawes, 1989; Godfrey, 1989*) have also supported this view when permanent teeth are lost or do not form. For example missing lateral incisors seem to be associated with dentoalveolar Class III malocclusions due to lack of dentoalveolus in the anterior maxilla. Is it possible that loss of other permanent teeth more posteriorly may lead to posterior crossbite? This might be due to posterior transverse arch contraction, or as mentioned earlier, rotation of upper 6s or 7s around their palatal roots. This author can only speculate as this mechanism has never been written up by him. However, *Pedersen et al (1978)* report increased crossbite posteriorly where deciduous molars are lost early.

M with GI total. Significant at $P < 0.001$. It is to be expected that if the majority of subgroups that make up this trait total score, correlate highly with M, then the GI total itself will. Thus this correlation is really only a reflection of the GI subgroup correlations.

COMPARISON OF PEEL'S FINDINGS (1973)
WITH PRESENT FINDINGS

Statistical analyst, *Kirby (1990)* felt that the methods employed to analyse *Peel's* results from 1973 were inappropriate. Basically, the data was not normally distributed and thus should not have been analysed by the student's t test.

Once this was done, it was necessary for the present writer to follow the same path to be able to compare contemporary results from *Tamworth 1988* and *Lismore 1989*.

Additionally, *Kirby* found that stated standard deviations of *Godfrey* traits were incorrect when recalculated by her. As a result, all 1963 and 1973 results published by *Peel (1973)* were recalculated for accuracy and significance.

As well, age group comparisons between 1963, 1973 vs 1988 and *Lismore 1989* results were from different age group make-up. *Peel's* Angle's Classification results were obtainable for 12 years and 15 years age groups. As assessments also allowed results from 12 and 15 years ages to be obtained.

However, suitable results for the GI traits were presented only as combined age groups 10-11 years, and 14-15-16 years and total sample (for each sex). Neither age group was totally akin to the 12 and 15 years age groups examined in 1988 and *Lismore 1989*. However, it was felt that the 14-15-16 years age group was the "best fit" comparison.

This was chosen because there were very few children still with deciduous teeth present in the 12 years group. So it was felt the

14-15-16 group was most applicable, covering the early full permanent dentition, (barring 8s).

The total age groups for each sex from *1963 and 1973* were not compared with *1988, 1989* because they used ages as young as 6-7. It was felt any developmental age effects might invalidate inclusion of comparison with the total group, especially in light of *Knutson's (1955)* observations.

Angle's Classification:

Peel (1973) 1963 vs. 1973

- * No statistically significant sex differences
- * S.S. increase in the number of *Normal* occlusions at $P < 0.001$.
- * S.S. decrease in the number of Class I malocclusions at $P < 0.001$.
- * No statistically significant difference between Class II Div. 1 and 2 malocclusion scores.
- * S.S. increase in Class III malocclusions at $p < 0.001$.

The findings from *1988* generally support the findings of *Peel (1973)*. *1988* showed significantly more *Normal* and Class III occlusions compared with *1963*, as well. However, for overall groups, the only significant difference between *1973 and 1988* was that there were significantly more *Class II Div. 2* malocclusions recorded in *1988*. As well, there was an age effect noted in all year groups (i.e. *1963, 1973, 1988*). This effect showed significantly more *Normal* occlusions in 15 year-olds. This last effect was not noted by *Peel (1973)*.

Additionally, significantly more Class II Div. 2 malocclusions occurred in 15 year-old rather than 12 year-old females in both 1973 (*and 1988*). Again this was not documented by *Peel*.

As (Subjectively Assessed Treatment-Need)

Peel (1973) 1963 vs. 1973

- * No s.s. sex difference
- * S.S. increase in *Normal* occlusion at $P < 0.001$.
- * S.S. increase in - scores (no treatment-need, minor malocclusions) at $P < 0.001$.
- * S.S. decrease in + scores (G.P. level treatment-need) at $P < 0.001$.
- * No s.s. difference for ++ scores between 1963 and 1973.
- * 13-17 years groups, male and female showed significantly increased N and - scores over +, ++ scores at $P < 0.001$ from 1963 to 1973.

1988 results revealed that for the comparison of 1973 vs. 1988, there was no statistically significant difference in N and - vs. + and ++ scores.

However, 1988 results showed a significantly greater proportion of ++ over + scores compared to *Tamworth 1963 and 1973*. This has been attributed to different examiner criteria between *Peel (1973)*, *Godfrey (1963)* and the present writer in 1988.

It is also possible that examiner factors didn't contribute to this + vs ++ difference as much as has already been speculated.

Peel (1973) feels that with the advent of fluoridation, the caries-related, preventable malocclusions have reduced significantly. This now means that ++ malocclusions now recorded are of the more severe skeletal nature, which are suspected to have a greater genetic input. Thus, these severe malocclusions dominate the ++ end of the malocclusion spectrum and have produced a polar distribution of malocclusions. Fluoride is felt to have countered many caries-related, environment-related potential malocclusions; now rendering many occlusions in the N and - categories. There is then a "jump" past + malocclusions to mainly severe genetically-related ++ malocclusions. This writer also supports this contention as being a good explanation of the observation of ++ over + recordings.

Treatment-Need vs. No Treatment-Need

Peel 1973 1963 vs. 1973

- * a S.S. decrease in +,++ at the expense of a S.S. increase in N,-, in all age groups from 1963-1973 ($P<0.001$).
- * No s.s. sex difference.
- * 13-17 year-olds, male and female, had a s.s. increase in those not requiring orthodontic treatment from 1963 to 1973 ($P<0.001$).

1988 results revealed no s.s. difference between scores for N,-vs. +,++ (i.e. No Treatment-Need vs Treatment-Need) for 1988 vs. 1973, for males or females.

An increase in No Treatment-Need occurred between 12 and 15 years groups. No s.s. sex difference was noted. As well, 1988, like 1973 showed a s.s. increase in N,-over +,++ scores, over the 1963 group.

Godfrey Index

Peel 1973 1963 vs. 1973

- Lower anterior crowding:

- * 14-15-16 years age group showed a slight but not s.s. increase in lower anterior crowding from 1963 to 1973.
- * 10-11 years age group showed a s.s. increase in lower anterior crowding at $P < 0.01$; from 1963 to 1973.

Kirby (1990) reworked results from 1973 and 1963. She concluded that there was a s.s. increase in crowding from 1963 to 1973, and from 1973 to 1988. This was, as mentioned, using Peel's published results for 1963 and 1973 for the combined 14-15-16 years age groups compared with the combined 12 and 15 years age groups from 1988. Thus, there is some disagreement with Peel's 1973 findings.

- Posterior Crowding

- * A decrease in posterior crowding was found in the 0-11 years age groups (except on the right side) from 1963 - 1973. This was not s.s.
- * 14-15-16 years age groups showed an increase in posterior crowding. However, females showed a decrease on the right side. None of these findings were s.s.

1988 results for PC_L showed that there was significantly less posterior crowding on the left side in 1988 compared with 1963 and 1973. As well, Peel's findings of no s.s. difference between 1963 and 1973 was confirmed.

PC_R scores followed the same pattern as PC_L scores. Again Peel's (1973) findings are confirmed.

- Overbite

* the 14-15-16 years age group showed a s.s. decrease in overbite at $P < 0.01$, from 1963 to 1973.

* There was also a sex effect. Females overall, and males overall showed a s.s. effect for overbite reduction from 1963 to 1973. Females at $P < 0.001$, and males at $P < 0.01$.

Since 1988 comparisons were made with combined sex groups for 14-15-16 year-olds from Tamworth 1963 and 1973, then no confirmation of the sex effect was attempted. However, 1988 reworking confirmed Peel's (1973) results that overbite decreased significantly from 1963 - 1973. Additionally, 1988 results showed a s.s. increase again, in overbite, from 1973 to 1988. This was to a level slightly less (but not significantly so) than the 1963 level.

- Openbite

* 14-15-16 years age groups showed no s.s. change in openbite from 1963 to 1973.

* The overall female group showed a s.s. increase in -V scores from 1963 to 1973 at a $p < 0.02$.

1988 results show no s.s. difference between 1963 and 1973 and 1988 for openbite. Reworking of Peel's results confirms his findings. No sex difference was examined in the reworked results.

- Overjet

* Most age groups showed a s.s. decrease in overjet between 1963 and 1973 at $P < 0.05$.

* Examination of the overall female and male groups also showed a s.s. decrease in overjet from 1963 to 1973, at $p < 0.01$ for females, and $p < 0.02$ for males.

The examination of combined sex groups showed that from 1973 to 1988 there was a s.s. increase in overjet at $P < 0.05$. Peel's (1973) findings were confirmed, that a s.s. decrease in overjet occurred from 1963 to 1973. As well, the overjet scores in 1963 were significantly less than those in 1988. As mentioned earlier, overjet showed a parallel relationship with overbite from 1963 to 1973 to 1988.

- Reverse Overjet

* No s.s. differences in reverse overjet scores were found between 1963 and 1973. 1988 results showed that there was no s.s. difference between 1963 and 1973 and 1988 for reverse overjet. As well, Peel's (1973) findings comparing 1963 and 1973 for reverse overjet were confirmed in the 1988 analysis.

Reverse overjet mean scores, rose slightly from 1963 to 1973, then dropped to approximately 1963 levels in 1988. As

mentioned, no s.s. difference existed between these years for reverse overjet.

- Buccal Segment Sagittal Relationship Deviations (Right Side)

- * A s.s. decrease in BAP_R scores from 1963 to 1973 ($p < 0.05$).
- * 14-15-16 year-old males showed a s.s. decrease in BAP_R from 1963 to 1973 ($p < 0.05$).
- * 14-15-16 year-old females showed a s.s. decrease in BAP_R from 1963 to 1973 ($P < 0.02$). 1988 results revealed that there was no s.s. difference between 1973 and 1988 BAP_R scores. As well, Peel's (1973) findings were confirmed, that there was a s.s. decrease from 1963 to 1973.

- Buccal Segment Sagittal Relationship Deviations - (Left Side)

- * 14-15-16 year-old males showed no s.s. change from 1963 to 1973.
- * 14-15-16 year old females showed a s.s. decrease in BAP_L scores from 1963 to 1973 at a $P < 0.01$.

1988 analysis revealed no s.s. difference between overall BAP_L scores for 1973 to 1988, although the 1988 scores were slightly lower.

For the combined 1963 vs 1973 vs 1988 groups, this author found that Peel's findings for 14-15-16 year-old females were carried through to the combined sexes 14-15-16 year-old group. This showed a s.s. decrease in BAP_L for the overall groups from 1963 to 1973.

- Buccal Segment Sagittal Relationship Distal Deviations (Right Side)

* 14-15-16 year-old males showed a s.s. decrease in BAP_R D scores from 1963 to 1973 at a $P < 0.02$.

* 14-15-16 year old females showed a s.s. decrease in BAP_R D scores from 1963 to 1973 at a $P < 0.01$.

1988 findings revealed a s.s. increase in BAP_R D scores from 1973 to 1988. Peel's (1973) findings were confirmed, that a s.s. decrease in BAP_R D scores occurred from 1963 to 1973. The Tamworth 1988 mean BAP_R D score was significantly higher than the 1963 mean score.

- Buccal Segment Sagittal Relationship Distal Deviations (Left Side)

* 14-15-16 year old males showed no s.s. difference between 1963 and 1973 for BAP_L D scores.

* 14-15-16 year-old females showed a s.s. decrease ($P < 0.02$) in BAP_L D scores from 1963 to 1973.

Peel's (1973) findings were confirmed by this writer's reanalysis of earlier results. In reanalysis, as before, 14-15-16 year-old males and females were combined so it is to be expected that the combined groups would exhibit significance if one of its subgroups showed significance in its comparisons.

The 1988 group showed a s.s. increase in BAP_L D scores over the 1973 group. As well, the BAP_L D scores from 1988 were significantly higher than the 1963 scores.

- Buccal Segment Sagittal Relationship Mesial Deviations (Right Side)

* no s.s. change was noted in either male or female 14-15-16 year-old groups, or overall male or female groups.

1988 results showed a s.s. increase in BAP_R M scores over 1973. The 1988 mean score was approximately 5 times higher than either 1963 or 1973.

Additionally, Peel's (1973) findings were confirmed, that there was no s.s. difference between 1963 and 1973 BAP_R M scores.

- Buccal Segment Sagittal Relationship Mesial Deviations (Left Side)

There was no s.s. difference between 1963 and 1973 groups for ages or sexes, for BAP_L M scores.

The 1988 results followed a similar pattern as for BAP_R M scores. The 1988 scores were significantly higher than 1973 and 1963 BAP_L M mean scores. The BAP_L M mean score for 1988 was nearly four times greater than either 1963 or 1973.

As well, Peel's (1973) findings were confirmed, that there was no s.s. difference between 1963 and 1973 for BAP_L M mean scores.

- Posterior Crossbites (CR_L) (Left side)

* 14-15-16 year old males showed a s.s. decrease in CR_L scores (P<0.02).

* 14-15-16 year-old females showed no s.s. difference in CR_L scores from 1963 to 1973.

The 1988 group showed a s.s. increase in CR_L scores, compared to the 1973 combined 14-15-16 year-old group. Peel's (1973) finding were basically confirmed by using combined sexes 14-15-16 years group.

The male 14-15-16 years subgroup is felt to have been largely responsible for the significance in difference between 1963 and 1973. Remember, the female group exhibited no s.s. difference between 1963 and 1973.

The 1988 CR_L mean was significantly less than the 1963 CR_L mean.

- Posterior Crossbite (Right Side)

* 14-15-16 year old males showed a s.s. decrease in CR_R scores from 1963 to 1973.

* 14-15-16 year-old females showed no s.s. difference in CR_R scores from 1963 to 1973.

The 1988 overall group showed no s.s. difference, when compared to the 1973 combined group for CR_R scores.

Peel's (1973) findings are basically supported when combined sexes 14-15-16 year-old groups are considered. Again it is felt that the male 14-15-16 year-old subgroup is largely responsible for the significant difference between CR_R scores for 1963 and 1973. Note that the female group in 1963 and 1973 showed no s.s. difference.

- Godfrey Index Total

* from 1963 to 1973 there was a s.s. decrease in GI total scores at $P < 0.01$. This was for the male and female 14-15-16 years groups.

1988 results showed a s.s. increase in GI total scores from 1973 to 1988 using the combined sexes 14-15-16 years group for 1963 and 1973.

There was no s.s. difference between 1963 and 1988 group GI total scores.

Peel's (1973) findings are confirmed, that there was a s.s. decrease in GI total scores from 1963 to 1973.

Overall, Peel's (1973) findings, when reworked for overall 14-15-16 years groups, yielded basically similar findings to his original results. The comparison of combined 12 and 15 year-old groups from 1988 with the 14-15-16 year-old groups of 1963 and 1973, was felt to be reasonable. Note also, that comparisons with Lismore, 1989, were with predominantly 13 and 14 year-olds.

Peel considered the changes in malocclusion between 1963 and 1973 could have been due to population changes; intra- and inter-examiner variability; the effect of local dental programs; or fluoridation. He attributed the majority of the 1963 to 1973 reduction in malocclusions to be due to fluoridation.

The present writer also considered the above factors as having input in the *1963 to 1973 to 1988* results. As mentioned above, there was very little difference of significance between *Tamworth 1973 and 1988*.

Tamworth has now grown to a population of 33,000, with a large immediate satellite population. However, the population has not changed qualitatively to any marked degree (*Atlas of N.S.W., 1987*).

The present writer feels that examiner variability entered the picture only for Subjectively Assessed Treatment-Need, + vs ++ scoring differences have been previously discussed. It was felt however, that N,- vs +,++ was quite valid inter-study, despite + vs ++ differences between *1963, 1973 and 1988*. Even if the Subjectively Assessed Treatment-Need method results are disregarded, it is felt that the Angle's Classification and Godfrey Index were performed objectively and that these results are valid.

The problem of assessing changes in private and school dental practice emphases was raised by *Peel (1973)*. Fissure sealants too, entered the scene in a big way from the late *1970s* and may have complemented the action of fluoride. Additionally, the use of pulpotomies rather than extractions, may have increased. However, many of these subjects examined derived care from private practices while others received school dental care. Gathering information on this was not attempted. From the evidence presented at the F.D.I. Joint Working Group Inquiry into the secular caries decline (*1985*), it appears that the input from this aspect is minimal.

However, note that the Joint Working Group did not address the input of fissure sealants. Peel concluded water-fluoridation produced the majority of the effect noticed for malocclusion reduction from 1963 to 1973. This writer also concludes that this agent is principally involved in *Tamworth* malocclusion reductions. Although, some input from fluoride toothpaste is also assumed. Despite the greater fluoride experience of contemporary 1988 *Tamworth* children, their malocclusion prevalence was not significantly different to *Tamworth (1973)* where water-fluoridation was the main agent involved. It is worth remembering that 12 and 15 year-olds in 1973 did not consume fluoridated water all their lives. This has led this author to propose a threshold level of fluoride exposure beyond which early deciduous and permanent tooth is minimal. Beyond this level of fluoride exposure, malocclusions do not significantly decline.

PART VI

SUMMARY AND CONCLUSIONS

The effectiveness of fluoride in its various forms and vehicles has been discussed in depth earlier. Principally of interest to this thesis are water fluoridation and fluoride toothpaste.

Water fluoridation has produced reductions in decay from 20-90%. The average reduction is approximately 50% for both dentitions, for lifelong consumers of fluoridated water.

Fluoride toothpastes may reduce caries up to 15 - 30% in non-water fluoridated communities. The average caries reduction is 20%. *Beltran and Burt (1988)* mention that average, superimposed caries reductions, in water-fluoridated areas are 17%.

Silverstone (1978), Mellberg and Ripa (1983), and Ophaug and Singer (1988) mention the systemic effects possible with the ingestion of fluoride toothpastes. In non-water fluoridated areas, this may be beneficial. In water-fluoridated areas, this may predispose to fluorosis. Generally, fluorosis has been found from ≥ 2 ppmF ingestion from fluoridated water. However, *Grahnen et al (1974)* found that 31% of subjects in their study showed fluorosis signs at 1 - 1.2 ppmF. Significant fluorosis was evident at ≥ 1.2 ppmF. On the other hand, *Craig (1990)* reported very few subjects (4% in 12 year-olds, 5% in 15 year-olds) with any fluorosis signs from the 1988 Tamworth Fluoridation Survey. It would appear that despite more assumed fluoride exposure, these Tamworth children are not exceeding the usually-accepted level of 2 ppm F. They may even be getting less than 1.2 ppmF if *Grahnen et al's study* has credence.

Brook (1975) demonstrated that fluoride has no association with an increase or decrease in anomalies such as hypodontia and hyperdontia, or form changes.

Fluoride has been shown to work by -

- a) increased enamel resistance
- b) increased rate of maturation post-eruptively
- c) remineralisation of incipient lesions post-eruptively
- d) altered tooth morphology
- e) antibacterial and intra-oral ecological changes

All modes were discussed fully earlier. The change in tooth morphology found in a number of studies favours decreased caries through shallower and wider pits and fissures. As well, the possibility of tooth size reduction as documented by some authors (*Division of Dental Research, Univ. of Toronto, 1954; Cooper and Ludwig, 1965; Goose and Roberts, 1979; Omar, 1987*) was raised as a mechanism which might lead to a diminished tooth size to basal bone size discrepancy (*Wallenius, 1959*).

No definitive statement could be made on this mechanism since variable results were obtained. As well, the secular trend for an increase in tooth size of offspring over their parents (*Garn et al, 1968; Lavelle, 1972*) complicates the interpretation of some studies. As well, this author is unaware of any confirmed secular trend for increasing or decreasing skeletal dimensions of offspring over their parents which may have orthodontic impact.

Current opinion favours the post-eruptive mechanism of fluoride as being more important than previously thought.

There is still a 25% difference in the U.S.A. between caries scores for children in water-fluoridated and non-water fluoridated areas. In the present study, in the age groups concerned, there was a 40% DMFT difference between *Tamworth 1988* and *Lismore 1989*.

This difference between fluoridated and non-fluoridated towns would have been higher if not for the secular decline in caries levels from the late *1970s*. This decline has been principally attributed to the introduction of, and widespread domination of the toothpaste market by fluoride toothpastes.

It was thus assumed that *Tamworth* children now obtain an additive systemic and topical effect from fluoride toothpaste. It is also assumed that *Lismore* children derive most of their fluoride from the topical and systemic effects of fluoride toothpaste.

Dramatic changes have occurred in DMFT and dmft scores for *Tamworth* from *1963* to *1988*. These changes highlight the effectiveness of fluoride. It is assumed that reductions in DMFT and dmft scores were principally attributable to water fluoridation up until *1979*. From *1979* on, the combined effects of water fluoridation and fluoride toothpaste are assumed to have been at work in *Tamworth*.

DMFT and dmft scores for *Tamworth* show that since *1973*, missing teeth have formed a very small part of the total DMFT and dmft scores.

It is concluded that it is primarily missing teeth due to caries

(principally 6s, and Es and Ds) that are responsible for most preventable, caries-related malocclusions.

Several authors (*Walther, 1966; Owen, 1971; Helm and Siersbaek-Nielsen, 1973; Miyamoto et al, 1976; Ronnerman and Thilander, 1977 and 1978; Pedersen, 1978; Dearing, 1981*) have documented the effects of early deciduous and permanent tooth loss. They also note the increased need of orthodontic treatment from the ensuing malocclusions that often occur. Correlation data obtained for *Lismore 1989* supported the important association of missing teeth (in this case permanent teeth) with malocclusion. Positive correlations existed at $p < 0.001$ for M (missing permanent teeth) with the Subjectively Assessed Treatment-Need, and all Godfrey Index components.

Also in support of the above conclusion are previous studies (*Plater, 1949; Salzmann and Ast, 1955; Okyay, 1957; Walther, 1960; Ast, Allaway and Draker, 1963; Murray, 1969*) which demonstrate the differences in malocclusion prevalence between fluoridated and non-fluoridated communities. *Hill, Blayney and Wolf (1959)* found that up to 50% of subjects with early deciduous or first permanent molar tooth loss don't develop a malocclusion. Working backwards, one can conclude that up to 50% of malocclusions are preventable if unwanted tooth loss can be curtailed by the use of preventive agents such as fluoride.

The results of the *1973 and 1988 Tamworth Malocclusion Surveys* also support the conclusion above, and demonstrate the effectiveness of fluoride in reducing caries-related malocclusions.

1973 and 1988 assessments of malocclusion in *Tamworth* by the Angle's Classification, Subjectively Assessed Treatment-Need, and Godfrey Index, demonstrate very little difference between the *1973 and 1988*

groups. However, a significant difference exists in most instances between the 1973, 1988 groups and the 1963 group. From 1963 to 1973 there were significant reductions in malocclusions.

There is very little difference between the two contemporary groups, *Tamworth* (water-fluoridated) 1988, and *Lismore* (non-water-fluoridated) 1989, with respect to the prevalence of malocclusion. It is felt by the writer that most significant differences are related to racial and gene pool differences between the two cities.

This writer has speculated that prior to the late 1970s, before widespread fluoride toothpaste availability, *Lismore* would have had malocclusion levels similar to *Tamworth* 1963.

It would also appear that there is a threshold amount of fluoride needed to substantially reduce caries and thus, tooth loss. For *Lismore*, obviously this level has been adequately supplied by fluoride toothpaste, in that malocclusion levels equate to those of water-fluoridated towns. However, this threshold level is not adequate to bring DMFT scores down to the level of a fluoridated town such as *Tamworth* 1988. Thus, the continuation of fluoridation of water supplies is urged. This is especially necessary for children from lower socioeconomic backgrounds.

These children are least likely to be able to afford other caries-reduction methods. There was a 71% caries-experience difference between children from lower social classes in fluoridated and non-fluoridated communities (*Carmichael et al, 1980*).

Summary of the Findings of the 1988 Tamworth and 1989 Lismore Malocclusion Surveys.

Angle's Classification

- * Significantly more Angle's Classification Normal occlusions were recorded in 1973 and 1988, compared with 1963.
- * Significantly more Class III malocclusions were recorded in 1973 and 1988 compared with 1963. This was felt to be due to decreased loss of upper Ds and Es. In situations where a Class III skeletal pattern is present, the common forward drift of upper 6s may mask this skeletal pattern by yielding a Class I, or in extreme drift cases, a Class II molar relationship. Since fluoridation has effected less early D and E loss, in the maxilla, the true skeletal pattern is complemented by the expected Class III molar relationship now that the upper first molars can no longer drift forward. Several authors (*Salzmann and Ast, 1955 and Cooper and Ludwig, 1965*) have demonstrated no skeletal effect due to fluoride consumption at the normal 1 ppmF level. As well, lower first molar forward drift in cases of lower D and E early loss has been shown to be minimal by *Walther (1966); Owen (1971); Pedersen et al (1978)*. Thus, early D and E loss, in the maxilla often favours a drift of upper 6s towards Class II molar relationship. However, early D and/or E loss in the mandible does not often increase the chance of lower 6s drifting towards a Class III molar relationship.

- * Significantly more Class II Div. 2 malocclusions were recorded in 1988 than in 1973 and 1963. This author proposes that these may be those Class II Div. 2 malocclusions where there is an underlying skeletal III base. The local gene pool of the Tamworth region is high in Irish and Scottish descendants. Luffingham and Campbell (1974) found this racial group to have a higher than normal proportion of Class III Malocclusions in their Scottish study.
- * 15 year-olds had significantly more normal than 12 year-olds. This was attributed to the effect of mandibular growth proceeding beyond maxillary growth.
- * Comparison of the contemporary water-fluoridated Tamworth 1988 group with the non-water fluoridated Lismore 1989 group revealed a significantly greater proportion of Class II Div. 2 malocclusions in Tamworth. Lismore had a significantly greater proportion of Class I malocclusions. These results may reflect racial/gene pool differences. Lismore has a high Italian and Anglo-Saxon population make-up.

Subjectively Assessed Treatment-Need

- * Significantly more ++ than + malocclusions were recorded in 1988 compared with 1963 and 1973. This can be explained by
 - a) the present writer acknowledges that he felt that general practitioners could capably treat very few malocclusions. This was related to this writer's feelings in being a fresh postgraduate student in orthodontics. Despite being more

lenient towards + level malocclusions, *Lismore 1989* scores still depict this view.

b) another explanation put forward by *Peel (1973)* supported by this author, is that the more severe malocclusions that are not amenable to change by fluoride, dominate the ++ end of the malocclusion spectrum. Thus, there are very few occlusions found in the + category now. Instead, malocclusions are either absent or very minimal; or severe, and require specialist level treatment.

* Significantly more *Normal* and minor malocclusions were recorded in *1973 and 1988* than in *1963*.

* Again, as in the Angle's Classification, there were a significantly greater number of *normal (N)* and minor malocclusions (-) in 15 year-olds compared with 12 year-olds. This was in *1963, 1973 and 1988*. This was also found in *Lismore 1989*. This is again attributed to the effect of later growth of the mandible over the maxilla. The later maturation of males may also partly explain this effect in the older 15-year-old groups.

* Comparison of *Tamworth 1988 and Lismore 1989* revealed only the age effect just discussed and the + vs ++ effect previously discussed. *Tamworth* had a significantly greater proportion of ++ than + scores.

Treatment-Need vs No Treatment-Need (+,++ vs N,-)

This assessment is really a subgroup of the Subjectively Assessed Treatment-Need.

- * As mentioned previously, there was no statistically significant difference between *Lismore 1989* and *Tamworth 1988*.
- * As well, there was no s.s. difference between *Tamworth 1973* and *1988* with respect to Treatment-needed vs. No treatment-needed.
- * However, there was significantly less treatment-need amongst the *1973* and *1988 Tamworth* groups than *Tamworth 1963*.

Godfrey Index

- * There was significantly more lower anterior crowding moving from *1963* to *1973* to *1988*. As well, *Lismore 1989* had significantly more crowding than *Tamworth 1988*.
- * Posterior crowding was not significantly different between *1963* and *1973*. However, *Tamworth 1988* had significantly less posterior crowding than *1963* and *1973*. On the left side, *Lismore 1989* had significantly more posterior crowding than *Tamworth 1988*. There was no s.s. difference however, for the right side.
- * Overbite and overjet both decreased significantly from *1963* to *1973*, then increased significantly from *1973* to *1988*. Overbite was not significantly different between *Tamworth 1988* and *Lismore 1989*, however, overjet was significantly higher in *Lismore 1989* than in *Tamworth 1988*. The parallel relationship between overbite and overjet was felt to be significant.

- * There was no s.s. difference between *Tamworth 1963, 1973 and 1988* for openbite and reverse overjet. As well, there was no s.s. difference between *Tamworth 1988 and Lismore 1989* for openbite. However, *Lismore* had significantly less reverse overjet than *Tamworth 1988*. This last point highlights the racial differences which are felt to contribute to more Class III malocclusions in *Tamworth*.
- * Overall buccal segment deviations showed significant reductions from *1963 to 1973*. From *1973 to 1988*, no significant change occurred.

For distal deviations in buccal segment relationships, there was a significant decrease from *1963 to 1973*. From *1973 to 1988* there was a significant increase. The *1988* scores were over 50% higher than the *1963* scores (and of statistical significance).

Mesial deviations in buccal segment relationships showed no significant difference between *1963 and 1973*. However, there was a significant increase in mesial scores from *1973 to 1988*. This gave *1988* means approximately 4 times higher than *1973 and 1963*.

Overall, for buccal segment deviations, *Lismore 1989* generally had significantly less deviation than *Tamworth 1988*.

This author cannot explain why distal deviations decreased significantly from *1963 to 1973* then rose significantly from *1973 to levels in 1988*, that were significantly higher than *1963* scores.

- * Posterior crossbites decreased significantly from 1963 to 1973. They rose significantly again only on the left side from 1973 to 1988. *Lismore 1989* had significantly lower posterior crossbite scores than *Tamworth 1988*, on the left side only. There was no s.s. difference on the right side between the two towns.
- * Godfrey Index Total scores dropped significantly from 1963 to 1973. There was a significant increase in scores from 1973 to 1988. There was no s.s. difference between the Godfrey Index means for *Tamworth 1963, 1988 and Lismore 1989*.

Utilisation of Orthodontic Treatment

In *Tamworth 1988*, 17.5% of the 12 and 15 year-old subjects eligible for the Survey were undergoing or had undergone orthodontic treatment. In *Lismore 1989*, 25% were in this category. In *Lismore*, females comprised 62.5% of those in the orthodontic treatment category. 14 year-old girls were the highest single treatment group.

A critique of the 3 methods of malocclusion registration used in the *Tamworth Malocclusion Surveys* highlighted some advantages, but also some shortcomings in each method.

As well, the Dental Aesthetic Index, and other recent indices were reviewed. Their advantages and shortcomings were highlighted.

Indeed, no ideal malocclusion index has yet been devised that covers all aspects of malocclusion recording and is valid in all situations.

This author believes that from the study of *Katz (1978)*, that some

modifications of the Angle's Classification may actually yield sufficient information as far as social adequacy, individual perceptions, and morphological deviation, to be a simple all-encompassing index. Modifications might indicate quantification of overjet, and crowding/rotations/displacements.

It was felt by the present writer, that the 3 methods used in the Tamworth Malocclusion Surveys actually complemented each other and covered most deficiencies of each single method.

Intimately related to the discussion above is the belief by this writer that there is still no consensus between authors as to when a malocclusion exists. Any definitions need to encompass the individual's self-perception of their occlusion; the degree the occlusion conforms to social norms; and the degree of deviation from morphological ideals. It would appear then, that once a valid definition of malocclusion is found, the more likely a valid, all encompassing malocclusion index will be devised.

PART VII
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APPENDIX

PREVALENCE OF CLASS III occlusion

Gardiner (1955) [cited by Jago, 1974]

Of 1,000 6 - 15 year-old subjects in an English study, 0.6% had Class III occlusions.

Goose Thomson and Winter (1957) [cited by Jago, 1974]

Of 2,956 subjects 7-15 years-olds, 6% had Class III occlusions. *Walther (1960)*

East Anglia schoolchildren. Approximately 10% had a Skeletal III tendency or a definite Skeletal III pattern.

Freer (1970)

Samples from white populations show generally less than 8% with a Class III malocclusion

Bowden et al (1973)

In a north-west English county, mandibular prognathism was 0.5%.

Luffingham and Campbell (1974).

Paisley (Scotland) 14 year-old schoolchildren showed 16% Skeletal Class III patterns. Dental Class III malocclusion was only 8%. These findings supported their personal clinical impressions that N.W. Scotland had a higher number of such occlusions.

McLain and Proffit (1985)

9.4% of American whites have Class III malocclusions.

However, for an Italian sample, *Chiavaro (1973)* cited by *Jago (1974)* found 8.9% of the subjects examined, 1,000 3-6 year-olds, had Class III malocclusions. *Lismore* has a marked Italian population. Note that this young age may not truly reflect later Class III malocclusion levels.

Lavelle (1976) noted malocclusion traits for a sample British population:

Trait	Present Day British Caucasoids		Anglo-Saxons Period	Medieval Period
	Male	Female		
Mandibular overjet	0.6%	0.2%	10%	14.3%
mesial molar occlusion	2.8%	3.2%	45%	23%