UNICYSTIC AMELOBLASTOMA: A CRITICAL APPRAISAL

SUVIR SINGH

Department Of Maxillofacial And Oral Surgery

Faculty Of Dentistry

University Of The Western Cape

UNICYSTIC AMELOBLASTOMA: A CRITICAL APPRAISAL

SUVIR SINGH

Thesis submitted in partial fulfillment of the requirements for the degree of Magister Chirugiae Dentium in the discipline of Maxillofacial and Oral Surgery in the Faculty of Dentistry of the University of the Western Cape.

SUPERVISORS : Professor M. Shear
Department of Oral Pathology
Faculty of Dentistry
University of the Western Cape
South Africa

Professor G. Kariem
Department of Maxillofacial and Oral Surgery
Faculty of Dentistry
University of the Western Cape
South Africa

EXAMINERS : Professor J. Morkel
Department of Maxillofacial and Oral Surgery
Faculty of Dentistry
University Stellenbosch
South Africa

Dr. B. Berezowski
Private Practice
Cape Town
South Africa

TABLE OF CONTENTS **PAGE**

TABLE OF CONTENTS **PAGE**

TABLE OF CONTENTS **PAGE**

LIST OF FIGURES **PAGE**

LIST OF FIGURES **PAGE**

LIST OF TABLES **PAGE**

DECLARATION

I, Suvir Singh, declare that "UNICYSTIC AMELOBLASTOMA: A CRITICAL APPRAISAL" is my own work and that all the sources I have used or quoted have been indicated and acknowledged by means of complete references.

Signed.....

S. Singh

DEDICATION

This thesis is dedicated to: my mom who gave me a goal; my dad who showed me how to attain that goal; and my wife, Nirvana, who helped me realise it.

ACKNOWLEDGEMENTS

I wish to express my sincere thanks to the following persons for their assistance with this research project.

1. Professor M. Shear, my promoter, whose support, encouragement and advice was central to the realisation of this thesis.
2. Professor G. Kariem, my promoter, for his advice and encouragement throughout this project.
3. Professor J. Hille for his encouragement and for making the records in the Department of Oral Pathology available to me and for assistance with the photomicrographs.
4. Messrs G. Du Toit and M. Petersen who willingly assisted me in the retrieval of the relevant records.
5. To all my colleagues and the staff at the Department of Maxillofacial and Oral Surgery at Groote Schuur Hospital for their support and with whom I had the pleasure of working.
6. The staff of the Maxillofacial and Oral Surgery Unit at King Edward VIII Hospital who graciously responded to my request for radiographs.
7. Ms Fionna Faroon for her patience and excellent computing skills of this manuscript.
8. Mr. Bradley Waltman for his invaluable contribution with the computer graphics.
9. Ms Prasheen Singh for her assistance with the statistical analysis of all the results.
10. My wife, Nirvana, for her sacrifice, perseverance and tolerance throughout my registrarship.

ABSTRACT

Robinson and Martinez first introduced the entity of unicystic ameloblastoma in 1977. Since then numerous case reports and series have been published. The evidence suggests that a more conservative approach can be used successfully to treat the unicystic ameloblastoma. The term unicystic is derived from the macro– and microscopic appearance of the lesion, whereas the term unilocular is used in radiological interpretation to describe a radiolucency having one loculus or compartment. Much confusion stems from the fact that a unicystic ameloblastoma might appear not only as a unilocular lesion, but also as what is often interpreted as a multilocular bone defect.

This study was carried out to appraise critically the lesions diagnosed as unicystic ameloblastomas in the Department of Oral Pathology at the University of the Western Cape.

This is a record based retrospective study analysing the unicystic ameloblastomas in the archives of the Department of Oral Pathology of the University of the Western Cape, since the inception of the biopsy service in 1977 to 1999. The sample was analysed according to thereferring hospital, age, sex, race of the patient, site of occurrence, clinical features, and radiological and histological features. The unicystic ameloblastoma can also give an apparently multilocular appearance and the Group 3 histological pattern (Ackermann *et al*, 1988) is the most common.

OPSOMMING

Robinson en Martinez het reeds in 1977 die entiteit unisistiese ameloblastoom bekendgestel. Sedertdien is verskeie gevalstudies en reekse gepubliseer. Van die inligting wil dit voorkom dat 'n meer konserwatiewe benadering gebruik kan word om die unisistiese ameloblastoom suksesvol te behandel. Die term unisisties word afgelei van die makro– en mikroskopiese voorkoms van die letsel terwyl die term unilokulêr gebruik word om in radiodeurskynende letsel met een lokulus te beskryf. Daar is baie verwarring omtrent die feit dat 'n unisistiese ameloblastoom nie net as 'n unilokulêre been defek geïnterpreteer kan word.

Hierdie studie was uitgedra om krities te kyk na letsels wat as unisistiese ameloblastome in die Departement Mondpatologie by die Universiteit Weskaap gediagnoseer is.

Hierdie is 'n rekord gebaseerde retrospektiewe studie wat die unisistiese ameloblastome in die argief van die Departement Mondpatologie van die Universitiet Weskaap sedert die begin van die biopsie diens in 1977 tot 1999, analiseer. Die proefsteek was ontleed volgens die verwysings hospitaal, die ouderdom, geslag en ras van die pasiënt, die plek waar die letsel voorkom, die kliniese tekens en die radiologiese en histologiese kenmerke. Die unisistiese ameloblastoom kan ook 'n waarskynlik multilokulêre voorkoms gee en die Groep 3 histologiese patroon (Ackermann *et al*, 1988) kom die mees algemeen voor.

INTRODUCTION

In the second edition of the World Health Organization's *Histological Typing of Odontogenic Tumours*, an ameloblastoma is defined as 'a benign but locally invasive polymorphic neoplasm consisting of proliferating odontogenic epithelium, which usually has a follicular or plexiform pattern lying in a fibrous stroma' (Kramer, Pindborg and Shear, 1992).

In a survey by Reichart, Philipsen and Sonner (1995) of 1500 publications in the English, German, French, Italian, Portuguese, Korean and Japanese literature, 3677 cases of ameloblastoma were documented between the years 1960 to 1993. This figure indicates the interest in the tumour but not the true incidence, which is defined as the number of new cases of a disease in a defined population over a fixed period of time. Shear and Rachanis (1979) found the age standardised incidence rate of ameloblastoma in the Witwatersrand, South Africa, to have been 5.17 per million population per year over the ten year period 1965–1974.

Currently three distinct types of ameloblastoma, based mainly on clinical behaviour and prognosis, can be distinguished:

i) the 'conventional or classical intraosseous', solid or multicystic ameloblastoma;
ii) the unicystic ameloblastoma; and
iii) the peripheral ameloblastoma (Philipsen and Reichart, 1998).

Robinson and Martinez (1977) were the first to introduce the entity unicystic ameloblastoma. Since then numerous case reports and series have been published. The available evidence suggests that a more conservative approach than that generally employed for the treatment of the 'conventional' ameloblastoma can be used successfully to treat the unicystic

ameloblastoma (Gardner and Pecak, 1980). However, in the case of Group 3 lesions *vide infra* in which there has been mural invasion, a more cautious approach to treatment is advisable (Ackermann, Altini and Shear, 1988).

The definition of the unicystic ameloblastoma has lacked precision. Roos, Raubenheimer and Van Heerden (1994) suggested that the unicystic ameloblastoma might be defined as a unilocular, cystic epithelial odontogenic tumour. Gardner (1999) emphasized that the definition of a unicystic ameloblastoma should be based on two main features: 'the lesion must be unilocular clinically and radiologically; and it must appear on microscopic examination as a single cystic lesion with the epithelial lining consisting of ameloblastoma'.

It is important that in the interpretation of these lesions, two definitions must be strictly adhered to. Firstly, that the term unicystic is derived from the macro– and microscopic appearance of the lesion, whereas, secondly, the term unilocular is used in radiological interpretation to describe a radiolucency having one loculus or compartment. Much confusion stems from the fact that a unicystic ameloblastoma may appear not only as a unilocular lesion but also as what is often erroneously interpreted as a multilocular bone defect. Furthermore, as I shall point out in the discussion, some lesions which appear radiologically to be unilocular, may turn out to be multicystic, and this factor might complicate the accuracy of diagnosis.

My interest in the subject was stimulated by this confusion between the radiological descriptions of unilocularity, multilocularity, and trabeculations; and the term unicystic, which can usually be determined only at operation or in the gross postoperative specimen. The diagnostic difficulty is aggravated for the pathologist when the lesion is not removed

intact. A yet further difficulty for the pathologist is the need to attempt a definitive diagnosis on a small biopsy.

In this study I have tried to clarify these issues in a sample of lesions diagnosed as unicystic ameloblastoma for which radiographs and microscopic sections have been available, including numbers of cases that I have treated myself.

LITERATURE REVIEW

Robinson and Martinez proposed the prognostically distinct entity 'unicystic ameloblastoma' in 1977. Since then, based on its histology, variants have been termed mural, intracystic, cystic or plexiform unicystic ameloblastoma (Ackermann *et al*, 1988). In the literature survey of Reichart *et al* (1995), unicystic ameloblastomas accounted for 6 percent of all intraosseous ameloblastomas.

(a) Clinical Presentation

Leider, Eversole and Barkin (1985) reported a clinicopathologic analysis of 33 cases of unicystic ameloblastomas. The lesions were either asymptomatic and discovered on routine radiographic examination or the patients noted an enlargement of the jaw without pain or parasthesia. All their cases occurred in the mandible with 77.4 percent in the molar–ramus region, 12.9 percent in the mandibular symphysis and 9.7 percent in the cuspid premolar region. Olaitan and Adekeye (1997) reported that swelling, ranging in duration from two months to eight years, was the principal finding in all their cases. Expansion of both buccal and lingual plates was noted in 85.7 percent of cases, whereas buccal expansion alone was seen in the remaining 14.3 percent. Only two of 21 patients complained of pain. In the latter study all the lesions were located in the mandible.

(b) ***Location***

Gardner, Morton and Worsham (1987); and Van Wyk, Thompson and Wyma (1986) each reported a unicystic ameloblastoma in the maxilla. In 1993 Thompson, Ferreira and Van Wyk reported a recurrence of their maxillary case. The lesion had been removed conservatively. Philipsen and Reichart (1998) stated that the location within the jaw bones greatly favoured the mandible with the ratio of mandible:maxilla in different studies ranging from 3:1 to 13:1. All the cases of Leider *et al* (1985) occurred in the mandible with 77.4 percent in the molar–ramus region, 12.9 percent in the mandibular symphysis and 9.7 percent in the cuspid premolar region. The lesion occurs most commonly in the mandibular third molar area and may be associated with an impacted tooth (Ackermann *et al*, 1988; Philipsen and Reichart, 1998). The latter authors referred to those unicystic ameloblastomas associated with an impacted tooth as 'dentigerous' variants and others as the 'non–dentigerous' variants.

(c) ***Age***

Age at the time of diagnosis is significantly younger ($p < 0.001$) for the unicystic ameloblastoma as opposed to the solid or multicystic ameloblastoma (Ackermann *et al*, 1988). In their series the mean age of the patients at the time of diagnosis was 23.8 years (SD 14.9), ranging from 6–77 years, with 48 percent occurring in the second decade and 86 percent occurring in the second to fourth decade. Leider *et al* (1985) found a similar age distribution in their series with a mean age of 26.9 years and 42 percent of lesions occurring in the second decade and 73 percent in the second and third decades. The reports by Philipsen and Reichart (1998) and Eversole, Leider and Strub (1984) have shown that the mean age at the time of diagnosis of the unicystic ameloblastoma correlates closely with the presence or absence of an impacted tooth. Almost 20 years separate the mean age of the 'dentigerous' variant from the 'non–dentigerous' variant (16.5 years versus 35.2 years), but neither set of data was analysed statistically.

(d) ***Gender***

The male to female ratio is approximately 1:1.3 (Leider *et al*, 1984; Ackermann *et al*, 1988; and Philipsen and Reichart, 1998).

(e) ***Race***

Shear and Singh (1978) showed that the age–standardized incidence rates of ameloblastoma on the Witwatersrand was much higher in South African blacks than whites with the ratios being 9.1:1 for black males versus white males and 3.7:1 for black females versus white females. However, they did not separate the unicystics from other forms of the lesion. In the series of Ackermann *et al* (1988) the majority of patients, 51 of 57 cases, were black.

Leider *et al* (1985) showed a different racial distribution with 45 percent White, 33 percent Black, 12 percent Hispanic and 10 percent Oriental. This distribution corresponded with that of the general population in the greater San Francisco Bay area.

(f) ***Radiological Features***

The radiological features of the unicystic ameloblastoma have received relatively little attention in the literature. A M*edline* literature search revealed only one article on this particular aspect, namely that of Eversole *et al* (1984) who conducted an extensive study of the radiological features of 31 unicystic ameloblastomas. Based on the two major categories of:

i) location and relationship to contiguous teeth; and

ii) radiographic configuration and pattern,

they identified six radiological patterns of the lesion:

(a) pericoronal, unilocular;

(b) extensive pericoronal, unilocular;

(c) pericoronal, scalloped;

(d) periapical, unilocular;

(e) interradicular; and

(f) multilocular.

Patterns (a) to (c) were associated with an impacted tooth, whereas (d) and (e) were not.

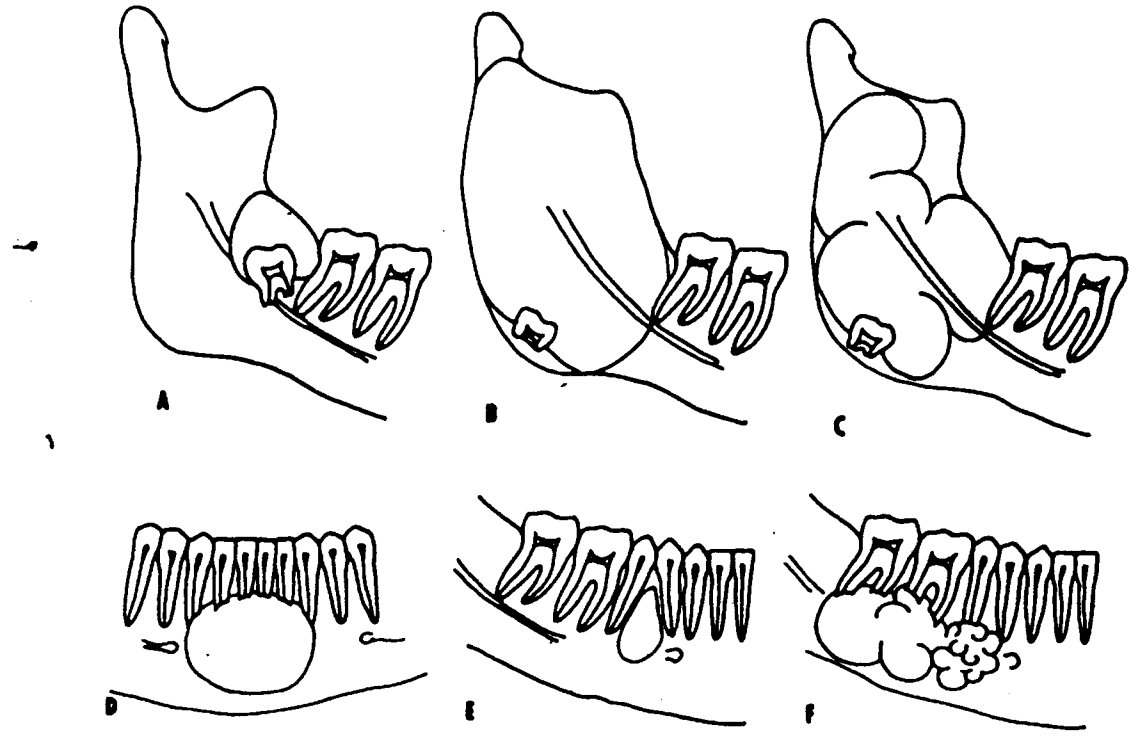

Figure 1: Diagrammatic representation of radiological patterns of the unicystic ameloblastoma. **A**, Pericoronal unilocular. **B**, Extensive pericoronal unilocular. **C**, Pericoronal scalloped. **D**, Periapical unilocular. **E**, Interradicular. **F**, Multilocular. (*Reprinted with permission of* Mosby, Inc. *from* Eversole LR, Leider AS, Strub D. Radiographic characteristics of cystogenic ameloblastoma. *Oral Surgery, Oral Medicine, Oral Pathology, Oral Radiology and Endodontics* 1984; 57:572–577.)

In all six patterns the lesions were radiolucent and well–defined, and occasionally a well demarcated peri–lesional corticated rim could be discerned. Expansion was common. Sixteen of the 31 cases were associated with a mandibular third molar and root development was variably arrested. The lesions not associated with an impacted tooth, showed root resorption or caused root divergence. The unilocular patterns were more common. The ratio of unilocular: 'apparently multilocular' patterns was 13:3 for the 'impaction associated' variant and 8:7 for the others.

When age was considered in relation to radiographic features, it was found that those unicystic ameloblastomas associated with impacted teeth occurred, on average, eight years earlier than those arising independent of impacted teeth. When both impaction and lesional configuration were considered together, it was found that the average age for unilocular impaction–associated tumours was 22 years whereas multilocular lesions without impaction occurred at an average age of 33 years.

Shear (1992) stated that the unicystic ameloblastoma appeared as either a well corticated unilocular radiolucency or the lesion may be trabeculated leading to an erroneous diagnosis of a multilocular cyst. Gardner (1999) raised the point that it is difficult to conceive that a true multilocular lesion may in fact be a unicystic ameloblastoma histologically. He elaborated by stating that a lesion that appears clinically and radiologically to occupy a single cavity, but which has an irregular, scalloped border, is sometimes referred to erroneously as being multilocular, and that such a lesion can be a unicystic ameloblastoma. Conversely an ameloblastoma that presents a radiologic appearance of being unilocular, that is, occupying a single compartment, may be either a unicystic ameloblastoma or a solid or multicystic (classic intraosseous) ameloblastoma. The distinction is made by histopathologic examination. In a personal communication to Gardner, Shear (1999) added that this

distinction might be made grossly at operation and by gross examination of the excised specimen as well as on histopathological examination.

Gardner (1999) stated that the definition of a unicystic ameloblastoma was important. It should be based on two features: the lesion must be unilocular clinically and radiologically; and secondly, it must appear on microscopic examination as a single cystic lesion with an ameloblastomatous epithelial lining.

Furuki *et al* (1997) reported the radiological findings in three recurrent unicystic ameloblastomas that had been initially treated by marsupialization. They identified six stages in the radiographic development of the recurrences:

Stage 1: Bone regeneration in the form of a ground–glass appearance occurred first at the periphery of the marsupialized cavity.

Stage 2: The surface of the regenerated bone soon showed a diffusely sclerotic band.

Stage 3: This became more evident and scalloped.

Stage 4: This scalloping extended downwards or laterally and became rounded.

Stage 5: The radiolucencies then became multilocular with a soap–bubble or honeycomb appearance.

Stage 6: In the final stage the recurrent lesion increased in size.

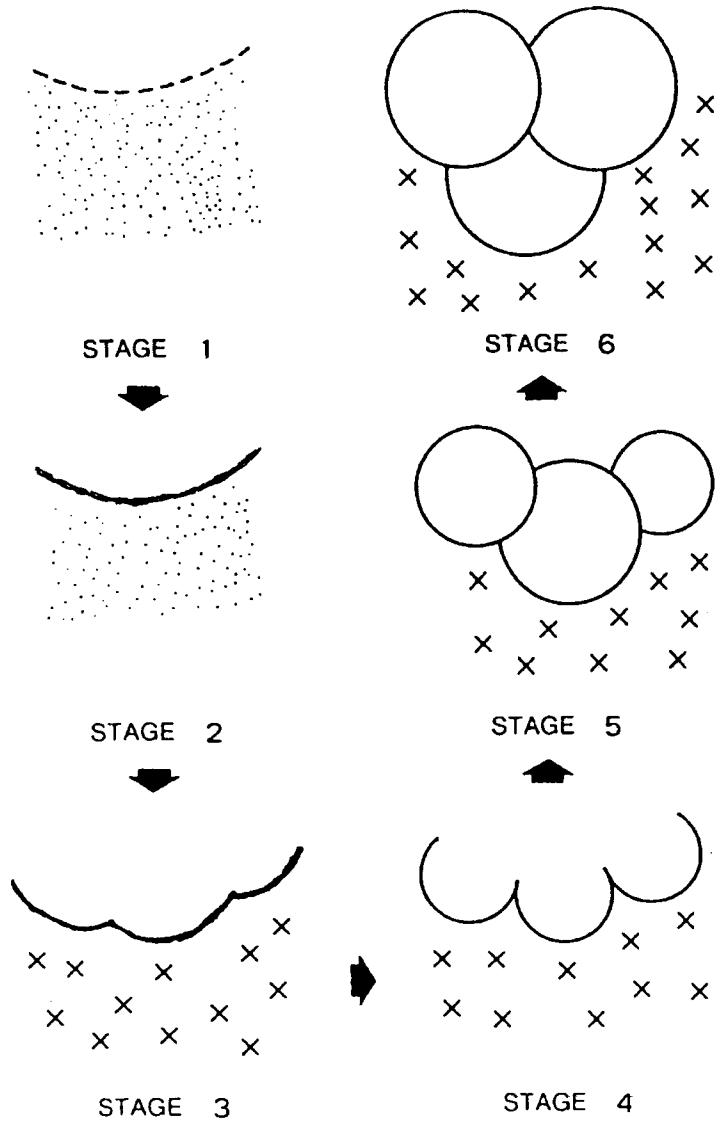

Figure 2: The six radiological stages in the development of a recurrent unicystic ameloblastoma. (*Reprinted with permission of* Nature Publishing Group *from* Furuki Y, Fujita M, Mitsugi M, Tanimoto K, Yoshiga K, Wada T. A radiographic study of recurrent unicystic ameloblastoma following marsupialization. Report of three cases. *Dentomaxillofacial Radiology* 1997; 26:214–218.)

They considered the scalloping of the sclerotic margin as the first obvious radiological sign of the recurrence. All the recurrent lesions showed the soap–bubble or honeycomb appearance radiologically. Furuki *et al* (1997) postulated that this might be the result of multicentric proliferation of the tumour. Another feature of interest in the Furuki study is the site of the recurrence. In each case, the recurrence was at the periphery of the regenerated bone, and not at the original margin of the lesion, or in adjacent cancellous bone.

Marks *et al* (1983) suggested that a preoperative computed tomography scan is an important part of the diagnostic armamentarium as it allows the surgeon better to establish the boundaries of the tumour and determine if the lesion extends beyond bone into the soft tissues.

(g) Macroscopic Features

Upon removal of the lesion, whether in total or piecemeal, it is important for the surgeon and pathologist to examine both the inside and outside of the cyst sac, as this may reveal important diagnostic clues (Philipsen and Reichart, 1998). The luminal surface of the sac may show one or several polypoid or papillomatous, pedunculated, exophytic masses. This subtype of unicystic ameloblastoma has been named intracystic, luminal, intraluminal, or mural ameloblastoma, and corresponds to the plexiform unicystic ameloblastoma (as termed by Gardner, 1981).

In addition to these intraluminal protruberances, the inside of the cyst may show one or several rounded and only slightly protruding nodules that in fact may also be viewed from the outside of the cyst wall. These formations are termed mural or intramural nodules and result from infiltrating and invading islands of ameloblastoma tissue. Philipsen and Reichart (1998) suggested the terms intraluminal unicystic ameloblastoma and intramural unicystic ameloblastoma for lesions displaying the protruberances and nodules respectively. The former term precisely indicates the location of the tissue proliferation.

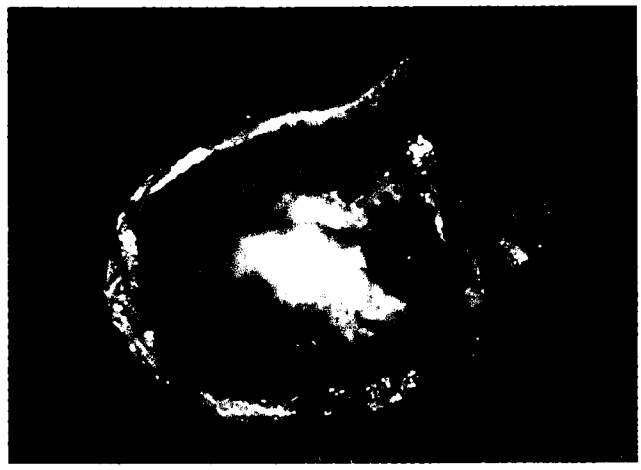

Figure 3: Bisected gross specimen of a simple unicystic ameloblastoma.

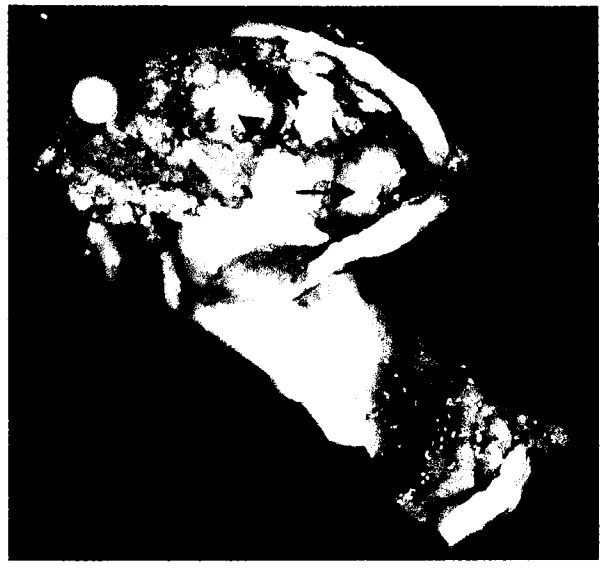

Figure 4: Opened gross specimen of a unicystic ameloblastoma showing intraluminal protruberances (indicated by arrows) – a so–called intraluminal unicystic ameloblastoma.

Figure 5: Opened gross specimen of a unicystic ameloblastoma showing intramural nodules (indicated by arrows) – a so–called intramural unicystic ameloblastoma.

(h) ***Histological Features***

Vickers and Gorlin (1970) studied 'ten examples of cystic lesions of the jaws that manifested a distinctly altered epithelial lining.' The histologic changes noted in this material were compared with published photomicrographs of early ameloblastomas, mural ameloblastomas, or examples of ameloblastoma arising in association with 'dental' cysts. They noted the following features which have since become established as the histological criteria for diagnosis of an ameloblastoma and are often referred to as the Vickers–Gorlin criteria.

These epithelial features were:

- hyperchromatism of basal cell nuclei of the epithelium lining the cystic cavities;
- palisading of the basal cells with polarization, sometimes referred to as reverse polarization, of the basal cell nuclei;
- cystoplasmic vacuolation of basal cells;
- marked intercellular spacing;
- homogenization or hyalinization of a thin, band–like area of fibrous tissue adjacent to the epithelium;
- bud–like proliferations of the basal layer; and
- epithelial nests seemingly detached from the extensions.

Hyperchromatism, palisading with polarization, and cytoplasmic vacuolation were constant histopathologic features of these cystic lesions. No feature appeared more significant and all three of these criteria should be present for the diagnosis of ameloblastoma.

Hyperchromatism of basal cell nuclei of the epithelium lining of the cystic cavities was observed in each of the ten specimens. It was apparent with low power examination and was photomicrographically reproducible (Vickers and Gorlin, 1970).

Palisading with polarization of basal cell nuclei of the epithelium lining the cystic cavities was observed in nine of ten specimens, with the exception being considered too small to be representative. Palisading is the term used to describe the orderly arrangement of epithelial cells with their long axes orientated at right angles to the basement membrane. Polarization, or reverse polarization, is a term describing the apparent movement of cell nuclei, away from the basement membrane. When observed together palisading and polarization of cell nuclei were considered noteworthy (Vickers and Gorlin, 1970).

Cytoplasmic vacuolation of basal cells of the epithelium lining the cystic cavities was observed in all but one specimen, the inadequate one. Cytoplasmic vacuolation was readily observed and was most prominent in that portion of the cell approximating the basement membrane. Intercellular spacing was also marked and suggested the possibility that an 'unidentified substance' was present between the cells. When cytoplasmic vacuolation and intercellular spacing occurred together and when they were most notable in basilar and parabasilar areas of the epithelium, they were considered noteworthy (Vickers and Gorlin, 1970).

The other histologic features of homogenization or hyalinization of a uniform, thin, band–like area of fibrous connective tissue adjacent to the epithelium, and bud–like proliferation of the epithelial lining were seen in six of the ten specimens. Epithelial nests, seemingly detached from the extensions, demonstrating histologic features of hyperchromatism, palisading with polarization, and cytoplasmic vacuolation with intercellular spacing, were also seen (Vickers

and Gorlin, 1970). The epithelial extensions may be considered neoplastic when they demonstrate the features of hyperchromatism of basal cell nuclei, palisading with polarization, and cytoplasmic vacuolation with intercellular spacing.

The importance of the narrow, eosinophilic, homogenous zone in the fibrous connective tissue adjacent to the altered epithelium of early ameloblastoma could not be determined. This was postulated to represent evidence of abortive dentine formation (Vickers and Gorlin, 1970).

(i) Histological Classifications of Unicystic Ameloblastomas

The first attempt at separating the varying histological patterns in the unicystic ameloblastoma was that of Robinson and Martinez in 1977. They identified four patterns:

1. A lining epithelim in which the basal cells were clearly columnar, with hyperchromatic nuclei, and the overlying cells were only loosely textured with absence of 'cohesiveness' – this separation of the suprabasilar cells could not be explained on the basis of inflammatory edema.
2. Downgrowth of the epithelium described in (1) into the connective tissue portion of the cyst wall.
3. The presence within the connective tissue portion of the cyst wall of epithelial islands composed of a periphery of columnar epithelial cells and a center identical with stellate reticulum.
4. Intraluminal nodules composed of anastomosing cords and islands of epithelium; the cells comprising these cords and islands are identical to those described in (3).

Ackermann *et al* (1988) elaborated on this description and proposed the following histological classification:

Group 1: Cyst lined by variable, often partly nondescript epithelium with no infiltration into the fibrous cyst wall, but having at least parts of the lining showing Vickers and Gorlin criteria. Inactive odontogenic rests might be present within the fibrous wall, but there is no infiltration of neoplastic epithelium.

Group 2: Cyst showing Group 1 features and in addition a nodule arising from the lining, projecting into the lumen of the cyst, comprising odontogenic epithelium with a plexiform pattern which closely resembles that seen in the plexiform ameloblastoma.

Group 3: Cyst with any features of Groups 1 and 2 and invasion of islands of ameloblastomatous epithelium into the connective tissue wall of the cyst. These islands may or may not be connected to the cyst lining. Nodules of tumour tissue similar to that seen in Group 2 may also be present. The invading islands of epithelium may be in either: (a) follicular, or (b) plexiform pattern.

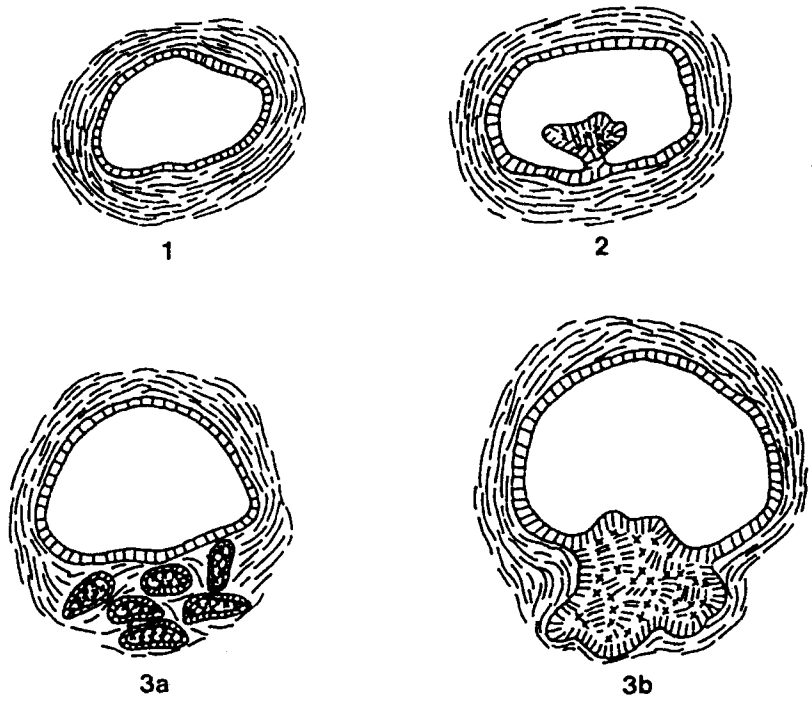

Figure 6: Diagrammatic representation of the histological classification of the unicystic ameloblastoma proposed by Ackermann *et al* (1988). (*Reprinted with permission of* Munksgaard *from* Ackermann EL, Altini M, Shear M. Unicystic ameloblastoma: a clinicopathological study of 57 cases. *Journal of Oral Pathology* 1988; 17:541–546.)

The distribution of their material according to this classification was 42 percent of cases classified as Group 1, 9 percent as Group 2 and 49 percent as Group 3. Roos *et al* (1994) reported a slightly different distribution as follows: Group 1 (50 percent), Group 2 (13.3 percent) and Group 3 (36.7 percent).

Philipsen and Reichart (1998) proposed a more elaborate classification modified from that of Ackermann *et al* (1988):

GROUP	INTERPRETATION
1	Simple UA
1,2	Simple and intralumenal UA
1,2,3	Simple, intralumenal and intramural UA
1,3	Simple and intramural UA

Altini and co–authors (2000) have reported an 81.5 percent positive staining of unicystic ameloblastoma for the calcium–binding protein, calretinin. This generally consisted of diffuse, intense nuclear and cytoplasmic staining of several cell layers of the more superficial cells both in the characteristic and nondescript areas of the cyst linings. Calretinin might be an important diagnostic aid. The value of this finding is that if a biopsy consists mainly of a nondescript epithelial lining when a unicystic ameloblastoma is suspected clinically, calretinin immunocytochemistry might prove invaluable in determining the diagnosis.

(j) Behaviour and Treatment

In 1977, Robinson and Martinez suggested that enucleation rather than partial or complete jaw resection appeared to constitute appropriate therapy. Gardner (1984) pointed out that there was a difference between the biological behaviour of those lesions that were simply cystic (Group 1) or showed intraluminal proliferations (Group 2) and of those in which the epithelium penetrated and breached the fibrous wall, and therefore had the capacity to invade cancellous bone (Group 3). Gardner and Corio (1984) reported a recurrence rate of 10.7 percent after treatment of unicystic ameloblastomas by enucleation or curettage. However, they reinforced the suggestion of Robinson and Martinez (1977) that the unicystic ameloblastoma should be treated by enucleation rather than segmental or marginal resection. Ackermann *et al* (1988) suggested that Groups 1 and 2 lesions could be treated conservatively, while Group 3 lesions should be treated aggressively, that is in the same manner as the conventional ameloblastomas.

Thompson *et al* (1993) reported the recurrence of a unicystic ameloblastoma of the maxilla. The lesion had been enucleated with the walls intact some six years earlier. Histological examination revealed infiltration of islands and strands of odontogenic epithelium into the

cyst wall. This corresponds to a Group 3 lesion and supports the view that Group 3 lesions be treated more aggressively.

Furuki *et al* (1997) reported that at the Hiroshima University Dental Hospital large cystic lesions of the jaws are treated by marsupialization alone. As pointed out above (p.8), they analysed radiographs of three recurrent lesions and described the radiographic patterns observed in the development of recurrences. They did not, however, discuss their total sample size and overall success rate with this treatment modality.

Li, Browne and Matthews (1995) used immunocytochemical techniques to study the expression of the markers Ki–67 and proliferating cell nuclear antigen (PCNA) in cyst linings, intraluminal nodules and invading tumour islands of unicystic ameloblastomas, and also in solid ameloblastomas. In the unicystic ameloblastomas the invading islands exhibited a significantly higher PCNA labelling index (29.2 ± 16.4 percent) than intraluminal nodules (13.6 ± 5.4 percent; $P < 0.05$). Unicystic tumour lining had relatively few PCNA positive cells and a labelling index (5.5 ± 3.3 percent) significantly lower than invading islands ($P < 0.001$) or intraluminal nodules ($P < 0.003$). The labelling indices of solid ameloblastomas of the follicular type (48.1 ± 12.9 percent) were significantly higher than those of cystic tumour lining ($P < 0.0001$), intraluminal nodules ($P < 0.001$) and invading islands ($P < 0.04$) in unicystic ameloblastoma. Similar relationships were found for Ki–67 expression except that the differences between invading islands and intraluminal nodules were not significant.

These results suggested to the authors that there were differences in the proliferative potential between different areas of unicystic ameloblastoma and between unicystic and solid lesions. Furthermore, the fact that invading tumour islands within the fibrous tissue wall showed

higher labelling indices than the unicystic linings and nodules provided biological support for the clinical observation that their presence might be related to recurrence after conservative surgery and indicated the need for a more radical surgical approach as the treatment of choice for this subgroup of lesions.

Philipsen and Reichart (1998) citing the above findings of Li *et al* (1995) further suggested that these methods of determining *in situ* proliferating activity might prove of value as an adjunct to histomorphological diagnosis in providing a better understanding of the biological behaviour of unicystic and solid ameloblastomas, and as guidelines for treatment planning.

Philipsen and Reichart (1998) questioned the value of a preoperative incisional biopsy on the grounds that it could be representative of the entire lesion in only very few instances and would probably result in an incorrect diagnosis and classification. The true nature of the lesion, they believed, might only become evident when the entire specimen was available for microscopy. The excised or operation specimens should be subjected to multiple or even serial sectioning to search for invading tumour islands in the cyst walls. If invading tumour islands were found, their presence should then indicate an aggressive surgical approach, possibly involving a second operation to remove adjacent bone and a follow–up period of at least 10 years (Philipsen and Reichart, 1998).

Roos and co–workers in 1994 reported a recurrence rate of 6.7 percent in their series of 30 cases. Recurrence rates for unicystic ameloblastomas after conservative treatment (curettage or enucleation) are generally reported to be less than 25 percent and a figure as low as 10.7 percent has been disclosed for unicystic ameloblastomas of the intraluminal, plexiform type (Philipsen and Reichart, 1998). This is considerably lower than the 50 to 90 percent

recurrence rates noted after curettage of solid and multicystic ameloblastomas. Gardner and Corio (1984) reported that one of their cases exhibited the histological appearance of a plexiform unicystic ameloblastoma (a Group 2 lesion) when first enucleated, but the recurrence two years later exhibited features of a typical conventional ameloblastoma. Another of their cases was a typical ameloblastoma when first operated, but later recurred as a plexiform unicystic ameloblastoma.

(k) Pathogenesis

Since their first description there has been much debate about the histogenesis of the unicystic ameloblastoma. Leider *et al* (1985) suggested three plausible pathogenic mechanisms:

Hypothesis 1: The reduced enamel epithelium associated with a developing tooth undergoes ameloblastomatous transformation with subsequent cystic development.

Hypothesis 2: The ameloblastomas may arise in a dentigerous or other type of odontogenic cyst in which neoplastic ameloblastomatous lining epithelium is preceded temporarily by a non–neoplastic stratified squamous epithelial lining.

Hypothesis 3: A solid tumour undergoes cystic degeneration of ameloblastomatous islands with subsequent fusion of multiple microcysts to develop a unicystic lesion.

They favoured hypothesis 2.

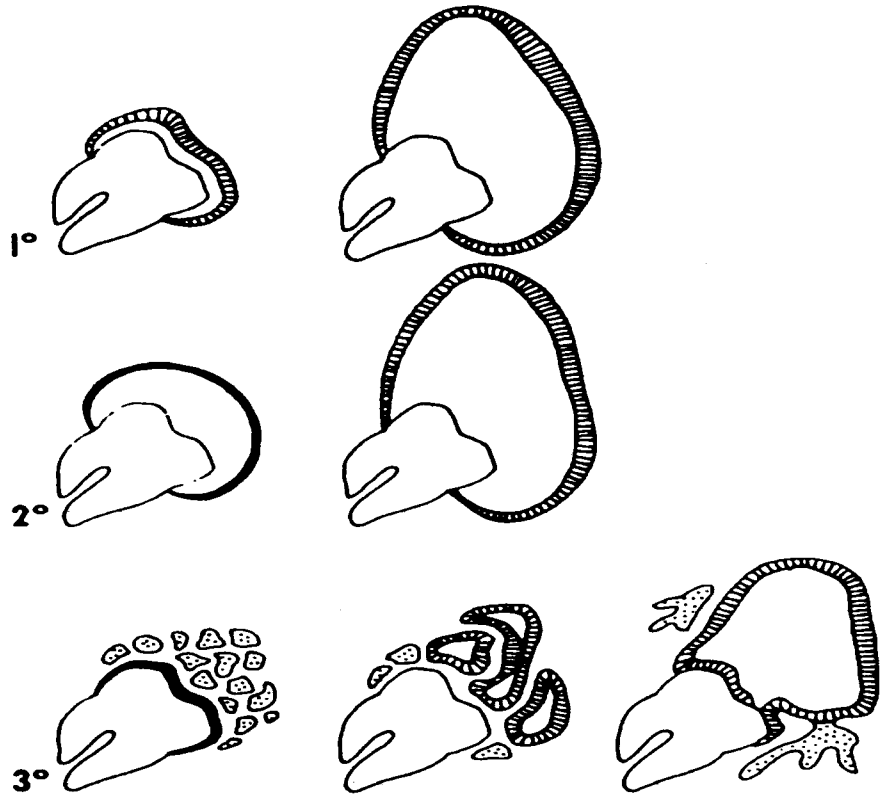

Figure 7: Diagrammatic representation of the hypotheses in the pathogenesis of the unicystic ameloblastoma. (*Reprinted with permission of* Mosby, Inc. *from* Leider AS, Eversole LR, Barkin ME. Cystic ameloblastoma. A clinicopathological analysis. *Oral Surgery, Oral Medicine, Oral Pathology, Oral Radiology and Endodontics* 1985; 60:624–630.)

Cahn (1933) is generally credited as the first to propose that an ameloblastoma could arise in a dentigerous cyst (Kahn, 1989). This theory found wide acceptance and several authors sought to describe the factor(s) that could be the initiating event in stimulating the ameloblastomatous transformation of the cyst lining. Kahn (1989) reported that some of the theories proposed over the years as the initiating event(s) included:

i) non–specific irritational factors such as extraction, caries, trauma, infection, inflammation, or tooth eruption;

ii) nutritional deficit disorders; and

iii) viral pathogens (polyoma virus, Epstein Barr virus, and human papilloma virus).

Shear and Singh (1978) have shown that the age–standardized incidence rates of ameloblastoma and dentigerous cyst differs. The age–standardized incidence rate of ameloblastoma was much higher in their sample of South African blacks than whites and conversely, that dentigerous cysts were much more common in whites. This made it unlikely, they, suggested, that dentigerous cysts predisposed to ameloblastoma formation.

Furthermore, neither Gardner (1981) nor Ackermann and co–authors (1988) could find histological evidence to support the ameloblastomatous transformation of odontogenic cyst lining. The latter group preferred the concept of the unicystic ameloblastoma arising *de novo*. Li and co–workers (1995) also concluded that they developed *de novo*. In their study referred to above, they compared the PCNA expressions in cystic tumour linings with published data on odontogenic cyst linings. The activity of cystic tumour linings was significantly different from those of the three main types of odontogenic cysts. All areas of cystic tumour epithelium contained significantly more PCNA positive cells than dentigerous cyst linings. This favoured the concept that unicystic ameloblastomas were *de novo* cystic neoplasms. When compared with the odontogenic keratocyst linings, the cystic tumour and odontogenic keratocyst linings had similar numbers of positive cells but their distribution differed.

Salo and co–workers (1999) using immunohistochemistry and *in situ* hybridization investigated the expression of the extracellular matrix protein laminin–5 in ameloblastomas and human fetal teeth. The tissue samples consisted of different types of ameloblastoma including the unicystic variant. In ameloblastomas, the immunoreaction for the laminin–5 gamma 2 chain was confined to the tumour cells of the peripheral area. Some peripheral epithelial cells and some invading small ameloblastoma cell islands showed intense

intracellular staining for the gamma 2 chain. They concluded that laminin–5 might contribute to the infiltrative and progressive growing potential of ameloblastomas.

AIM OF THE PRESENT STUDY

1. To appraise critically the lesions diagnosed as unicystic ameloblastomas in the Department of Oral Pathology at the University of the Western Cape; and

2. To try, by studying and comparing radiographs and histological sections of a series of these unicystic ameloblastomas, to clarify the apparent contradiction of the concept of a multilocular unicystic ameloblastoma.

OBJECTIVES

To attain these aims, the following objectives were identified:

1. To do a critical analysis of the literature.
2. To analyse the sample according to:
    a) age, gender, and race of the patient;
    b) the anatomical site of the lesion;
    c) the size of the lesions measured from orthopantomograms submitted with the specimen;
    d) the referring hospital. There are three major hospitals that submit specimens to the Department of Oral Pathology at the University of the Western Cape. These are Groote Schuur Hospital in Cape Town; King Edward VIII Hospital in Durban; and Frere Hospital in East London.

3. To assess the clinical features of the lesions such as:

   a) site;

   b) swelling – intraoral and extraoral;

   c) expansion of the cortices of the mandible or maxilla. There may be buccal/labial or lingual/palatal expansion or both;

   d) mobility and vitality of the teeth that are involved in the lesion;

   e) the presence or absence of parasthesia or anaesthesia especially in mandibular lesions as they encroach on or displace the mandibular canal.

4. To describe the radiological features taking the following features into consideration:

   a) the specific anatomical sites involved;

   b) the radiolucent, radio–opaque or mixed nature of the lesion;

   c) the margins of the lesion which may be distinct, indistinct, corticated or scalloped;

   d) the unilocular or multilocular nature of the lesion;

   e) the effect on the teeth involved, such as root resorption or tooth displacement;

   f) the presence of associated impacted teeth and their relationship with the lesion;

   g) the effect on the mandibular canal which may be displaced to the inferior border.

5. To verify the histological diagnoses and to classify the lesions into Groups 1, 2 and 3 as described by Ackermann *et al* (1988).

MATERIALS AND METHODS

This is a retrospective study analysing the unicystic ameloblastomas in the archives of the Department of Oral Pathology of the Faculty of Dentistry, University of the Western Cape, since the inception of the biopsy service in 1977 to 1999 inclusive.

Seventy–six cases of unicystic ameloblastomas were retrieved from the archives. Only cases with complete records were included in the study. Completed records were considered to comprise:

- the information submitted by the referring clinician;
- the original radiograph or a copy of the original;
- the macroscopic description of the lesion received in the laboratory; and
- the slides for histological assessment.

The original referring notes and the slides were readily available in the archives of the Department of Oral Pathology. The radiographs were obtained by searching the files of the Department of Oral Pathology, Maxillofacial and Dental Radiology, and by contacting the referring hospitals (*see Appendices 1 and 2*). Only orthopantomograms were used for the study.

In the above manner, the complete records of 28 cases of unicystic ameloblastoma were available for the study. These were then appraised critically, taking the following into account:

- the clinical information submitted by the clinician;
- the radiological features from the orthopantomograms submitted by the clinicians; and
- the histological features.

The information was transcribed onto tables and the appraisal addressed the features outlined in the list of objectives, *vide supra*. Data were recorded on a Microsoft Excel Spreadsheet and statistical analyses were done where relevant and are referred to under each result.

RESULTS

(a) ***Referring Hospital***

The cases were obtained from six hospitals in South Africa and there was one case submitted by a surgeon in private practice. The six hospitals are (Fig.8):

i) Groote Schuur Hospital in Cape Town. The Maxillofacial and Oral Surgery Unit of this institution is a satellite clinic of the Faculty of Dentistry of the University of the Western Cape.

ii) Livingstone Hospital in Port Elizabeth.

iii) Faculty of Dentistry of the University of the Western Cape.

iv) King Edward VIII Hospital in Durban.

v) Conradie Hospital in Cape Town – at the time of submission of the specimen from here, this institution housed a satellite clinic for the Maxillofacial and Oral Surgery Unit from Groote Schuur Hospital and the University of the Western Cape.

vi) Frere Hospital in East London.

The highest number of cases, 11 of 28 or 39.3 percent of the cases was submitted from Groote Schuur Hospital. When one includes the Faculty of Dentistry of the University of the Western Cape, and its satellite clinics, the submissions increase to 15 of 28 cases or 53.6 percent.

Six cases (21.4 percent) were submitted from King Edward VIII Hospital and three cases each or 10.7 percent from Livingstone and Frere Hospitals. One case was from a surgeon in private practice in Cape Town.

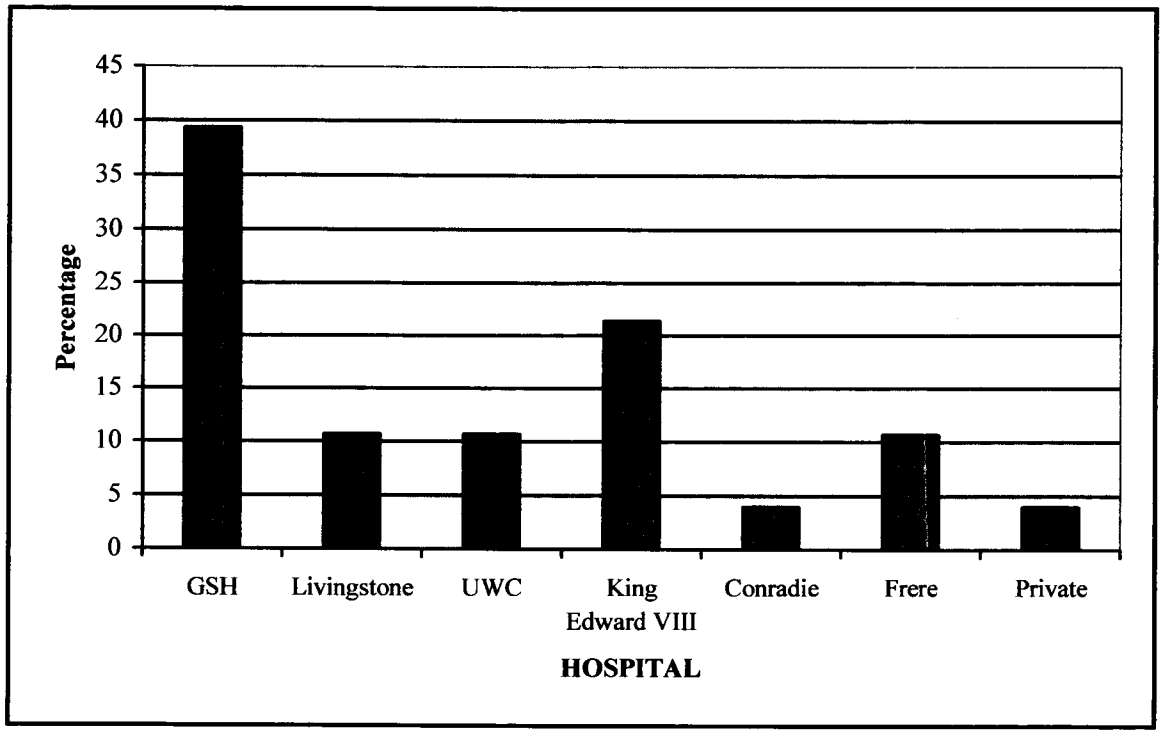

Figure 8: Referring Hospital

(b) Gender Distribution

The gender distribution is 64 percent (18 patients) male and 36 percent (10 patients) female, giving a male to female ratio of 1.8:1 (Fig.9).

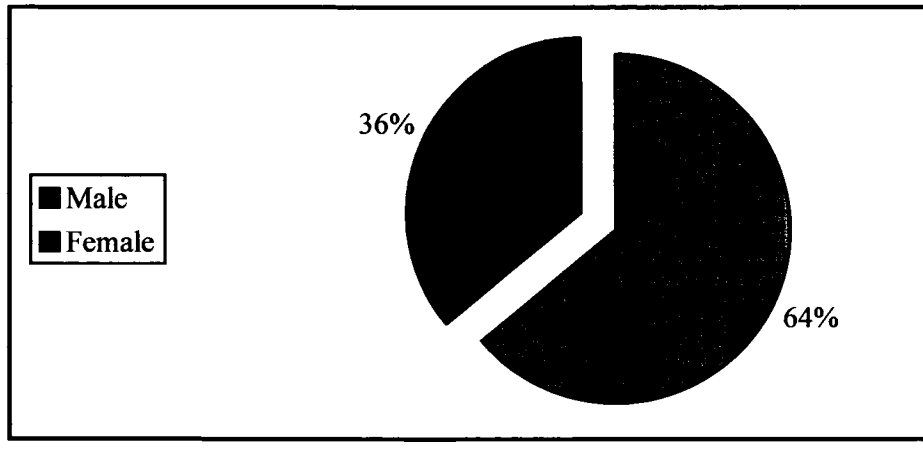

Figure 9: Gender Distribution

When the gender of the patients was considered separately for the dentigerous[1] and non–dentigerous variants of the lesions the following emerged: (i) there were 11 dentigerous variants of unicystic ameloblastoma in the sample population – seven of these patients were male and four female giving a male to femsale ratio of 1.75:1; and (ii) seventeen patients had the non–dentigerous variant of the lesion – there were 11 males and six females giving a male to female ratio of 1.83:1.

(c) ***Age*** (Figs.10–12)

The ages of the patients ranged from eight years to 69 years, with a mean of 22 years (SD ± 13.8) and a median age of 17.5 years. There was a peak distribution in the second decade (Fig.12).

For the impaction associated unicystic ameloblastomas ('dentigerous variant') (n=11) the mean age was 14.8 years (SD ± 5.2) and the median 14 years. The mean age for the 'non–dentigerous variant' (n=17) was 26.7 years (SD ± 15.6) and the median 21 years. The

[1] The dentigerous variety was one in which the crown of an unerupted tooth was enclosed in the cyst cavity and the wall of the cyst was attached to its neck.

difference in the age groups of the two variants was statistically significant at the 95 percent confidence level ($p = 0.05$).

The mean age patients with the apparently multilocular unicystic ameloblastomas was 20 years (SD ± 9) and the median 19.5 years; whereas for the patients with unilocular lesions the mean age was 22.4 years (SD ± 14.5) and median 17.5 years (not significant).

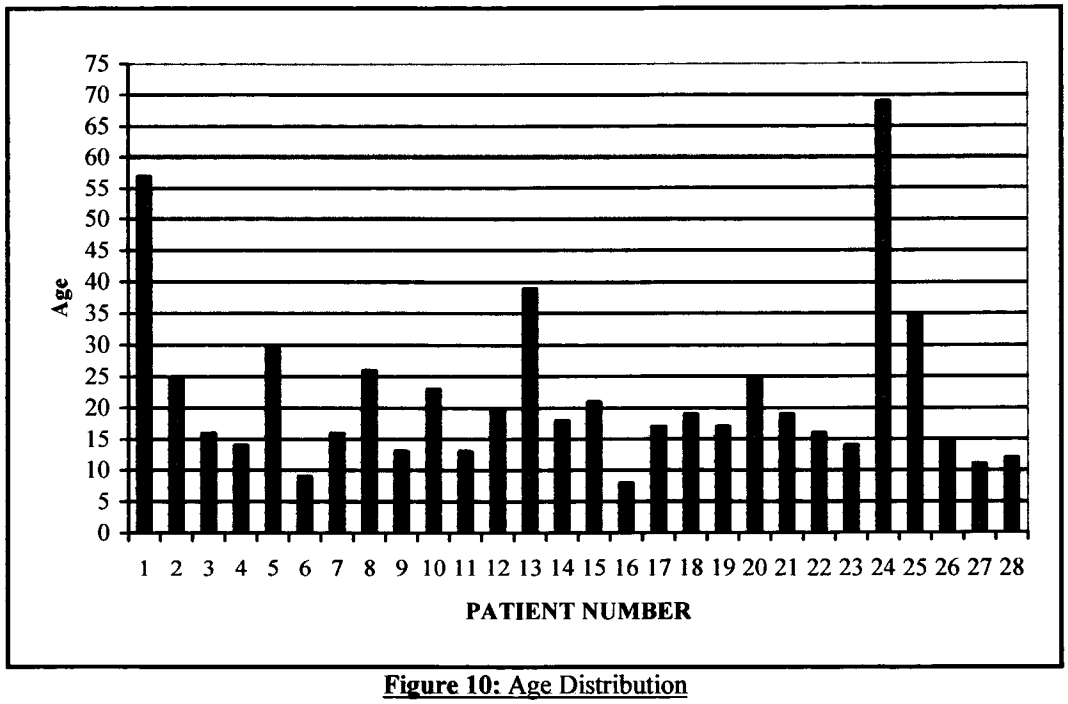

Figure 10: Age Distribution

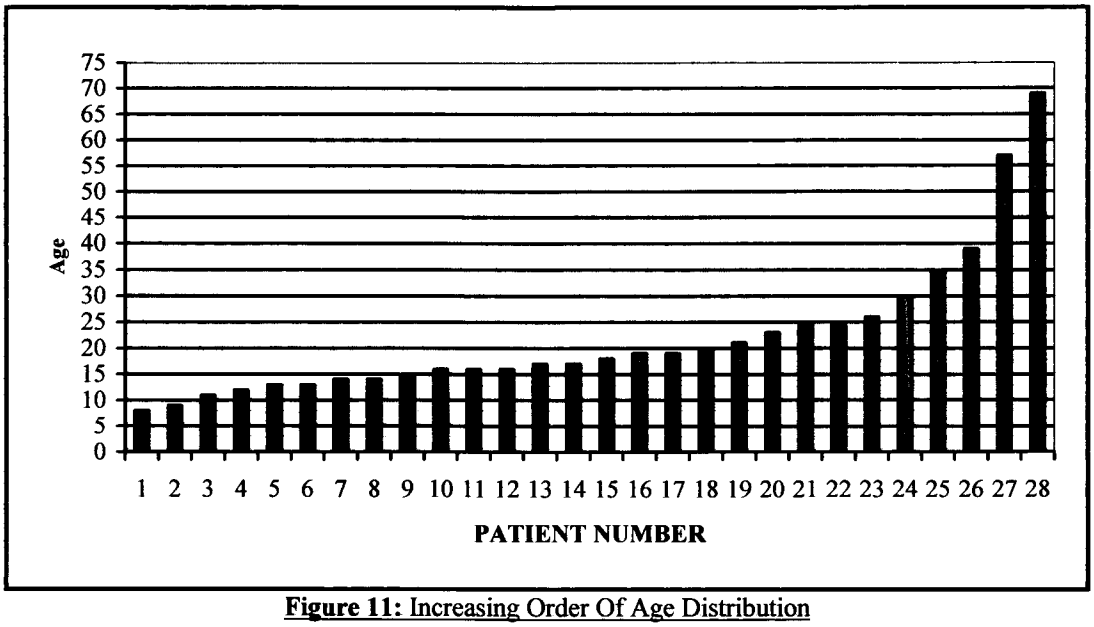

Figure 11: Increasing Order Of Age Distribution

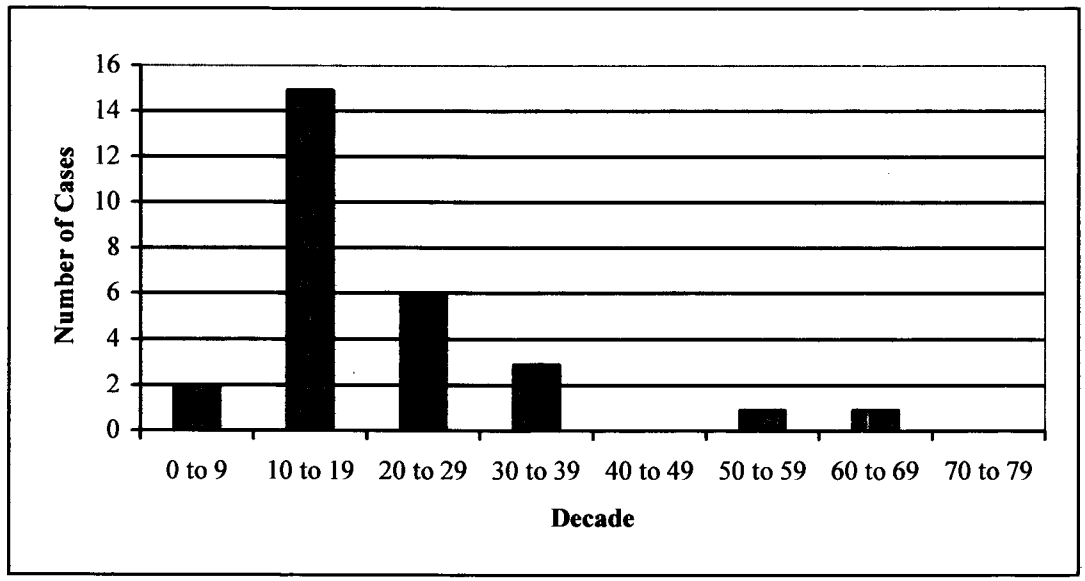

Figure 12: Age Distribution by Decade

(d) ***Racial Distribution*** (Fig.13)

The race of the patients could only be ascertained in twenty–six cases. Fifty–four percent (14 cases) were black, and 42 percent were coloured. There was only one white patient, the case from private practice, and there were no Asian patients in the sample.

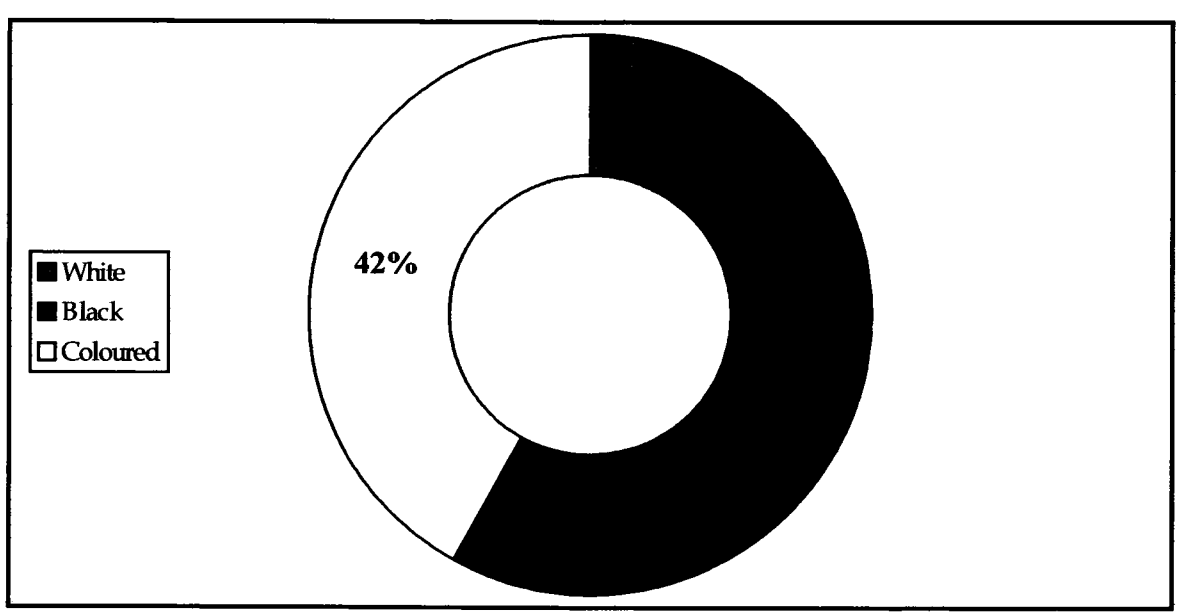

Figure 13: Racial Distribution

(e) ***Site of Occurrence***

The mandible is by far the more common site with 26 cases having occurred in the mandible and only two in the maxilla.

When considering the specific anatomical areas of the mandible or maxilla, it becomes difficult to determine accurately the distribution of the lesions, as most were large involving more than one region.

As a method of determining the anatomic distribution of the lesions within the mandible or maxilla, I used the epicentre of the lesion (as seen on the orthopantomograms) as the site. The lesions were evenly distributed within the mandible – nine each in the body and anterior regions and eight in the angle of the mandible. In the maxilla one lesion occurred anteriorly, and the other in the second quadrant.

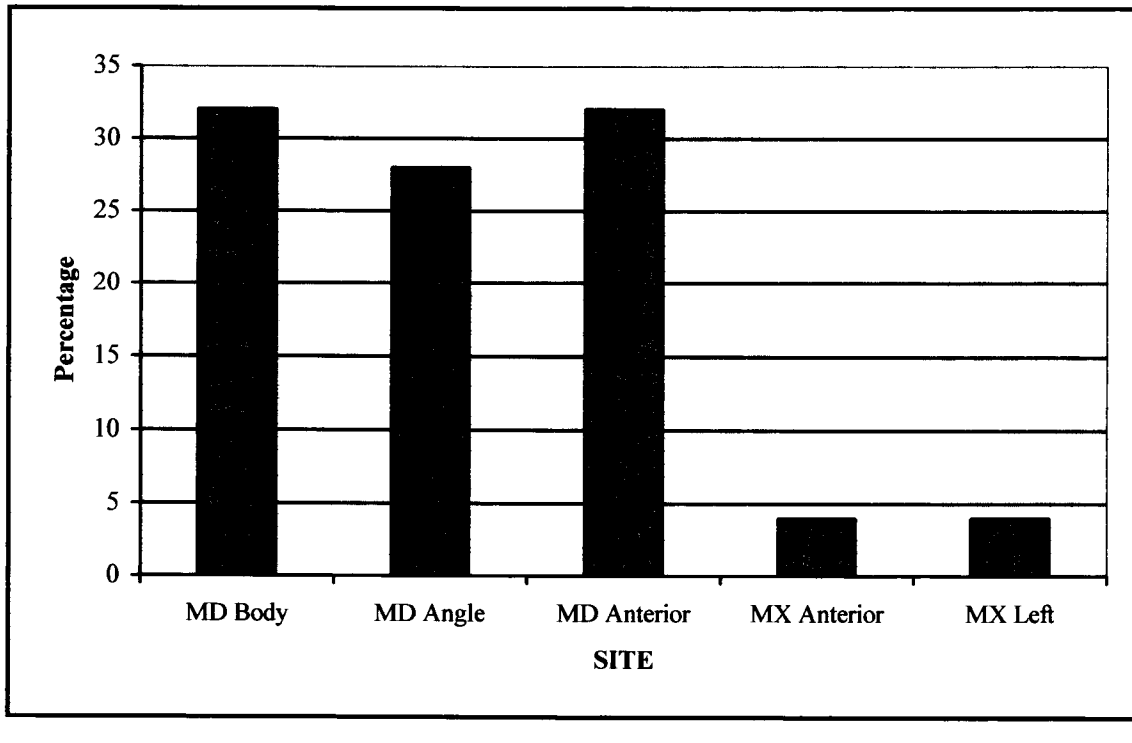

Figure 14: Site of Occurrence (by epicentre)

(f) Size

The sizes of the lesions were measured using the orthopantomograms submitted with the specimens. They varied, by length and height, from 16x20 mm to 126x54 mm. As the orthopantomograms came from different institutions, they cannot be regarded as standardized, and these measurements can be regarded only as a crude guide to the size and variability of the lesions.

(g) Clinical Features

The clinical features were obtained from the information supplied by the clinician. A few clinicians gave detailed clinical descriptions; whereas others submitted minimal information. The following features were noted:

- Swelling occurred in 16 patients – the detail of whether this was internal or external swelling was not recorded by the clinicians.
- Expansion of the affected jaw was noted in 14 patients.
- Mobility of teeth was described in two patients and displaced teeth in four.
- Parasthesia of the mental nerves was noted bilaterally in one patient.
- In eight patients the duration of signs and symptoms was declared and this varied from one month to 12 months. In three other patients the signs and symptoms were described as long standing.
- Pain was a feature in three patients.
- In one patient, who had a lesion in the anterior mandible, the left central incisor was non–vital.

(h) ***Radiological Features***

Margins (Fig.15)

In 16 radiographs (57 percent) the lesions had distinct margins and a further eight lesions (29 percent) had corticated margins. The remaining four lesions (14 percent) had indistinct radiological margins.

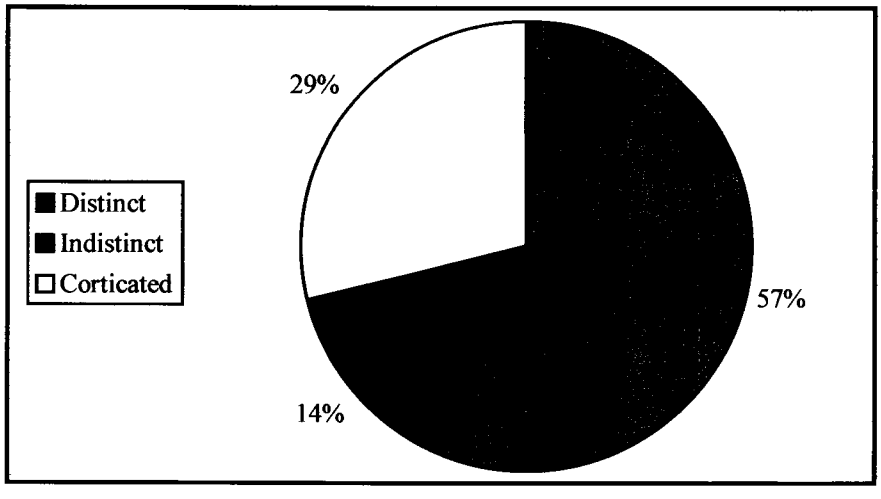

Figure 15: Radiological Margins

Locularity on the radiographs (Fig.16)

Twenty–four or 86 percent of the sample of lesions were interpreted as unilocular. Four or 14 percent of the lesions were interpreted as multilocular, hereinafter referred to as 'apparently multilocular'. When the ages of the patients and the locularity of the lesions were compared, the patients with unilocular lesions had a mean age of 22.4 years (SD ± 14.5) with a median age of 17.5 years

The patients with 'apparently multilocular' lesions were aged 11, 14, 25 and 30 years. The latter sample is too small for meaningful statistical evaluation. The locularity of the lesions was also considered with regard to associated impacted teeth (see Fig.19).

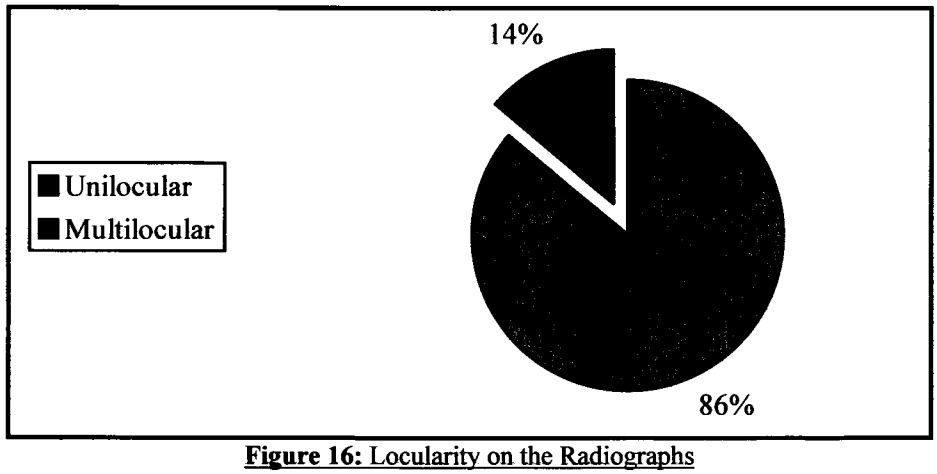

Figure 16: Locularity on the Radiographs

There were 26 dentulous and two edentulous patients in the sample. Of the former, root resorption of adjacent teeth was noted in 14 cases and not seen in 12 cases.

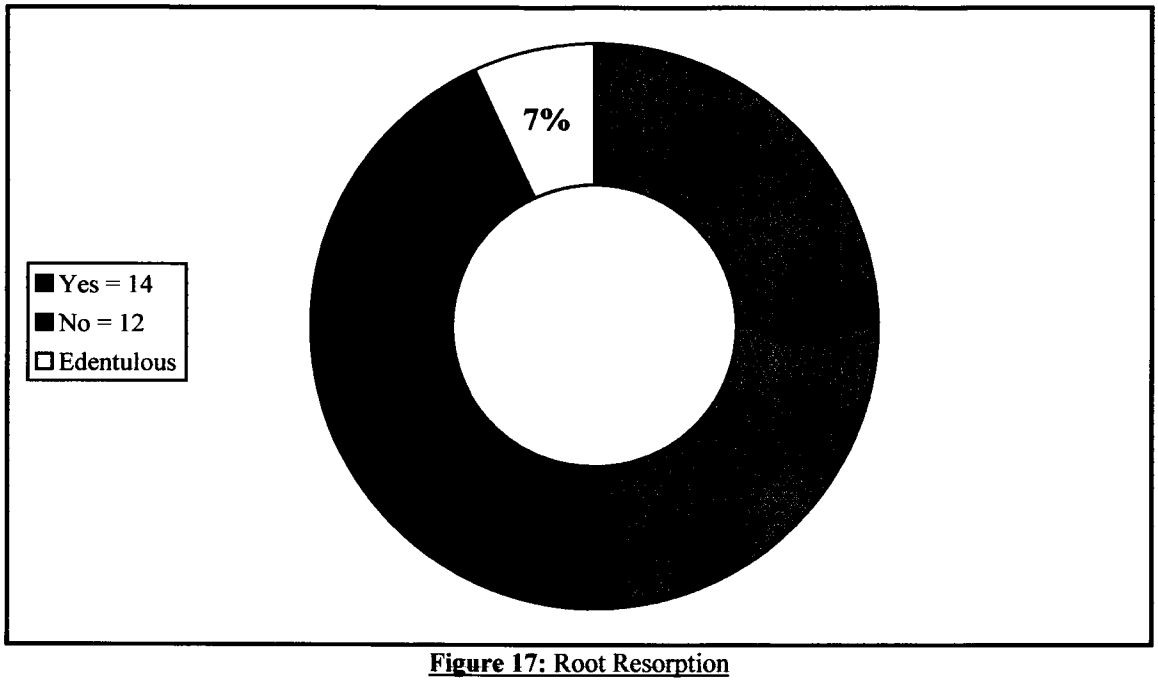

Figure 17: Root Resorption

Tooth displacement (Fig.18)

Tooth displacement occurred in 13 of the 26 dentulous patients.

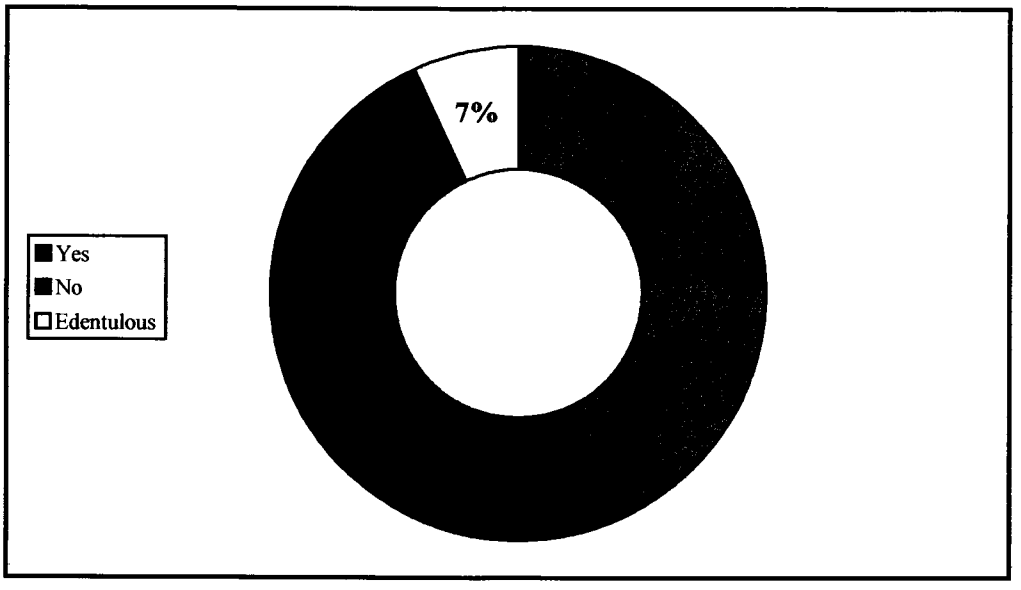

Figure 18: Displacement of Adjacent Teeth

Associated impacted teeth (Fig.19)

Eleven cases had an associated impaction (referred to as the 'dentigerous variant' by Philipsen and Reichart, 1998) and 17 cases did not (the 'non–dentigerous variant').

The age distributions of the patient with the 'dentigerous variant' and those with the other unicystic ameloblastomas differed (*see age on page 28*).

The locularity of the lesions was also considered with regard to associated impacted teeth. The following was noted:

- Two 'apparently multilocular' lesions had no associated impactions.
- Two 'apparently multilocular' lesions had associated impacted teeth.
- Fifteen unilocular lesions had no associated impactions.
- Nine unilocular lesions had associated impacted teeth.

These results were subjected to the Fishers Test, but no specific result was yielded because of the small number of cases in the 'apparently multilocular' group.

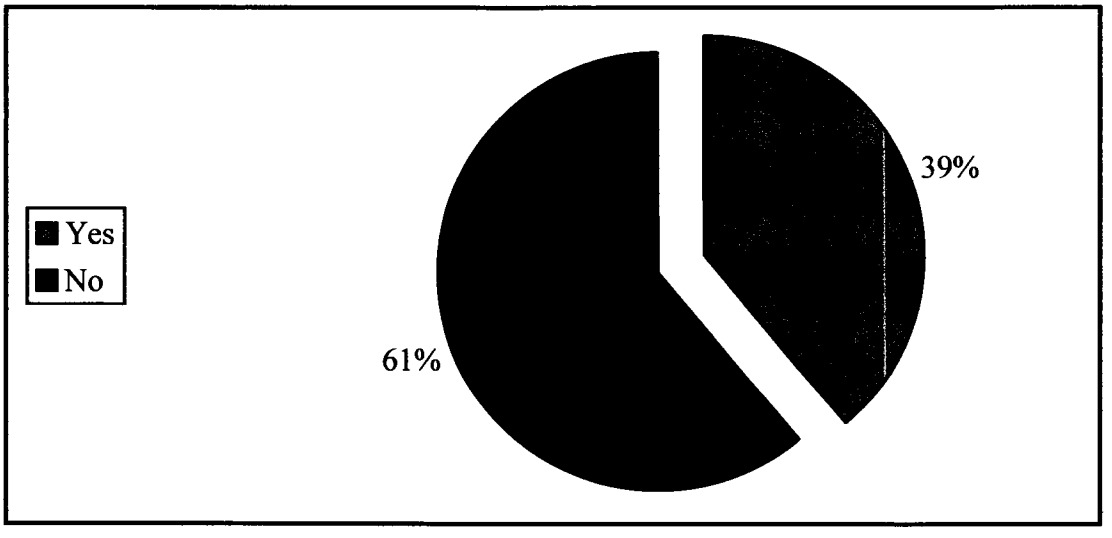

Figure 19: Associated Tooth Impactions

Mandibular canal

The mandibular canal was displaced inferiorly in 11 cases. In another 11 cases, nine of which were in the anterior mandible and the two lesions that occurred in the maxilla, would not have had any effect on the mandibular canal.

Nature of the lesion

Twenty–seven cases were radiolucent and only one showed a mixed radiolucent/radio–opaque lesion.

Radiological type (according to Eversole *et al*, 1984) (Fig.20)

I have attempted to classify all the radiological patterns in the sample according to the six radiological types identified by Eversole *et al* (1984). The breakdown was as follows:

Eversole Type	Number of Cases
B – Extensive, Pericoronal, Unilocular	7
C – Pericoronal, Scalloped	2
D – Periapical, Unilocular	12
E – Interradicular	2
F – Multilocular	4

One case was unclassified; this was a lesion in an edentulous patient. The other lesion in an edentulous patient was classified as a multilocular lesion and therefore included as an Eversole type F lesion.

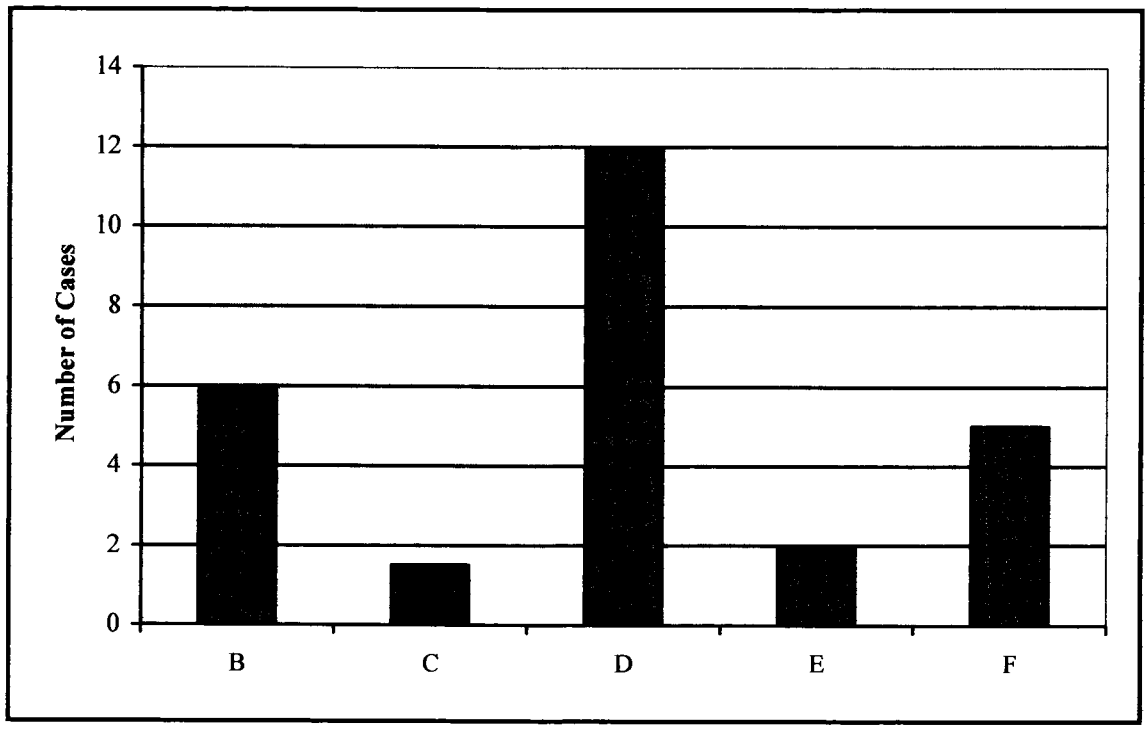

Figure 20: Radiological Type (according to Eversole et al, 1984)

(i) Histology

The sections of all the cases in the sample were reviewed and the lesions classified according to the classification proposed by Ackermann *et al* (1988). The results were as follows:

Histological Classification	Number of Cases
Group I	4
Group II	6
Group III	18

All the results were subjected to statistical analysis using Microsoft Excel. Specific tests used included the 2 x 2 analysis (Chi–Square Test) and the Fishers Test.

DISCUSSION

The sample consisted of 28 cases with complete records collected from a total of 76 cases diagnosed as unicystic ameloblastomas in the archives of the Department of Oral Pathology at the University of the Western Cape. In a worldwide literature survey Philipsen and Reichart (1998) reviewed 193 cases of unicystic ameloblastoma, with the largest series of 57 cases reported by Ackermann, Altini and Shear in 1988. The addition of 28 cases to this 193 in the literature thus far will supplement this number of documented cases by 14 percent.

This is a retrospective study and as such suffered from the limitations of such a study. These included:

1. Some of the data were obtained from information submitted by the clinicians. In many instances this lacked important clinical information.
2. Complete records, particularly radiographs, were not available for most cases.

Gardner (1999) emphasized the limitation of such a retrospective study, but also pointed out that while a great deal of additional knowledge would come most effectively from a large long term prospective study, the difficulties in establishing such a study were formidable, and it could be 20 years before meaningful data was collected.

(a) Referring Hospital

The cases were submitted from six hospitals in South Africa and there was one case submitted by a surgeon in private practice.

Fifteen of the 28 cases (53.6 percent) were submitted from the various clinics of the Maxillofacial and Oral Surgery Department of the University of the Western Cape. These clinics are at Groote Schuur Hospital; the Oral Health Centre; and at the time Conradie Hospital. Six cases (21.4 percent) were from King Edward VIII Hospital and three cases (10.7 percent), each, from the Livingstone and Frere Hospitals.

No demographic conclusions could be drawn from this distribution of cases as I was able readily to access the records of the Faculty of Dentistry and Groote Schuur Hospital. Although letters requesting outstanding radiographs were sent to the Maxillofacial and Oral Surgery Departments at both King Edward VIII and Frere Hospitals, there was a limited response from the former and none from the latter. This has skewed the relative distribution of the cases.

Only one case was submitted by a surgeon in private practice. That this number is not higher is not surprising as private pathology services are available.

(b) Gender Distribution

The gender distribution of 64 percent male and 36 percent female, which represents a male to female ratio of 1.8:1 is higher than the 1.3:1 in the review article by Philipsen and Reichart (1998). The latter authors went further to calculate the male:female ratio for the 'dentigerous' type of unicystic ameloblastoma as 1.5:1 and for the 'non–dentigerous' type as 1:1.8.

In my study the male to female ratio for the 'dentigerous' and 'non–dentigerous' variants reflected that of the overall sample population – 1.75:1 and 1.83:1 respectively. This differs from the findings of Philipsen and Reichart (1998) especially with regard to the 'non–

dentigerous' variant. They did not speculate as to why (in their findings) the 'non–dentigerous' variant might be more common in females.

(c) Age

The ages of the patients correlated very closely with the other South African study by Ackermann *et al* (1988). The patients in our sample ranged from eight years to 69 years, with a mean at the time of diagnosis of 22 years (SD ± 13.8) and a median of 17.5 years. Ackermann *et al* (1988) reported the mean age at diagnosis as 23.8 years (SD ± 14.9) with a range from six years to 77 years.

Philipsen and Reichart (1988) reported that almost 20 years separate the mean age of the 'dentigerous' from the 'non–dentigerous' variant (16.5 years versus 35.2 years). My study confirmed this difference with a mean age for the 'dentigerous' variant as 14.8 years and 26.7 years for the 'non–dentigerous' variant ($p = 0.05$).

In view of the small numbers of the 'apparently multilocular' group no meaningful age difference can be deduced between this group of patients and those with unilocular lesions (mean ages of 20 years and 22.4 years respectively). Eversole *et al* (1984) reported a difference of approximately five years in the mean ages of these two groups of patients (29.4 years for multilocular lesions against 24.3 years for unilocular lesions). They had 10 'apparently multilocular' lesions in a total sample of 31 cases.

(d) Racial Distribution

The race of the patients could only be ascertained in 26 cases as a few of the submitting clinicians did not declare this information. Fifty–four percent were black; 42 percent coloured and four percent white. There were no Asian patients among the 26. Ackermann *et al* (1988)

reported that 89.5 percent of their sample of patients were black. Shear and Singh (1978) found the incidence of all ameloblastomas on the Witwatersrand to be very much higher in blacks than in whites.

The present study and the two other South African studies cited above differ, with regard to racial distribution, from the findings of Leider and co–authors (1985). They reported a distribution of 45 percent white, 33 percent black, 12 percent Hispanic, and 10 percent Oriental. This distribution conforms to that of the general population in the greater San Francisco Bay area.

My study with 54 percent black patients differs from that of Ackermann and co–workers (1988) with 89.5 percent. The reason for this is probably the different demographic distribution between the Western Cape and the region previously known as the Witwatersrand, where the Ackermann study was done.

(e) Site of Occurrence

The majority of the lesions, 92.9 percent, occurred in the mandible. This is in keeping with other reports that have shown a marked preponderance for the mandible. Leider *et al* (1985) reported that all of their 33 cases of unicystic ameloblastoma occurred in the mandible. Ninety–two percent of the series of Ackermann *et al* (1988) occurred in the mandible as did all of the 21 cases of Olaitan and Adekeye (1997).

Gardner (1984) reported that the unicystic ameloblastoma appeared to "occur exclusively in the mandible, where they have a distinct predilection for the third molar region". In their 1998 review, Philipsen and Reichart supported this view that the posterior mandible was the single region most often affected. In my study the distribution of the 26 lesions within the

Sampson and Pogrel (1999) attempted to draw up what they called a 'treatment algorithm for mandibular ameloblastomas' (all types). They suggested that if the lesion was greater than 1cm on plain film radiographs then a computed tomography scan was indicated, and if the lesion was less than 1cm then one could proceed with surgery. The difficulty I have with this is that I have not seen nor read about ameloblastomas that are less than 1cm in its widest dimension. These small lesions could easily be treated by an excision biopsy.

(g) Clinical Features

The most commonly reported clinical feature was swelling of the affected side of the face. This was seen in 16 patients. 'Expansion' was reported in 14 patients by the submitting clinicians. Expansion of the lingual cortex was mentioned specifically in only four patients. Lingual expansion has been widely considered to be a feature that distinguished the unicystic ameloblastoma from odontogenic cysts and was a prominent feature of the study by Olaitan and Adekeye (1997) who noted buccal and lingual expansion in 85.7 percent of cases; whereas buccal plate expansion alone was seen in only 14.3 percent of cases. Hence it is surprising that this feature was reported in so few cases; or perhaps it was not looked for, or was omitted from the clinical description.

The duration of the swelling and expansion varied between one and 12 months, but in three patients was mentioned just as long–standing. These long 'waiting periods' before seeking help were not surprising as many patients live in rural areas and are unable to get to suitable treatment facilities timeously. Olaitan and Adekeye (1997) reported that swelling, ranging in duration from two months to eight years was the principal finding in all their cases. Tharanon *et al* (1999) analysed 184 cases of ameloblastoma in Thailand. The most common complaint was facial deformity (54.3 percent) and 7.1 percent of the patients presented for treatment more than five years after first noticing the disease.

The features of mobility of teeth (in two patients); displaced teeth (in four cases); parasthesia/anaesthesia of affected nerves (noted bilaterally in one patient); pain (in three patients); and vitality of teeth (a non–vital lower incisor in one lesion in the anterior mandible), were only rarely described by the submitting clinicians. This represents a major shortcoming of this type of retrospective study in that certain useful information might not have been declared.

The mobility and displacement of teeth was expected in lesions that involved the tooth–bearing regions of the jaws. The vitality of all the teeth that are apparently involved in a cystic lesion should be tested as this gives important information about the potential source of the lesion.

The presence of parasthesia/anaesthesia of the regional nerves is often difficult to ascertain especially when language barriers exist between the patient and the clinician. Parasthesia of the mental nerve distribution is an unusual feature of the unicystic ameloblastoma and was not mentioned in the large studies by Ackermann *et al* (1988) and Olaitan and Adekeye (1997); nor in the case reports of Rittersma, Hadders and Feenstra (1979); Isacsson *et al* (1986); and Haug *et al* (1990).

Pain might be present if the lesion is infected or causes pressure on adjacent structures. In this study pain was a feature in only three patients. In the case reports of Haug *et al* (1990) one of the two patients presented with pain. In that case the lesion was infected as pus exuded from an extra–oral communication.

(h) ***Radiological Features***

Margins

In sixteen patients (57 percent) the lesions had distinct margins, corticated margins were noted in eight cases (29 percent), and in four cases (14 percent) the margins were indistinct. Neville *et al* (1995) described the radiological margins of the unicystic ameloblastoma as a circumscribed or sharply defined radiolucent area.

Of the four cases with indistinct margins, two occurred in the anterior mandible, an area that is sometimes unclear on the orthopantomograms; one case occurred in the maxilla in an eight year old child. Here the radiographic margins were probably affected by the dental follicles present. The last case was a poor radiographic image.

If the radiological outline of lesions that usually have distinct or corticated margins is indistinct, one might have expected inflammation or infection of the lesion. None of these four lesions had histological features of inflammation or infection. Therefore it is likely that these indistinct margins were the result of poor radiographic technique.

Locularity on radiographs

Twenty–four patients (86 percent) had lesions that were unilocular. The remaining four cases (14 percent) were 'apparently multilocular'.

The terms unicystic, multicystic, unilocular and multilocular

At this stage of the discussion, it is important to clarify the terms unicystic, multicystic, unilocular and multilocular. Reichart *et al* (1995) pointed out problems in the nomenclature of these terms. 'Unilocular' and 'multilocular' are radiological terms and are often confused

with, or used interchangeably with, the histological terms 'unicystic' and 'multicystic'. It may be difficult for many people to conceive that an apparent multilocular lesion may in fact be a unicystic ameloblastoma. Gardner (1999) shared that difficulty. Neville *et al* (1995) defined the terms 'unilocular' and 'multilocular' as follows: "unilocular – describing a radiolucent lesion having a single compartment; multilocular – describing a radiolucent lesion having several or many compartments".

Gardner (1999) stated that "a true multilocular lesion, that is one composed of numerous separate compartments or cysts, cannot by definition be a unicystic ameloblastoma. A terminological problem exists in that a lesion that appears clinically and radiologically to occupy a single cavity, but which has an irregular, scalloped border, is sometimes referred to erroneously as being multilocular. Such a lesion can be a unicystic ameloblastoma." Shear (1992) stated that "the lesions were either well corticated unilocular radiolucencies or showed trabeculations which may lead to an erroneous diagnosis of multilocular cyst". Shafer, Hine and Levy (1983) pointed out that the radiographic film indicated only the relative presence or absence of calcified tissue.

My feeling is that this controversy exists because of the limitations of viewing a three–dimensional structure in a two–dimensional image. The differential resorption of the bone by the lesion can lead to scalloping of the margins and over a period of time produce ridges and craters which can become more prominent, giving rise to incomplete septa. The resultant radiographic image will appear to be multilocular whereas the lesion is in fact unicystic. Diagrammatically this can be expressed as follows:

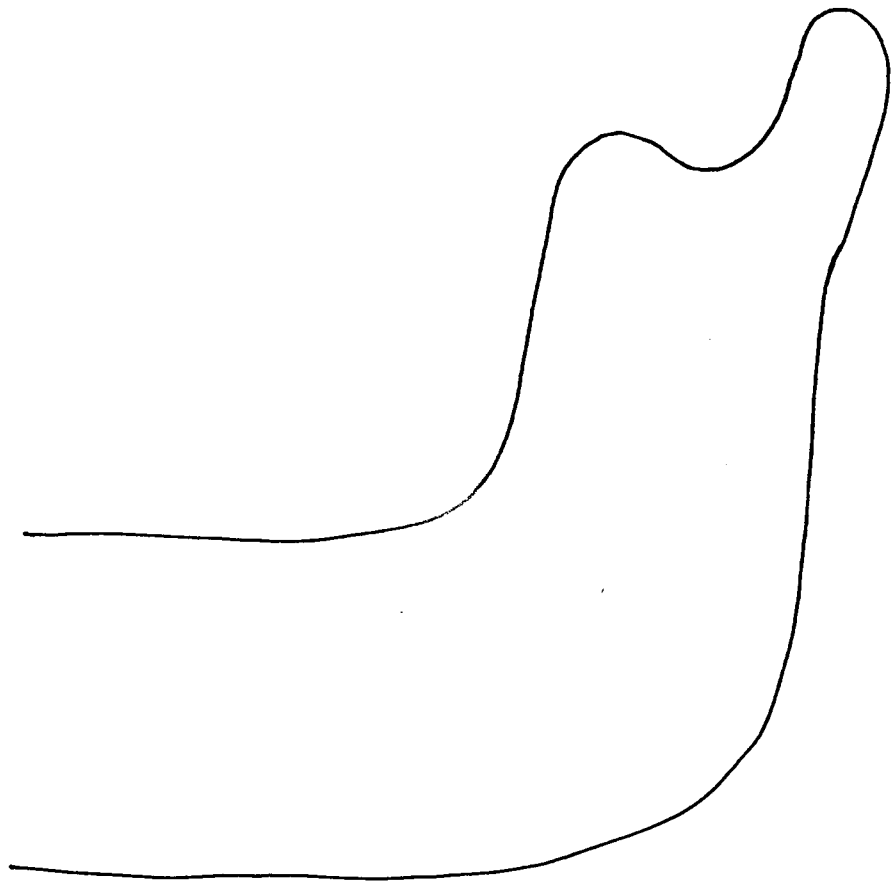

Figure 21: The multilocular appearance of a lesion in the mandile.

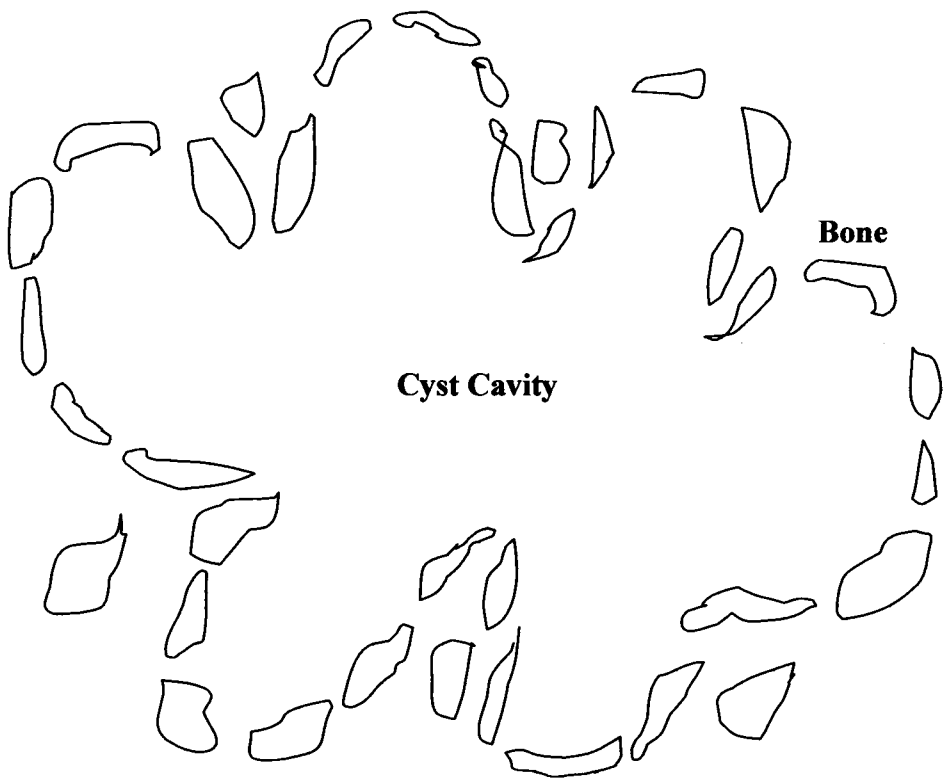

Figure 22: Cross–section of a unicystic lesion with differential areas of bone resorption.

Furuki *et al* (1997) discussed the radiological features of three recurrent unicystic ameloblastomas. The first obvious sign of recurrence was a scalloping of the sclerotic margin of the lesion. This might be further evidence of differential growth rates of the lesion, as one would expect in a neoplasm compared with a benign cyst. This scalloping later progressed to give a soap bubble or honeycomb appearance in all their cases. They postulated that the multilocular pattern of recurrence results from multicentric proliferation of the tumour. Delbalso (1990) distinguished between the terms 'honeycomb' and 'soap bubble'. 'Honeycomb' was used to describe a multilocular lesion in which the locules may be smaller than 1cm in diameter and numerous. 'Soap bubble' is used to describe a multilocular lesion in which larger locules which tend to be fewer in number, and expansion is invariably present.

In the present study the mean and median ages of the patients with unilocular lesions were very close to those of the patients with multilocular lesions (discussed earlier).

The association between locularity of the lesions and impacted teeth, was also considered. Two multilocular lesions had no associated impactions; two multilocular lesions had associated impactions; 15 unilocular lesions had no associated impactions; and nine unilocular lesions had associated impacted teeth. These differences were not statistically significant (Fisher's Test). A predictable assumption was that the presence of an associated impacted tooth did not influence the locularity of the lesion.

Using the histological classification of Ackermann *et al* (1988), three of the four multilocular lesions were group 3 lesions and the other was a group 1 lesion. One could postulate, as Furuki *et al* (1997) did, that multicentric activity of the ameloblastoma epithelium in the

walls of the group 3 lesions may have given rise to the multilocular appearance. The radiological features of the remaining group 1 lesion might have been due to differential growth rates of specific regions of a large cyst.

Langlais (1990) stated that with the cystogenic ameloblastoma (= unicystic ameloblastoma) "although some were unilocular, others formed incomplete locules; thus, the peripheral outline was scalloped, with few bony septa within the central portion. In this case the lesion resembled an odontogenic keratocyst but without the cloudiness of the lumen." This could easily be interpreted as a multilocular lesion.

Root resorption, displacement of teeth and mandibular canal

The resorption of the apex of one or several teeth in association with a lesion is a sign of a benign process (Langlais, 1990). Of the six radiological patterns reported by Eversole *et al* (1984), two were associated with root resorption – the unilocular periapical radiolucency, and the periapical multilocular radiolucent lesion.

In this study root resorption was seen in 14 of the 26 (53.8 percent) lesions that occurred in dentate individuals. This supports numerically the study of Roos *et al* (1994). They reported root resorption in 13 of their 30 cases. It is difficult to compare actual percentages as they do not state if any of their patients were edentulous. Numerous other papers have reported the resorption of the roots of related teeth as a radiological feature of the unicystic ameloblastoma (Shear, 1995; El–Abdin and Ruprecht, 1988), and root resorption in ameloblastomas is also well documented (Struthers and Shear, 1976).

Figure 24: A unicystic ameloblastoma causing root resorption.

The displacement of adjacent or impacted teeth and of the mandibular canal are considered to be radiological signs of a benign lesion. Thirteen of the 26 (50 percent) lesions in dentate patients in this study displayed tooth displacement. Roos *et al* (1994) also reported a 50 percent frequency of tooth displacement in their series. However, as already mentioned we do not know how many of their patients were edentulous.

Figure 25: Unicystic ameloblastoma causing tooth displacement.

In my study 11 lesions showed both root resorption and tooth displacement. It is difficult to speculate why some unicystic ameloblastomas will show:

(i) root resorption or;

(ii) tooth displacement or;

(iii) root resorption and tooth displacement, or;

(iv) none of the above.

Perhaps this is the result of differential pressure resorption with the lesion taking the path of least resistance. If a lesion resorbs the interdental bone before the roots of the adjacent teeth, growth of the lesion will result in splaying of the roots of these teeth.

Bone type and quality are likely to influence the intraosseous growth of a lesion. Implantologists use the following classification of bone type (Floyd, Palmer and Palmer, 1999):

Type 1 – mainly cortical.

Type 2 – dense cortex and cancellous space.

Type 3 – thinner cortex and less dense cancellous bone.

Type 4 – very thin cortex and sparse bone trabeculae in the medullary spaces.

It would seem that unicystic ameloblastomas occurring in jaws displaying types 1 and 2 bone are likely to have a lesser capacity for resorbing the bone. In these cases roots may be at a greater risk for resorption. The opposite might apply with lesions occurring in jaws with types 3 and 4 bone. The thin and less dense bone would be more easily resorbed, resulting in displacement of adjacent teeth.

The mandibular canal was displaced inferiorly in 11 patients in this study. The epicentre for 10 of these lesions was in the body or angle of the mandible. In the remaining case, the epicentre was in the anterior mandible, but the lesion extended into the left body of the mandible. The displacement of the mandibular canal can only be a feature of lesions that occur in that part of the mandible that houses the canal. The increased density of the bone surrounding the mandibular canal is likely to be more resistant to pressure resorption caused by the enlarging unicystic ameloblastoma. This protects the inferior alveolar nerve within the

canal, and accounts for the absence of parasthesia or anaesthesia along the distribution of this nerve.

Associated impacted teeth

In this study there were 11 cases of the 'dentigerous variant' and 17 of the 'non–dentigerous variant' of the unicystic ameloblastoma. The presence or absence of associated impacted tooth or teeth was considered together with gender distribution, age of patient, and the locularity of the lesions on the radiographs. All these were discussed earlier in the relevant sections.

Nature of the lesion

Twenty–seven lesions were radiolucent. The remaining tumour was described as 'a mixed radiolucent/radio–opaque lesion in the anterior mandible...' This was seen in patient number 25 in my sample. The submitting clinicians reported that the mandibular left central incisor was non–vital and the adjacent right central and lateral incisors were mobile. They suspected a radicular cyst. Histological examination of the lesion revealed a group II unicystic ameloblastoma (according to the classification of Ackermann *et al*, 1988). In addition, there was an intense acute and chronic inflammatory infiltrate.

The unicystic ameloblastoma is radiolucent (Olaitan and Adekeye, 1997), but the intense inflammation in the case mentioned above, probably led to the formation of pus within the lesion. This together with the distortions of the anterior mandible (produced on orthopantomograms) may have resulted in the mixed appearances of this particular lesion.

Radiological type (as described by Eversole *et al*, 1984)

Twenty seven of the lesions in my sample could be classified according to the radiological types described by Eversole *et al* (1984). The distribution was as follows:

Radiological Type	Number of Cases
B – Extensive, Pericoronal, Unilocular	7
C – Pericoronal, Scalloped	2
D – Periapical, Unilocular	12
E – Interradicular	2
F – Multilocular	4

There were no type A (pericoronal unilocular). All the lesions in this study were large and there is no distinct boundary between pericoronal unilocular, and extensive pericoronal unilocular. This allowed for subjectivity in the study and could be considered a flaw of the classification, at least in the population sample in the present study.

Another flaw was detected when I was unable to classify one lesion – a unilocular radiolucent lesion in an edentulous mandible. The categories drawn up by Eversole *et al* (1984) did not allow for an edentulous mandible.

The 11 lesions associated with impacted teeth fell into categories B(7), C(2) and F(2); and the 'non–dentigerous variants' were in categories D(12), E(2) and F(2). This showed a very useful feature of these groupings in that categories A, B and C were the 'dentigerous variants' and D and E the 'non–dentigerous variants'. Group F (as I understand it) was of multilocular lesions irrespective of whether there was an associated impacted tooth or not.

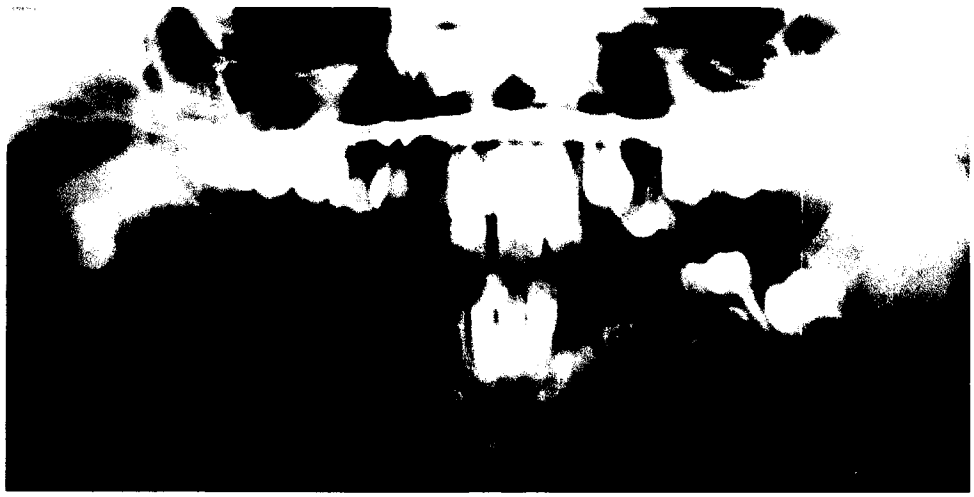

Figure 26: An Eversole type B lesion – extensive pericoronal unilocular.

Figure 27: An Eversole type C lesion – pericoronal scalloped.

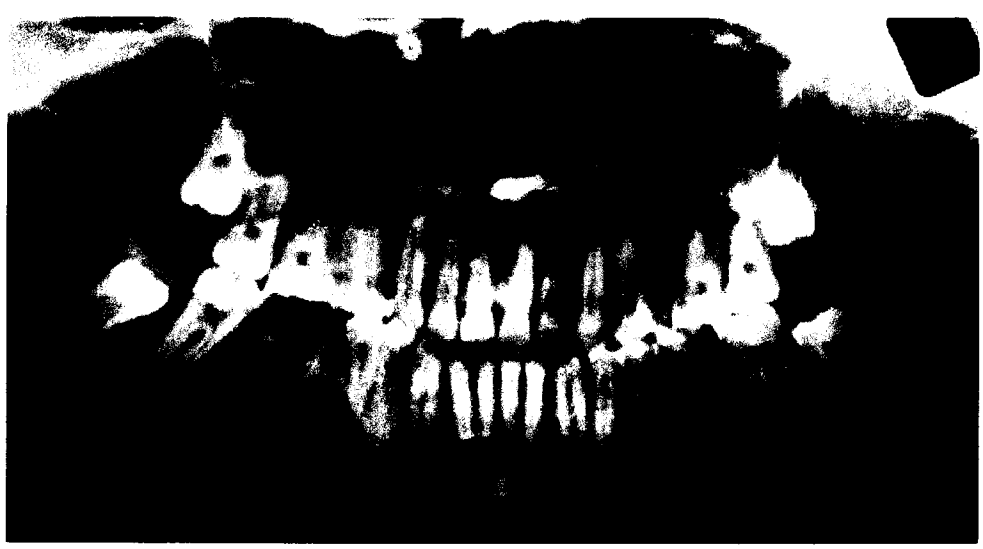

Figure 28: An Eversole type D lesion – periapical unilocular.

Figure 29: An Eversole type E lesion – interradicular.

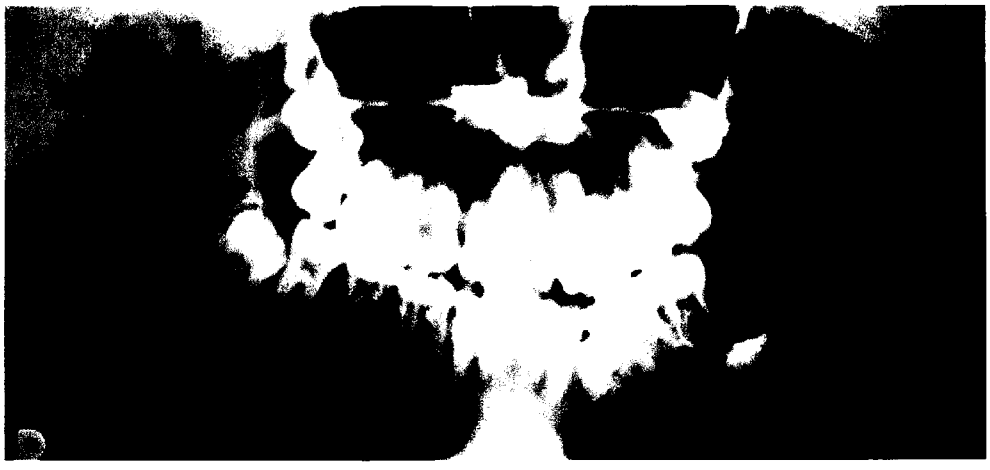

Figure 30: An Eversole type F lesion – multilocular.

Figure 31: Unicystic ameloblastoma in an edentulous mandible – unclassified.

(i) ***Histological Type***

The histology of the lesions was reviewed and grouped according to the classification proposed by Ackermann *et al* (1988). There were four Group 1 lesions (14.3 percent), six Group 2 lesions (21.4 percent) and 18 Group 3 lesions (64.3 percent). These results differed from those of Ackermann *et al* (1988) and Roos *et al* (1994). The differences are shown in the following table:

Table 1: ***Comparison of the histological classifications of unicystic ameloblastoma***

Group	Present Study %	Ackermann *et al*, 1988 %	Roos *et al*, 1994 %
1	14.3	42	50
2	21.4	9	13.3
3	64.3	49	36.6

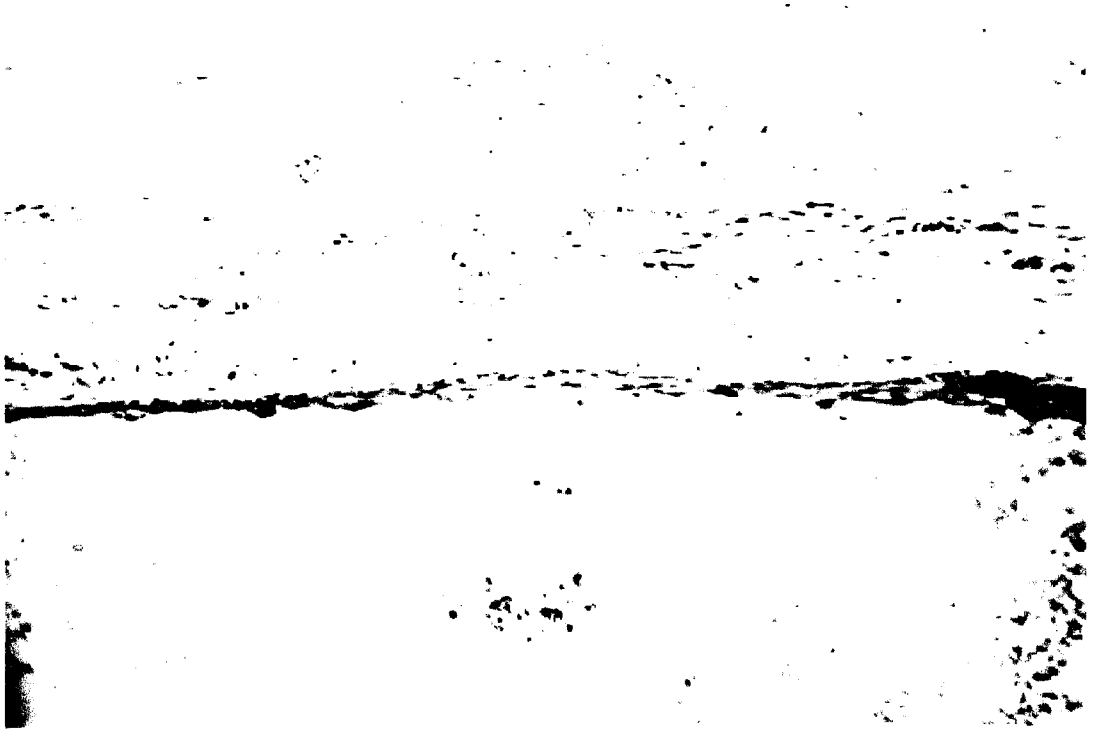

Figure 32: The nondescript epithelium as seen in a Group 1 unicystic ameloblastoma.

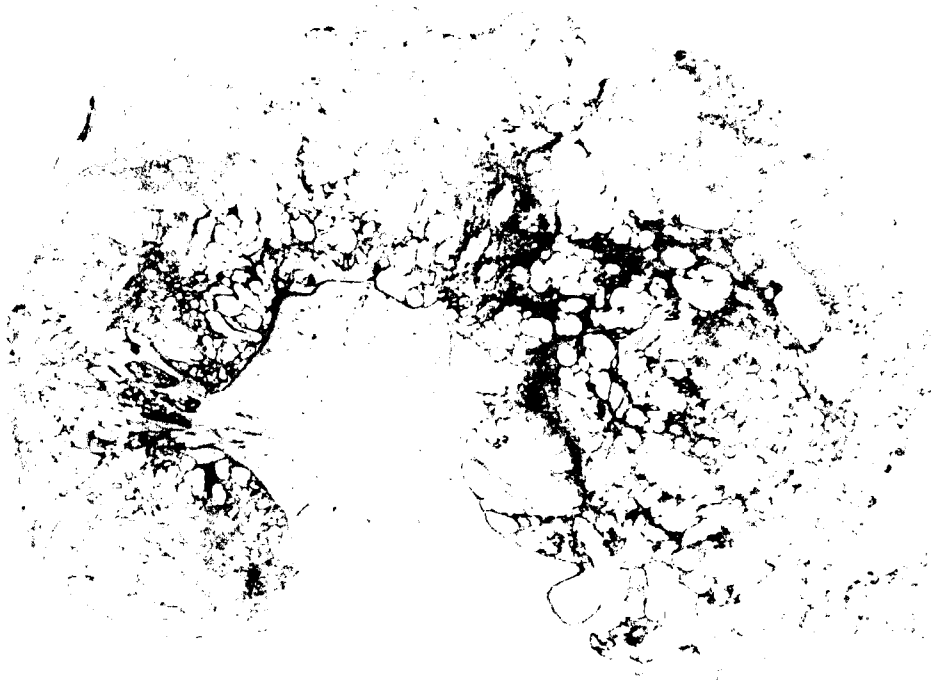

Figure 33: The intraluminal epithelial thickening seen in Group 2 unicystic ameloblastoma (*reprinted with permission from Prof. M. Shear*).

Figure 34: Epithelial islands within the wall of a Group 3 unicystic ameloblastoma.

This study has a low proportion of Group 1 lesions and a high proportion of Group 3 lesions compared with the two other studies. This may arise from differences in interpretation as to when to classify a lesion into Group 3. Ackermann *et al* (1988) described Group 3 as: "The presence in the connective tissue wall of the cyst, of invasive islands of ameloblastomatous epithelium which might or might not be connected to the cyst lining. Mural nodules of tumour tissue similar to that seen in Group 2 may also be present. The cyst lining shows features of ameloblastoma in parts, but usually not throughout." In my opinion this description does not give enough attention to one feature mentioned by Robinson and Martinez (1977), that is, "downgrowth of the epithelium into the connective tissue portion of the cyst wall." I interpreted these downgrowths of epithelium as invasion of the cyst wall and classified them as Group 3 lesions even if there were no distinct epithelial islands in the wall of the lesions.

Figure 35: Downgrowth of epithelium into the cyst wall.

Another reason, perhaps, was that the differences in histological classification may be related to the differences noted in racial distribution of this study and that of Ackermann *et al* (1988). In this study 54 percent of the patients were black and 42 percent coloured; whereas in that of Ackermann *et al* (1988) 89.5 percent of their patients were black. Shear and Singh (1978) stated "there are no grounds for concluding that the racial bias is genetically determined and speculation about possible environmental factors may be more profitable." They (Shear and Singh, 1978) cited the possible relationship between oesophageal carcinoma in black South Africans and environmental carcinogens such cigarette smoking and alcohol consumption (especially illicit beverages which have been found to contain the carcinogen dimethyl nitrosamine or a similar substance). As early as 1968, Herrold showed that odontogenic tumours may be induced in Syrian hamsters which received N–methyl–N–nitrosourea intragastrically. This, however, is only suggestive of environmental factors as the possible aetiology of ameloblastoma, and not of histological differences. The question of whether the factor responsible for the ethnic differences are genetic or environmental, has still to be answered.

(j) Treatment

Robinson and Martinez (1977) reported that of the 17 cases of unicystic ameloblastoma treated by enucleation, only three recurred. Gardner and Pecak (1980) suggested that enucleation with long–term follow–up should be adequate treatment for this lesion, but they cautioned that the posterior maxilla represented a dangerous location for potentially invasive tumour. In such cases, marginal resection should be employed.

Shteyer, Lustman and Lewin–Epstein (1978) reported that the recurrence rate of the mural ameloblastoma after enucleation was less than 10 percent. Gardner (1984) suggested that the "reason for the good prognosis was that, in most cases, the tumour was well localised by the

fibrous capsule of the cyst. Once the tumour had breached the periphery of the fibrous connective tissue capsule, it could have infiltrated the surrounding cancellous bone and behaved like a solid or multicystic ameloblastoma." He suggested that in cases where the fibrous capsule had not been infiltrated, a cure was expected with just enucleation of the lesion. Thereafter periodic examination of the surgical site for at least five, or preferably 10 years was all that was required. The most reliable treatment for lesions that invaded the connective wall was considered to be a marginal resection following the initial enucleation or curettage.

These views were supported by Ackermann *et al* (1988) when they suggested that a unicystic lesion radiologically and at operation should be enucleated *in toto* and submitted for histological examination. Further excision or resection of remaining bone would be necessary in the case of Group 3 lesions. These authors also emphasized that an incisional biopsy is of little value as the true nature of the lesion would only become evident when the entire specimen was available for macro and microscopic examination. Roos *et al* (1994) cautioned that "all unicystic ameloblastomas, irrespective of grouping, are neoplastic in nature and will recur if incompletely removed."

The American Association of Oral and Maxillofacial Surgeons found "that the absence of standardized terminology for methods of excision and the omission of critical details were major deficiencies in the surgical literature on ameloblastoma" (Gold, Upton and Marx, 1991). In an attempt to standardize surgical terminology, Gold *et al* (1991), proposed the following definitions:

1. *Enucleation* – separation of a lesion from bone, with preservation of bone continuity, by virtue of the lesion's containment within an encapsulating or circumscribing connective tissue envelope derived from the lesion or surrounding bone.
2. *Curettage* – removal of a lesion from bone, with preservation of bone continuity, by scraping or morcellation necessitated by the friability of the lesion or absence of an intact encapsulating or circumscribing connective tissue envelope derived from the lesion or surrounding bone.
3. *Marsupialization* – surgical exteriorization of a lesion by removal of overlying tissue to expose its internal surface to the oral cavity, or another body cavity, by excision of a portion of the lesion bordering that surface or cavity.
4. *Resection without continuity defect* – excision of a lesion, including a measurable perimeter of investing bone, without interruption of bone continuity.
5. *Resection with continuity defect* – excision of a lesion, including a measurable perimeter of investing bone, with interruption of bone continuity.
6. *Disarticulation* – special form of resection with continuity defect involving the temporomandibular joint.
7. *Recontouring* – surgical reduction of the size and/or shape of the surface of a bony lesion or bone part.

Williams (1993) suggested that after a clinical and radiological evaluation of a patient suspected of having an ameloblastoma, the decision to perform an incisional or excisional biopsy would be dependent on the size of the lesion and its clinical features. An incisional biopsy would be advantageous if a representative specimen can be obtained. However, if an excisional biopsy is performed and histological examination revealed a unicystic ameloblastoma, the need for further surgery would be determined by:

- the extent of the initial procedure;
- the histological grading of the lesion;
- the age of the patient;
- the size of the lesion and its location; and
- whether there was perforation of cortical bone with soft tissue involvement.

He (Williams, 1993) also suggested that a preoperative computed tomography scan or magnetic resonance imaging would be useful in determining the extent of the lesion.

Feinberg and Steinberg (1996), using definitions of Gold *et al* (1991) stated that the conservative approach to the surgical management of ameloblastoma would include enucleation and curettage, whereas the radical approach included resection (with or without continuity defect) and disarticulation of the temporomandibular joint. They (Feinberg and Steinberg, 1996) discussed the treatment of all types of ameloblastoma according to the anatomic location within the jaws, and suggested the following:

1. *Anterior mandible (cuspid to cuspid)*

Radical resection with continuity defects of the anterior mandible are complex reconstructive cases and if at all possible the lower border should be spared. Furthermore, this region is far from major anatomic structures and thus allows for a more conservative approach to treatment.

2. *Posterior mandible (cuspid to condyle)*

Unicystic ameloblastomas of the posterior mandible could be treated conservatively with curettage or peripheral ostectomy if adequate follow–up is possible. However, if there was invasion into the connective tissue wall a more radical approach was indicated.

3. *Anterior maxilla (cuspid to cuspid)*

The authors do not comment on the unicystic ameloblastoma specifically in this area but suggest that for ameloblastoma in general a less radical approach than for a lesion in the posterior maxilla could be used.

4. *Posterior maxilla (cuspid to pterygoid plates)*

Feinberg and Steinberg (1996) suggested that the relationship of this area to the pterygomaxillary fossa, infratemporal fossa, orbit, and base of the skull made definitive initial management of ameloblastoma mandatory. There was a lack of maxillary cortical bone to contain these tumours which allowed for spread outside the maxillary boundaries. As mentioned earlier, Gardner and Pecak (1980) supported the aggressive treatment of unicystic lesions in this area, whereas Scaccia *et al* (1991) suggested that the unicystic ameloblastoma could be treated more conservatively. Feinberg and Steinberg (1996) suggested a conservative approach to unicystic ameloblastoma in this area, but if there was evidence of connective tissue invasion, then a more radical approach was indicated.

Other treatment modalities for ameloblastoma, in general, that have been reported over the years included radiation, chemical cautery and cryotherapy (Gardner, 1984; Williams, 1993; Feinberg and Steinberg, 1996; and Sampson and Pogrel, 1999). These however, have not found wide acceptance.

Furuki *et al* (1997) reported that they have used marsupialization as a conservative treatment for unicystic ameloblastoma regardless of the histological subclassification. They acknowledged that the literature suggested the forms of therapy mentioned above, but defended their position by stating that they "were able to identify recurrence early because of close follow–up." Yet they give a detailed description of radiological changes in recurrent lesions following marsupialization.

In the Department of Maxillofacial and Oral Surgery of the University of the Western Cape and Groote Schuur Hospital, patients with unicystic ameloblastomas of the mandible are managed in the following manner: a thorough history is taken and a clinical examination performed. The patients are referred for appropriate plain film radiographs. With all lesions a computed tomography scan is obtained.

Smaller lesions (where the lower border of the mandible is easily preserved) are removed *in toto* (enucleation with peripheral ostectomy); and in larger lesions the lesion is marsupialised and the 'lid' sent for histological evaluation. Although it is accepted that an incisional biopsy is only of limited value, this preliminary histological examination does confirm that we are dealing with a neoplasm and not an odontogenic cyst. The lesion is packed with ribbon gauze soaked in bismuth iodoform paraffin paste (BIPP).

The patient is closely followed up and radiographs obtained at periodic intervals (usually every two months). At each follow–up visit the BIPP ribbon gauze is shortened. Once the lesion has decreased in size, the unicystic ameloblastoma is enucleated and a peripheral ostectomy performed. In this protocol, the marsupialization is the first stage in a two stage procedure. Case 27 in this sample is a good example of this. The extensive 'apparently multilocular' lesion was marsupialized and treated in the manner described above. Four

months later the lesion (now much reduced in size) was enucleated and a peripheral ostectomy performed. In this manner an 11 year old child was spared the resection of almost half her mandible. One year after the definitive operation the patient remains free of disease.

There is no protocol for unicystic ameloblastoma in the maxilla as these lesions are very rare.

The data in this study were obtained from the records within the Department of Oral Pathology and as such, details of treatment of all these patients, or of recurrences, were not available.

Figure 36: An example of a unicystic ameloblastoma in the maxilla.

Marsupialization is a decompression of the cyst by creating the largest surgical 'window' into the cyst cavity which is compatible with the surrounding anatomy (Killey, Seward and Kay, 1975). By maintaining a patent 'window' "permanent drainage of the liquid contents results in shrinkage of the cyst lining. What is unclear is whether this is due simply to mechanical

decompression or to the removal of a chemical stimulation of the bone resorptive abilities of the lining, or perhaps to both mechanisms" (Seward, 1992). This applies to odontogenic cysts. The unicystic ameloblastoma is a neoplastic lesion with an inherent growth potential in the lining. It is unclear why there would be a regression in the size of a marsupialized unicystic ameloblastoma.

Furuki *et al* (1997) used marsupialization as the only method of treatment for unicystic ameloblastomas and claim good results. In the Department of Maxillofacial and Oral Surgery at Groote Schuur Hospital/University of the Western Cape, marsupialization is used as the first step in a two step process in the treatment of larger unicystic ameloblastomas. The initial step allows for decompression of the lesion:

- preliminary histological assessment;
- some bone regeneration; and
- a more conservative definitive procedure during the second stage of surgery.

Unfortunately, a thorough audit of all the unicystic ameloblastomas treated in this manner in this department has not been done and may be the subject of a future study. The success rate, therefore, cannot be reported.

Recurrences

In their extensive review Reichart and Philipsen (1995) reported a recurrence rate of unicystic ameloblastoma was 13.7 percent. Gardner (1996) suggested that the reason for this better prognosis was that in many examples the ameloblastoma involved only the epithelial lining of the cyst or projected into the lumen and was therefore confined by the fibrous connective

tissue wall. The lesion was consequently removed completely by enucleation and theoretically, cannot recur.

As a result of this low recurrence rate there have been very few articles published that gave this particular aspect significant attention. Thompson *et al* (1993) reported a unicystic ameloblastoma of the maxilla that recurred six years after the original tumour was enucleated. They suggested that the recurrence was due to tumour residue rather than seeding during the operation as the lesion had been removed *in toto* without a breach of the wall. Furthermore, there had been epithelial islands present in the fibrous wall of the lesion (a Group 3 lesion) and this is a more likely explanation for the recurrence.

The report by Furuki *et al* (1997) *vide supra* in which they described six stages in the radiological sequence of the recurrent lesions is of interest to me in that (a) their recurrent lesions were multilocular soap bubble or honeycomb in appearance; and (b) the site of the recurrence was at the periphery of the regenerated bone.

The soap bubble or honeycomb pattern, they suggested, might be due to multicentric proliferation of the tumour. If the lesion remained a single cavity, then this finding could be further evidence that the unicystic ameloblastoma may appear to be multilocular. Unfortunately, they do not discuss the histology of the recurrent lesions.

The sites of all the recurrences in the Furuki study were at the periphery of the regenerated bone rather than at the original tumour margin. They suggested that the reduction in the intracystic hydrostatic pressure of the cyst allowed for bone formation from the inner surface of the cavity and thereby displaced the ameloblastoma cells together with their submucosal connective tissue toward the centre. Once renewed growth of the unicystic ameloblastoma

occurred, the site of this recurrence is likely to be the existing and not the original margin of the lesion. This finding is likely to occur in all the histological types of unicystic ameloblastoma.

However, I feel that the Group 3 lesions may in addition to the above, show signs of recurrence at the original margin or anywhere within the thickness of the regenerated bone, as tumour islands might have infiltrated the surrounding bone. This invasion of surrounding bone would create multiple growth centres for the lesion and possibly give rise to a multicystic lesion. If this argument is taken further it might suggest that the Group 3 unicystic ameloblastoma may be a precursor to a multicystic/conventional ameloblastoma.

Gardner and Corio (1984) reported that a plexiform unicystic ameloblastoma (= Group 2 lesion) recurred as a conventional ameloblastoma. They also had an example of a conventional ameloblastoma that recurred as a plexiform unicystic ameloblastoma. Punnia–Moorthy (1989) reported a unicystic ameloblastoma (originally diagnosed as a dentigerous cyst) that recurred 21 years later as a follicular ameloblastoma.

Gardner and Corio (1984) cautioned, however, that a radiolucency in the site of previous surgery for ameloblastoma does not necessarily imply a recurrence, and might represent fibrous connective tissue, or even a traumatic neuroma. In such circumstances a biopsy should be performed in an attempt to avoid unnecessarily extensive surgery.

Ethical considerations in treatment

When planning treatment, the surgeon must remember that he/she is treating a patient and not a lesion. Various factors must be considered, including:

- the general health of the patient;
- the nature of the lesion and its biological behaviour;
- the size and extent of the lesion;
- the age of the patient; and
- the ability of the patient and/or family (or guardian) to understand the treatment plan and to comply with instructions:
    - patient reliability for follow–up; and
    - the psychological impact of the surgery on the patient/family.

Roos *et al* (1994) cautioned that "all unicystic ameloblastomas, irrespective of grouping, are neoplastic in nature and will recur if not completely removed." They also emphasized that a recurrent lesion may occur as either a unicystic or multicystic ameloblastoma.

In view of the above it is clear that the aim of treatment should be to remove the entire lesion, then to follow–up the patient for possible recurrences. However, it is clear from the sample in this study and the other South African studies, that the unicystic ameloblastoma can reach considerable dimensions before diagnosis, resulting in extensive destruction of the involved jaw (usually the mandible). To remove the lesion in total, would often mean a hemimandibulectomy. This is mutilating surgery, especially when one takes into consideration that most of the patients with unicystic ameloblastomas are under 20 years of age. In view of the above, the two stage treatment option utilized at Groote Schuur Hospital/University of the Western Cape seems attractive.

My feeling is that the patient should be treated in the least mutilating manner. By this I propose that what is left behind is more important than what is removed.

(k) ***Histogenesis***

With regard to histogenesis Leider *et al* (1985) favoured the proposal that the ameloblastomas may arise in a dentigerous or other type of odontogenic cyst in which neoplastic ameloblastic lining epithelium is preceded temporarily by a non–neoplastic stratified squamous epithelial lining.

On the other hand Ackermann *et al* (1988) favoured the concept that the unicystic ameloblastoma arose *de novo* probably from reduced enamel epithelium. Li *et al* (1995) supported this concept when they demonstrated that PCNA activity in cystic tumour linings was greater than in dentigerous cyst linings. In my opinion that does not necessarily imply that the unicystic ameloblastoma arose *de novo*. Perhaps, once the dentigerous cyst transforms into a neoplasm, its PCNA activity would increase.

There does not appear to be much support for the third alternative that a solid tumour undergoes cystic degeneration of ameloblastomatous islands with subsequent fusion of multiple microcysts to develop a unicystic lesion.

Unfortunately these remain speculations and might possibly be resolved only after further laboratory investigation. However, before that several other questions should be addressed:

- what are the initiating factors in the development of ameloblastoma; and
- how do they grow?

CONCLUSION

From the findings of this study the following conclusions can be drawn:

1. The unicystic ameloblastoma is more common in males.
2. This lesion occurs primarily in younger patients with a median age at the time of diagnosis of 17.5 years, and mean of 22 years.
3. The 'dentigerous variant' of the unicystic ameloblastoma occurs almost a decade earlier than the 'non–dentigerous variant'.
4. The unicystic ameloblastoma is most common in blacks.
5. The mandible is by far the more common site of occurrence but the lesion can occur in the maxilla.
6. These lesions may reach very large dimensions.
7. Swelling with bony expansion of the affected jaw is a commonly reported feature.
8. The radiological margins of the unicystic ameloblastoma are usually distinct or even corticated.
9. These lesions usually appear to be unilocular and radiolucent but a small number may be interpreted as multilocular lesions.
10. Root resorption and tooth displacement are seen in approximately 50 percent of all cases.
11. The Group 3 lesions (according to the classification of Ackermann *et al*, 1988) were the most common histological subtype of unicystic ameloblastoma in this series.

REFERENCES

1. Abaza NA, Gold L, Lally E. Granular cell odontogenic cyst: A unicystic ameloblastoma with late recurrence as follicular ameloblastoma. *J Oral Maxillofac Surg* 1989; 47:168–175.
2. Ackermann GL, Altini M, Shear M. The unicystic ameloblastoma: a clinicopathological study of 57 cases. *J Oral Pathol* 1988; 17:541–546.
3. Aguirre A, Takai Y, Meenaghan M, Neiders ME, Natiella JR. Lectin histochemistry of ameloblastomas and odontogenic keratocysts. *J Oral Pathol Med* 1989; 18:68–73.
4. Altini M, Coleman H, Doglioni C, Favia G, Maiorano E. Calretinin expression in ameloblastomas. *Histopathology* 2000; 37:27–32.
5. Altini M, Coleman H, Kieser J, Kola H, Sneider P. Three–dimensional computed tomography reconstruction in treatment planning for large ameloblastoma. *Oral Surg Oral Med Oral Pathol Oral Radiol Endod* 1996; 81:619–622.
6. Benn A, Altini M. Dentigerous cysts of inflammatory origin. A clinicopathologic study. *Oral Surg Oral Med Oral Pathol Oral Radiol Endod* 1996; 81:203–209.
7. Coleman HG, Altini M, Groeneveld HT. Nucleolar organizer regions (AgNORs) in odontogenic cysts and ameloblastomas. *J Oral Pathol Med* 1996; 25:436–440.
8. El–Abdin H, Ruprecht A. Unicystic ameloblastoma in the Sudan. *Int J Oral Maxillofac Surg* 1989; 18:64–67.
9. Eversole LR, Leider AS, Strub D. Radiographic characteristics of cystogenic ameloblastoma. *Oral Surg Oral Med Oral Pathol Oral Radiol Endod* 1984; 57:572–577.
10. Feinberg SE, Steinberg B. Surgical management of ameloblastoma: Current status of the literature. *Oral Surg Oral Med Oral Pathol Oral Radiol Endod* 1996; 81:383–388.

11. Floyd P, Palmer P, Palmer R. Radiographic techniques practice dental implants. *Brit Dent J* 1999; 187:359–365.

12. Furuki Y, Fujita M, Mitsugi M, Tanimoto K, Yoshiga K, Wada T. A radiographic study of recurrent unicystic ameloblastoma following marsupialization. Report of three cases. *Dentomaxillofacial Radiology* 1997; 26:214–218.

13. Gardner DG. A pathologist's approach to the treatment of ameloblastomas. *J Oral Maxillofac Surg* 1984; 42:161–166.

14. Gardner DG. Critique of the 1995 review by Reichart *et al* of the biologic profile of 3677 ameloblastomas. *Oral Oncology* 1999; 35:443–449.

15. Gardner DG. Plexiform unicystic ameloblastoma: A diagnostic problem in dentigerous cysts. *Cancer* 1981; 47:1358–1363.

16. Gardner DG. Some current concepts on the pathology of ameloblastomas. *Oral Surg Oral Med Oral Pathol Oral Radiol Endod* 1996; 82:660–669.

17. Gardner DG, Corio RL. The relationship of plexiform unicystic ameloblastoma to conventional ameloblastoma. *Oral Surg Oral Med Oral Pathol Oral Radiol Endod* 1983; 56(1):54–60.

18. Gardner DG, Corio RL. Plexiform unicystic ameloblastoma. A variant of ameloblastoma with a low–recurrence rate after enucleation. *Cancer* 1984; 53:1730–1735.

19. Gardner DG, Morton TH Jr., Worsham JC. Plexiform unicystic ameloblastoma of the maxilla. *Oral Surg Oral Med Oral Pathol Oral Radiol Endod* 1987; 63:221–223.

20. Gardner DG, O'Neill PA. Inability to distinguish ameloblastomas from odontogenic cysts based on expression of blood cell carbohydrates. *Oral Surg Oral Med Oral Pathol Oral Radiol Endod* 1988; 66:480–482.

21. Gardner DG, Pecak AMJ. The treatment of ameloblastoma based on pathologic and anatomic principles. *Cancer* 1980; 46:2514–2519.

22. Gold L, Upton GW, Marx RE. Standardized surgical terminology for the excision of lesions in bone: An argument for accuracy in reporting. *J Oral Maxillofac Surg* 1991; 49:1214–1217.

23. Haug RH, Hauer CA, Smith B, Indresano AT. Reviewing the unicystic ameloblastoma. *JADA* 1990; 121:703–705.

24. Herrold K McD. Odontogenic tumours and epidermoid carcinomas of the oral cavity. *Oral Surg.* 1968; 25:262–272.

25. Isacsson G, Andersson L, Forsslund H, Bodin I, Thomsson M. Diagnosis and treatment of the unicystic ameloblastoma. *Int J Oral Maxillofac Surg* 1986; 15:759–764.

26. Kaffe I, Buchner A, Taicher S. Radiologic features of desmoplastic variant of ameloblastoma. *Oral Surg Oral Med Oral Pathol Oral Radiol Endod* 1993; 76:525–529.

27. Kahn MA. Ameloblastoma in young persons: A clinicopatholic analysis and etiologic investigation. *Oral Surg Oral Med Oral Pathol Oral Radiol Endod* 1989; 67:706–715.

28. Kameyama Y, Takehana S, Mizohata M, Nonobe K, Hara M, Kawai T, Fukaya M. A clinicopathological study of ameloblastoma. *Int J Oral Maxillofac Surg* 1987; 16:706–712.

29. Killey HC, Seward GR, Kay LW. *An Outline of Oral Surgery.* Part I. 1975, John Wright and Sons Ltd., Bristol, Ch X, 171–182.

30. Kramer IRH, Pindborg JJ, Shear M. *Histological Typing of Odontogenic Tumours,* 2nd ed, 1992, Springer–Verlag, Berlin, 11–13.

31. Kramer IRH, Pindborg JJ, Shear M. The WHO histological typing of odontogenic tumours. A commentary on the second edition. *Cancer* 1992; 70:2988–2994.

32. Kumamoto H, Ooya K. Immunohistochemical analysis of bcl–z family proteins in benign and malignant ameloblastomas. *J Oral Pathol Med* 1999; 28:343–349.

33. Langlais RP in Delbalso. *Maxillofacial Imaging*. 1990, W.B. Saunders Company, Philadelphia, Ch 9, 328–334.

34. Leider AS, Eversole LR, Barkin ME. Cystic ameloblastoma. A clinicopathologic analysis. *Oral Surg Oral Med Oral Pathol Oral Radiol Endod* 1985; 60:624–630.

35. Li TJ, Browne RM, Matthews JB. Expression of proliferating cell nuclear antigen (PCNA) and Ki–67 in unicystic ameloblastoma. *Histopathology* 1995; 26:219–228.

36. Marks R, Block M, Sanusi D, Lowe B, Gross BD. Unicystic ameloblastoma. *Int J Oral Surg* 1983; 12:186–189.

37. Neville BW, Damm DD, Allen CM, Bouquot JE. *Oral and Maxillofacial Pathology*. 1995, W.B. Saunders Company, Philadelphia, Ch 15, 516–519, 664.

38. Ng KH, Siar CH. Desmoplastic variant of ameloblastoma in Malaysians. *Brit J Oral Maxillofac Surg* 1993; 31:299–303.

39. Olaitan AA, Adekeye EO. Unicystic ameloblastoma of the mandible: A long–term follow–up. *J Oral Maxillofac Surg* 1997; 55:345–348.

40. Olaitan AA, Adeola DS, Adekeye EO. Ameloblastoma: clinical features and management of 315 cases from Kaduna, Nigeria. *J Craniomaxillofac Surg* 1993; 21:351–355.

41. Philipsen HP, Reichart PA. Unicystic ameloblastoma. A review of 193 cases from the literature. *Oral Oncology* 1998; 34:317–325.

42. Punnia–Moorthy A. An unusual late recurrence of unicystic ameloblastoma. *Brit J Oral Maxillofac Surg* 1989; 27:254–259.

55. Shear M. Odontogenic tumours and cysts with both unicystic and multicystic variants. Are the cysts benign tumours? *J Jpn Soc Oral Tumors* 1995; 7:299–303.

56. Shear M, Rachanis CC. Epidemiology of odontogenic lesions in South Africa. *J Dent Ass S Afr* 1979; 34:685–688.

57. Shear M, Singh S. Age–standardized incidence rates of ameloblastoma and dentigerous cyst on the Witwatersrand, South Africa. *Comm Dent Oral Epidemiol* 1978; 6:195–199.

58. Shteyer A, Lustmann J, Lewin–Epstein J. The mural ameloblastoma: A review of the literature. *J Oral Surg* 1978; 36:866–872.

59. Stewart DJC. Unicystic ameloblastoma of the mandible. *Brit J Oral Maxillofac Surg* 1984; 22:307–310.

60. Stoelinga PJW, Bronkhorst FB. The incidence, multiple presentation and recurrence of aggressive cysts of the jaws. *J Craniomaxillofac Surg* 1988; 16:184–195.

61. Struthers PJ, Shear M. Root resorption produced by the enlargement of ameloblastomas and cysts of the jaws. *Int J Oral Surg* 1976; 5:128–132.

62. Tharanon W, Changsiriwatanatumrong V, Rochanawuthanant S, Kanvongkit P, Kumplananont P, Arummethawee S. Ameloblastoma: An analysis of 184 cases. Abstract 14th Int. Conf. on OMFS, Washington DC, USA 24–29/04/9. *Int J Oral Maxillofac Surg* 1999; Suppl. 1, 28.

63. Thompson IOC, Ferreira R, van Wyk CW. Recurrent unicystic ameloblastoma of the maxilla. *Brit J Oral Maxillofac Surg* 1993; 31:180–182.

64. Underhill L, Bradfield D. *Introstat*, 2nd ed, 1996, Juta and Co., Kenwyn.

65. Van Wyk CW, Thompson IOC, Wyma G. A unicystic ameloblastoma mimicking a 'globulo–maxillary' cyst: A case report. *Brit J Oral Maxillofac Surg* 1986; 24:422–425.

66. Vickers RA, Gorlin RJ. Ameloblastoma: Delineation of early histopathologic features of neoplasia. *Cancer* 1970; 26:699–710.

67. Williams TP. Management of ameloblastoma: A changing perspective. *J Oral Maxillofac Surg* 1993; 51:1064–1070.

68. Wood RE, Pharoah MJ, Nortje CJ, Stoneman DW, Farman AG. *Handbook of Signs in Dental and Maxillofacial Radiology*. 1988, Warthog, Toronto.

APPENDICES

Appendix 1: Table 1: Table of Results

Table 2: Radiological Features

Table 3: Locularity vs Associated Impactions

Appendix 2: Request for Radiographs
- King Edward VIII Hospital

Appendix 3: Request for Radiographs
- Frere Hospital

Appendix 4: Request for Permission and Reply
- Mosby

Appendix 5: Request for Permission and Reply
- Munksgaard International Publishers, Ltd.

Appendix 6: Request for Permission and Reply
- Stockton Press

Table 1: *Table Of Results*

1	217/97	Groote Schuur	83189134	Groote Schuur	Female	57	Coloured	Left body of mandible
2	494/96	King Edward VIII	29494	King Edward VIII	Male	25	Black	Left angle of mandible
3	269/94	Frere	20378071	Frere	Male	16	Black	Right body-angle mandible
4	496/96 and 613/96	King Edward VIII	646789	King Edward VIII	Male	14	Black	Left angle-ramus mandible
5	731/93	Groote Schuur	70697206	Groote Schuur	Male	30	Coloured	Right anterior mandible
6	475/92	Groote Schuur	77960003	Groote Schuur	Male	9	Coloured	Right body-ramus mandible
7	97/91	Groote Schuur	75753707	Groote Schuur	Male	16	Black	Left body to right parasymphysis of mandible (44-37)
8	552/94	Groote Schuur	83189134	Groote Schuur	Male	26	Coloured	Body to ramus mandible-right
9	276/91	King Edward VIII	116987	King Edward VIII	Male	13	Black	Left angle to body of mandible
10	123/90	Livingstone	3073/90	Livingstone	Male	23	Black	Anterior maxilla (11-25)
11	201/90	Frere	1452079	Frere	Female	13	Black	Mandible (46-36)
12	705/89	Livingstone	95096/89	Livingstone	Female	20	Black	Anterior mandible midline
13	532/89	Private		Private	Male	39	White	Left body of mandible (35-38)
14	449/99	Frere	4167	Frere	Female	18	Black	Right body of mandible
15	70/89	UWC	8901-224	University of the Western Cape	Female	21		Left body of mandible
16	91/88	UWC	886-074	University of the Western Cape	Male	8		Maxilla (21-26)
17	465/94	Groote Schuur	80551849	Groote Schuur	Male	17	Black	Anterior mandible (43-33)
18	597/91	Groote Schuur	69688398	Groote Schuur	Female	19	Coloured	Left body-angle of mandible
19	310/86 and 151/85	Groote Schuur	52179751	Groote Schuur	Male	17	Black	Anterior mandible (43-35)
20	615/87	Conradie	30023956	Conradie	Male	25	Coloured	Anterior-left body mandible (42-36)
21	235/97	King Edward VIII	723386	King Edward VIII	Female	19	Black	Right angle-ramus of mandible (46-coronoid)
22	69/97 and 100/98	Groote Schuur	60151867	Groote Schuur	Male	16	Coloured	Mandible (46-48 area) ramus (to sigmoid notch and coronoid)
23	129/98	King Edward VIII	774781	King Edward VIII	Male	14	Coloured	Mandible
24	349/99	King Edward VIII	98145574	King Edward VIII	Female	69	Black	Mandible (anterior)
25	354/99	UWC	9803-507	University of the Western Cape	Male	35	Black	Mandible (anterior)
26	440/99	Livingstone	55386	Livingstone	Female	15	Coloured	Mandible (left body to anterior) 35 to 45 areas
27	334/99	Groote Schuur	70909080	Groote Schuur	Female	11	Coloured	Mandible (right body, angle, ramus coronoid)
28	239/99	Groote Schuur	70454152	Groote Schuur	Male	12	Coloured	Mandible left body angle ramus

		Groote Schuur	= 11	Male	= 18	Black	= 14
		Livingstone	= 3	Female	= 10	Coloured	= 11
		University of the Western Cape	= 3			White	= 1
		King Edward VIII	= 6				
		Conradie	= 1				
		Frere	= 3				
		Private	= 1				

Mandible	Body	73 x 40	Cardiac patient with mitral valve replacement. Jaw lesion present for unknown period of time. At operation thick lining easily removed from cavity.
Mandible	Angle	74 x 35	1 year swelling left angle of mandible
Mandible	Angle, body	74 x 51	Expansion if buccal plate and lower border – retained root 46
Mandible	Angle, ramus	61 x 99	Swelling left mandible 2 months duration
Mandible	Symphysis	33 x 27	Expansion of labial cortex from 44-31 regions
Mandible	Body, ramus	82 x 35	Buccal expansion with displacement of 45, 46, 47
Mandible	Body, parasymphysis	126 x 54	6 months history of swelling left body of mandible and anterior region – mobile and displaced teeth – fluctuant swelling
Mandible	Body, ramus	62 x 50	Buccal and lingual expansion – at operation broke through superior surface – solid mass
Mandible	Angle, body	indistinct margins	Swelling left angle – no caries complains of painful teeth – present for 4 months
Maxilla	Symphysis	45 x 43	Swelling anterior maxilla with buccal and palatal expansion displacement of teeth – overlying mucosa normal colour
Mandible	Body, symphysis, body	135 x 45	Very little buccal plate remaining – clear fluid aspirated
Mandible	Symphysis	16 x 20	Cyst anterior mandible – all teeth vital
Mandible	Body	48 x 35	Gradually increasing swelling left mandibular buccal sulcus for few months – intermittent discomfort – no changes in 5th and 3rd nerves – at operation thick wall cyst – neurovascular bundle freed
Mandible	Body	29 x 28	Buccal expansion with erosion of plate – missing 36
Mandible	Body	41 x 30	Retained roots 36 – aspirated lesion – cholesterol crystal obtained
Maxilla	2nd quadrant	43 x 22	Swelling left maxilla for 3 months – no pain – buccal and palatal expansion – eggshell crackling
Mandible	Symphysis	27 x 30	Non-painful swelling anterior mandible causing a expansion and displacement of 43, 42, 41, 31, 32, 33 – overlying mucosa normal in colour and texture – buccal cortex perforated – 42 and 41 non-vital
Mandible	Angle, body		Swelling left angle of unknown duration – measures 50mm wide in diameter with expansion of buccal and lingual cortices
Mandible	Symphysis	40 x 23 and 56 x 31	Lesion present for 2 months – minimal pain – bilateral mental nerve parasthesia – thin cortex
Mandible	Body, anterior	50 x 26	Extra-oral swelling in 31-34 region – intra-oral buccal sulcus swelling – long standing lesion – perforated both buccal and lingual cortices
Mandible	Angle, ramus	85 x 52	Swelling right mandible – present for 1 year
Mandible	Angle, ramus	85 x 51	Large lesion, right mandible with buccal and lingual expansion
Mandible	Angle, ramus	110 x 56	Swelling left mandible
Mandible	Symphysis	58 x 43	Swlling chin of mandible – long standing – thickened mucosa intra-orally
Mandible	Symphysis	29 x 20	Mandibular anterior region – 31 non-vital – mobile 42, 41, 31
Mandible	Body symphysis	73 x 46	Buccal/Labial and Lingual expansion – long standing, non-painful – marsupialised
Mandible	Angle, ramus	91 x 36	Right bony hard buccal swelling in 48 area – no mental nerve parasthesia – no surface ulceration
Mandible	Angle, ramus	97 x 53	Bony hard swelling left angle of mandible – one month duration – eggshell crackling, mucosa intact? – mental parasthesia or pain

Mandible	= 26	Body	= 4	Body, symphysis, body	= 1
Maxilla	= 2	Angle	= 1	2nd quadrant	= 1
		Angle, body	= 3	Body, anterior	= 1
		Angle, ramus	= 6	Body, symphysis	= 1
		Symphysis	= 7		
		Body, ramus	= 2		
		Body, parasymphysis	= 1		

Radiolucent lesion with corticated margins. Uniform Radiolucency. Broke through alveolar crest.
Multilocular radiolucent lesion with corticated margins. Eversole type F.
Radiolucent lesion with well demarcated margins. Roots of 45, 46, 47 involved in lesion. Mandibular canal displaced to inferior cortex. Eversole type D.
Multilocular radiolucent lesion from 35 to condyle and coronoid. Developing 38 and roots of 36, 37 within lesion. Expansion of lower border. Eversole type C.
Well defined multilocular radiolucent lesion. Eversole type F.
Well defined unilocular radiolucent lesion with displacement of 47 into ramus; 46 to inferior border; 45 mesioinferiorly; and mandibular canal inferiorly. Eversole type B.
Unilocular radiolucent lesion from 44 to 37 areas. Root resorption 42, 42, 31, 32, 33, 34, 35, 36 and displacement of teeth. Mandibular canal displaced inferiorly. Eversole type D.
Large unilocular radiolucent lesion of right angle of mandible; attached to neck of impacted 48. Eversole type B.
Poor radiograph. Radiolucent lesion 38 area within distinct margins.
Unilocular radiolucent lesion anterior maxilla from 11 to 25. Displacement of teeth and resorption of 21 and 22. Extends to floor of nose. Lateral wall of left maxillary sinus displaced laterally.
Uniform unilocular radiolucent lesion from 46 to 36. Root resorption 42, 41, 31, 32. Displacement of teeth. Eversole type D.
Well demarcated radiolucent lesion anterior mandible between 41 and 31 (splayed with root resorption). Unilocular. Eversole type D.
Uniform radiolucent lesion from 35 to 38. Well demarcated. Root dresorption of mesial root of 38. Mandibular canal displaced inferiorly. Eversole type D.
Uniform radiolucent lesion left body of mandible. Evidence of bony trabeculae. Roots of 37 and 35 into cavity. Well circumscribed. Extends to alveolar crest. Eversole type D.
Radiolucent lesion extending from 35 to 37. Corticated margins. Evidence of trabeculae. Mandibular canal displaced inferiorly. Retained roots 36 associated with lesion as are roots of 35 and 37 (mesial root). Eversole type D.
Radiolucent lesion extends from 21 to 26. Ill-defined margins. Developing teeth associated with lesion.
Uniform radiolucent lesion with indistinct margins extending from 43 to 33. There is splaying of the teeth starting between 42 and 41 and extending to involve the 43 and 32. Appears unilocular.but difficult to tell. Margins are not corticated.
Unilocular radiolucent lesion with distinct margins involving left body and ramus of mandible with tooth 38 within lesion.
Two orthopantomograms: *First* - large well demarcated uniform radiolucent lesion extending from 43 to 35. There are 2 locules. The roots of 41, 42, 43, 31, 32, 33, 34, 35 project into the lesion. Loss of lamina dura. There is no root resorption. The 34 is displaced posteriorly. The margin of the sesion extends to an includes the left mental foramen to interradicular area between 33 and 34 (size = 56 x 31). *Second*: Uniform unilocular radiolucent lesion with roots of 41, 42, 43, 31, 32, 33 projecting into the lesion. There is loss of lamina dura on all these teeth except 42 which shows an increased periodontal space at the apex. The 3 has uprighted and the mental foramen appears intact. There is a considerable reduction in the size of the lesion over one year (size = 40 x 23 mm). The occlusal radiograph shows buccal expansion adjacent to the 33, 34, 35.
Large radiolucent lesion with indistinct margins extending from the 42 to 36. The margins are scalloped. The 41, 31, 32, 33, 34 project into the lesion. Root resorption of 33, 34 and tooth displacement of 31, 32, 33 is also present. The lesion extends into the interradicular area of the 42 and 43. The left mandibular canal i inferiorly displaced.
Large radiolucent lesion with corticated margins extending from the 46 to the ramus and into the coronoid process of the mandible; and from the alveolar crest to the inferior cortex. There is "bowing" of the inferior buccal cortex of the mandible. There is a developing 48 in the middle of the lesion. There is som trabeculations which give a multilocular appearance. There is some resorption of the distal root of the 47. Eversole type C.
Large unilocular radiolucent lesion. Right mandible from 46 periapical area to coronoid process. The margins are scalloped but distinct and there is expansion with bowing the buccal cortex. There is a developing 48 with less root formation that on the 38 close to the centre of the lesion. ? Eversole type C. The mandibul canal is displaced inferiorly.
Unilocular radiolucent lesion extending from 36 mesial root to left coronoid and condyle. The margins are indistinct. There is resorption of the distal root of the 37. The 38 lies within the lesion and has been displaced posteriorly. The mandibular canal cannot be clearly distinguished. Eversole type B
Unilocular radiolucent lesion anterior mandible extending from 35 to 44. The margins are corticated except at the crest of the alveolus where the lesion appears to have broken into the soft tissues. The inferior border of the mandible has been spared. The left mental foramen is not involved in the lesion. There i displacement of the 43, 42, 41, 33, 34 with resorption of the roots of the 42, 41, 34. Eversole type E.
Mixed radiolucent radio opaque lesion anterior mandible extending from the 33 to 42. There is root resorption of the associated teeth. There is no tooth displacement. Eversole type D. Corticated. Margins. Unilocular.
Well defined radiolucent lesion anterior mandible (corticated margins) extending from 35 to 45 and from the crest of the alveolus in the 33/34 area to 6mm from the lower border of the mandible. The 33 is seen in the depths of the lesion distal aspect of the lesion and the crown of a developing premolar (?34 or 35) is i dentigerous association with the distal aspect of the lesion. There is root resorption of the 43, 42, 41, 31, 32 as well as displacement of these tooth roots towards the right. The 33, 34, 35 are not in occlusion but only the 33 and one premolar crown are noted in the lesion. The lesion is unilocular.
Large multilocular radiolucent lesion extending from 46 apex to right corponoid process and from alveolar crest to inferior cortex. The mandibular canal is not visualised. The developing 47 and 48 are noted in the lesion. The lesion also extends from the anterior border of the ramus almost to the posterior border. No roc resorption or displacement of the 46 is noted. Margins are well defined.
Large unilocular radiolucent lesion with distinct margins. The lesion extends from the 35 onto the left ramus approaching the sigmoid notch. There is no root resorption of the 36 and 35. The developing 37 and 38 have been displaced inferiorly to the lower border and posteriorly to the sigmoid notch respectively. Th follicle of the 38 appears to have been fused/given rise to the lesion. The lesion also extends from crest of alveolus 37/38 area to the inferior cortex leaving a thin rim of lower border. The mandibular canal is not visualized.

Suggestive of inflamed unicystic ameloblastoma (Group 1).
Unicystic ameloblastoma (Group 3).
Suggestive of unicystic ameloblastoma (Group 3).
Consistent with unicystic ameloblastoma with possible mural proliferation (Group 3).
Follicular cystic ameloblastoma (features predominantly of unicystic ameloblastoma) (Group 3).
(Plexiform) unicystic ameloblastoma (Group 2).
Cystic ameloblastoma (unicystic should be entertained) (Group 2).
Consistent with unicystic ameloblastoma (Group 3).
Unicystic ameloblastoma (Group 1).
Unicystic ameloblastoma (Group 3).
Unicystic ameloblastoma (Group 1).
Unicystic ameloblastoma (Group 2).
Unicystic ameloblastoma with mural infiltration (Group 3).
Inflamed unicystic ameloblastoma (Group 3).
Inflamed unicystic ameloblastoma (Group 2).
Unicystic ameloblastoma (Group 2).
Plexiform unicystic ameloblastoma (Group 3).
Plexiform unicystic ameloblastoma (Group 3).
Unicystic ameloblastoma (Group 3).
Cystic ameloblastoma with granular cell change (Group 3).
Consistent with unicystic ameloblastoma (Group 2).
Plexiform Group 3 unicystic ameloblastoma.
Unicystic amelobastoma (Group 3).
Unicystic ameloblastoma (Group 3).
Unicystic mural ameloblastoma (Group 3).
Unicystic amelobastoma (NB: because of odontogenic epithelium and epithelial lined daughter cysts in the capsule, the presence of infiltrating ameloblastoma in the capsule of the remaining cyst cannot be excluded) (Group 3).
Cystic ameloblastoma (Group 1).
Suggestive unicystic (Group 3).

Group 1 – 4

Group 2 – 6

Group 3 – 18

Table 2: *Radiological Features*

1	MD Body	Corticated	Unilocular	Edentulous	Edentulous	No	Inferiorly	Radiolucent	
2	MD Body	Corticated	Multilocular	Yes	No	No	Inferiorly	Radiolucent	F
3	MD Body	Distinct	Unilocular	No	No	No	Inferiorly	Radiolucent	D
4	MD Angle	Distinct	Multilocular	No	No	Yes		Radiolucent	F
5	MD Anterior	Distinct	Multilocular	Edentulous	Edentulous	No		Radiolucent	F
6	MD Body	Distinct	Unilocular	No	Yes	Yes	Inferiorly	Radiolucent	B
7	MD Body	Distinct	Unilocular	Yes	Yes	No	Inferiorly	Radiolucent	D
8	MD Angle	Distinct	Unilocular	No	No	Yes	Inferiorly	Radiolucent	B
9	MD Body	Indistinct	Unilocular	No	No	No		Radiolucent	D
10	MX Anterior	Distinct	Unilocular	Yes	Yes	No		Radiolucent	D
11	MD Anterior	Distinct	Unilocular	Yes	Yes	No		Radiolucent	D
12	MD Anterior	Distinct	Unilocular	Yes	Yes	No		Radiolucent	E
13	MD Body	Distinct	Unilocular	Yes	No	No	Inferiorly	Radiolucent	D
14	MD Body	Distinct	Unilocular	No	No	No	Inferiorly	Radiolucent	D
15	MD Body	Corticated	Unilocular	No	No	No	Inferiorly	Radiolucent	D
16	MX Left	Indistinct	Unilocular	Yes	Yes	Yes		Radiolucent	B
17	MD Anterior	Indistinct	Unilocular	No	Yes	No		Radiolucent	D
18	MD Angle	Distinct	Unilocular	No	No	Yes		Radiolucent	B
19	MD Anterior	Corticated	Unilocular	No	No	No		Radiolucent	D
20	MD Anterior	Indistinct	Unilocular	Yes	Yes	No	Inferiorly	Radiolucent	D
21	MD Angle	Corticated	Unilocular	Yes	Yes	Yes		Radiolucent	C
22	MD Angle	Distinct	Unilocular	No	No	Yes	Inferiorly	Radiolucent	C
23	MD Angle	Distinct	Unilocular	Yes	Yes	Yes		Radiolucent	B
24	MD Anterior	Corticated	Unilocular	Yes	Yes	No		Radiolucent	E
25	MD Anterior	Corticated	Unilocular	Yes	No	No		Mixed	D
26	MD Anterior	Corticated	Unilocular	Yes	Yes	Yes		Radiolucent	B
27	MD Angle	Distinct	Multilocular	No	No	Yes		Radiolucent	F
28	MD Angle	Distinct	Unilocular	Yes	Yes	Yes		Radiolucent	B
	MD Angle = 8 MD Anterior = 9 MX Anterior = 1 MX Left = 1	Indistinct = 4 Corticated = 8	Multilocular = 4	No = 12 Edentulous = 2	No = 13 Edentulous = 2	No = 17		Mixed = 1	C = 2 D = 12 E = 2 F = 4

Table 3: ***Locularity vs Associated Impactions***

Unilocular	No
Multilocular	No
Unilocular	No
Multilocular	Yes
Multilocular	No
Unilocular	Yes
Unilocular	No
Unilocular	Yes
Unilocular	No
Unilocular	No
Unilocular	No
Unilocular	No
Unilocular	No
Unilocular	No
Unilocular	No
Unilocular	No
Unilocular	Yes
Unilocular	No
Unilocular	Yes
Unilocular	No
Unilocular	No
Unilocular	Yes
Unilocular	Yes
Unilocular	Yes
Unilocular	No
Unilocular	No
Unilocular	Yes
Multilocular	Yes
Unilocular	Yes

2	Multi	–	No
2	Multi	–	Yes
15	Uni	–	No
9	Uni	–	Yes

Department of Maxillofacial & Oral Surgery
Faculty of Dentistry & WHO Oral Health Collaborating Centre
UNIVERSITY OF THE WESTERN CAPE
Private Bag X08, Mitchells Plain 7785
CAPE TOWN

8 October 1998

Dr V. Rughubar
Maxillofacial Unit
King Edward Hospital
Congella
DURBAN
4001

Dear Dr Rughubar

Re: MChD THESIS - UNICYSTIC AMELOBLASTOMA

I am currently in the Department of Maxillofacial and Oral Surgery of the University of the Western Cape and Groote Schuur Hospital.

As you are aware, a mini-thesis must be submitted as partial fulfilment of the requirements for an MChD degree. I have set out to prove/challenge the widely held belief that:-

All Unicystic Ameloblastomas histologically are unilocular lesions radiographically.

The project is supervised by Professor M. Shear.

Unfortunately we do not have copies of all the radiographs of specimens against which a diagnosis of Unicystic Ameloblastoma was rendered. Enclosed is a list of Hospital numbers of cases submitted from King Edward Hospital for which the radiographs are outstanding. Please could you send me the radiographs of these cases. I will copy them and return the originals to you.

Thank you for your help and co-operation.

Yours sincerely

SUVIR SINGH

Tel -27-21-328116; Fax -27-21-323250

File Numbers of Outstanding Radiographs from King Edward Hospital

316805/93
917583
950237523
603395/96
624328
719489
723386
736023
27664

Department of Maxillofacial & Oral Surgery
Faculty of Dentistry & WHO Oral Health Collaborating Centre
UNIVERSITY OF THE WESTERN CAPE
Private Bag X08, Mitchells Plain 7785
CAPE TOWN

8 October 1998

Dr A. Garwood
P.O. Box 11144
Southernwood
EAST LONDON
4213

Dear Dr Garwood

Re: MChD THESIS - UNICYSTIC AMELOBLASTOMA

I am currently in the Department of Maxillofacial and Oral Surgery of the University of the Western Cape and Groote Schuur Hospital.

As you are aware, a mini-thesis must be submitted as partial fulfilment of the requirements for an MChD degree. I have set out to prove/challenge the widely held belief that:-
All Unicystic Ameloblastomas histologically are unilocular lesions radiographically. The project is supervised by Professor M. Shear. Unfortunately we do not have copies of all the radiographs of specimens against which a diagnosis of Unicystic Ameloblastoma was rendered. Enclosed is a list of Hospital numbers of cases submitted from Frere Hospital for which the radiographs are outstanding. Please could you send me the Orthopantomograms of these cases. I will copy them and return the originals to you.

Thank you for your help and co-operation.

Yours sincerely

SUVIR SINGH

Tel -27-21-328116; Fax -27-21-323250

File Numbers of Outstanding Radiographs from Frere Hospital

1263803
05467659
03025871
20679619
2074572
20780425
25771/95
20921961
21098991
21593900
2853836
21719877
21742507
21803036
02853836

Private Case - Dr Garwood 10201

Private Bag X17
Bellville, 7535
South Africa
Telegraph: UNIBELL
Telex: 526661
Telephone: (021) 959-2911

University of the Western Cape

Universiteit van Wes-Kaapland

Privaatsak X17
Bellville, 7535
Suid-Afrika
Telegram: UNIBELL
Teleks: 526661
Telefoon: (021) 959-2911

Dir. line/lyn .....

Dept. Maxillo-Facial and Oral Surgery

Ref./Verwys. .....

The Manager
Journals Permission Department
Mosby
6277 Sea Harbor Drive
Orlando, Florida 32887

Dear Sir/Madam

Re: Permission to reprint figures

I am a post graduate student in Maxillofacial and Oral Surgery at the Faculty of Dentistry of the University of the Western Cape in South Africa. In partial fulfilment of the requirements of a Masters degree, I am completing a thesis entitled:
A Critical Appraisal of Unicystic Ameloblastomas
My supervisors are Professors M. Shear and G. Kariem.

I will be grateful for your permission to reprint diagrams in the following references:-

1. Eversole,et.al.Radiographic characteristics of cystogenic ameloblastoma. Oral Surg. Oral Med. Oral Path. 57;572-577, 1984. (Figure 1.)

2. Leider,et.al. Cystic ameloblstoma A clinicopathologic analysis. Oral Surg.Oral Med.Oral Path. 60;624-630, 1985. (Figures 8 and 9)

Please send your reply to:
Dr Suvir Singh
2nd Floor
City Park Chambers
87 Loop Street
CAPE TOWN 8001
SOUTH AFRICA TEL: 27214806252 FAX: 27214264084

Thank you for your generosity.

Yours sincerely

.....
DR SUVIR SINGH
26 May 2000

Harcourt
Health Sciences

- W.B. Saunders
- Mosby
- Churchill Livingstone

June 12, 2000

Dr. Suvir Singh
2nd Floor
City Park Chambers
87 Loop Street
CAPE TOWN 8001
SOUTH AFRICA

Dear Dr Singh:

Permission is granted to include Vol. 57: 572-577 (Fig. 1) and Vol. 60: 624-630, 1985 (Fig. 8 & 9) from the **ORAL SURGERY, ORAL MEDICINE, ORAL PATHOLOGY, ORAL RADIOLOGY AND ENDODONTICS** in your thesis, provided that you give complete credit to the source, including the proper copyright line. If commercial publication should result, please reapply.

We realize that University Microfilms must have permission to sell copies of your thesis, and we agree to this. However, we are not granting permission for separate sale of your article.

Sincerely.

Stacey Keomany
Paralegal I
Journal Permissions Department
Mosby, Inc.
A Harcourt Health Sciences Company
Orlando, Florida 32887-6777

Private Bag X17
Bellville, 7535
South Africa
Telegraph: UNIBELL
Telex: 526661
Telephone: (021) 959-2911

University of the Western Cape

Universiteit van Wes-Kaapland

Privaatsak X17
Bellville, 7535
Suid-Afrika
Telegram: UNIBELL
Teleks: 526661
Telefoon: (021) 959-2911

Dept. MAXILLO FACIAL AND ORAL SURGERY

Dir. line/lyn .....

Ref./Verwys. .....

The Secretary
Munksgaard International Publishers, Ltd
35 Norre Sogade, Post Box 2148
DK-1016 Copenhagen K
DENMARK

Re: Permission to reprint figures

I am a post graduate student in Maxillofacial and Oral Surgery at the Faculty of Dentistry of the University of the Western Cape in South Africa. In partial fulfilment of the requirements of a Masters degree, I am completing a thesis entitled:
A Critical Appraisal of Unicystic Ameloblastomas
My supervisors are Professors M. Shear and G. Kariem.

I will be grateful for your permission to reprint the diagrams in figure 1 in the followng reference:
Ackermann E.L., Altini M., Shear M. Unicystic ameloblastoma: a clinicopathological study of 57 cases. J. Oral Pathol 1988: 17: 541-546.

Please send your reply to ...
Dr Suvir Singh
2nd Floor
City Park Chambers
87 Loop Street
CAPE TOWN 8001
SOUTH AFRICA TEL: 27214806252 FAX: 27214264084

Thank you for your generosity.
Yours sincerely

.....
DR SUVIR SINGH
26 May 2000

TO DR SUVIR SINGH FAX: 272 14 26 40 84

FROM : Dr 2 Apr. 25 2000 01:21PM P1

University of the Western Cape

Universiteit van Wes-Kaapland

Private Bag X17
Bellville, 7535
South Africa
Telegraph: UNIBELL
Telex: 526661
Telephone: (021) 959-2911

Privaatsak X17
Bellville, 7535
Suid-Afrika
Telegram: UNIBELL
Telex: 526661
Telefoon: (021) 959-2911

Dept. MAXILLO FACIAL AND ORAL SURGERY

Dr. fine/lyn .....

Ref./Verwys. .....

The Secretary
Munksgaard International Publishers, Ltd
35 Norre Sogade, Post Box 2148
DK-1016 Copenhagen K
DENMARK

Re: Permission to reprint figures

I am a post graduate student in Maxillofacial and Oral Surgery at the Faculty of Dentistry of the University of the Western Cape in South Africa. In partial fulfilment of the requirements of a Masters degree, I am completing a thesis entitled:
A Critical Appraisal of Unicystic Ameloblastomas
My supervisors are Professors M. Shear and G. Kariem.

I will be grateful for your permission to reprint the diagrams in figure 1 in the followng reference:
Ackermann E.L., Altini M., Shear M. Unicystic ameloblastoma: a clinicopathological study of 57 cases. J. Oral Pathol 1988: 17: 541-546.

Please send your reply to ...
Dr Suvir Singh
2nd Floor
City Park Chambers
87 Loop Street
CAPE TOWN 8001
SOUTH AFRICA TEL: 27214806252 FAX: 27214264084

Thank you for your generosity.
Yours sincerely

.....
DR SUVIR SINGH
26 May 2000

Permission granted by the copyright owner, provided complete credit is given to the original source and © date Munksgaard International Publishers Ltd. Copenhagen, Denmark.

per: 29 MAJ 2000

MUNKSGAARD
35 Norre Segade · P.O. Box 2148
DK-1016 Copenhagen K

Journals Division .....

Therese Møller
Email : tm@munksgaard.dk

Private Bag X17
Bellville, 7535
South Africa
Telegraph: UNIBELL
Telex: 526661
Telephone: (021) 959-2911

University of the Western Cape

Universiteit van Wes-Kaapland

Privaatsak X17
Bellville, 7535
Suid-Afrika
Telegram: UNIBELL
Teleks: 526661
Telefoon: (021) 959-2911

Dir. line/lyn .....

Dept. MAXILLO FACIAL and ORAL SURGERY

Ref./Verwys. .....

Ms S. Findlay
Dentomaxillofacial Radiology
Stockton Press
Houndsmills, Basingstoke
Hampshire RG21 6XS
United Kingdom

Dear Ms Findlay

Re: Permission to reprint figures

I am a post graduate student in Maxillofacial and Oral Surgery at the Faculty of Dentistry of the University of the Western Cape in South Africa. In partial fulfilment of the requirements of a Masters degree, I am completing a thesis entitled:
A Critical Appraisal of Unicystic Ameloblastomas
My supervisors are Professors M. Shear and G. Kariem.

I will be grateful for your permission to reprint the diagrams in figure 4 in the following reference:-
Furuki,et.al.A radiographic study of recurrent unicystic ameloblastoma following marsupialization. Report of three cases Dentomaxillofacial Radiology, 1997, 26. 214-218.

Please send your reply to ...
Dr Suvir Singh
2nd Floor
City Park Chambers
87 Loop Street
CAPE TOWN 8001
SOUTH AFRICA TEL: 27214806252 FAX: 27214264084

Thank you for your generosity.
Yours sincerely

.....
DR SUVIR SINGH
26 May 2000

31 MAY 2000

University of the Western Cape
Universiteit van Wes-Kaapland

Private Bag X17
Bellville, 7535
South Africa
Telegraphic UNIBELL
Telex: 526661
Telephone: (021) 959-2911

Privaatsak X17
Bellville, 7535
Suid-Afrika
Telegram: UNIBELL
Teleks: 526661
Telefoon: (021) 959-2911

Dept. MAXILLO FACIAL and ORAL SURGERY

Dit. teken/ons .....

Ref./Verwys. .....

Ms S. Findlay
Dentomaxillofacial Radiology
Stockton Press
Houndsmills, Basingstoke
Hampshire RG21 6XS
United Kingdom

Dear Ms Findlay

Re; Permission to reprint figures

I am a post graduate student in *Maxillofacial and Oral Surgery* at the Faculty of Dentistry of the University of the Western Cape in South Africa. In partial fulfilment of the requirements of a Masters degree, I am completing a thesis entitled:
A Critical Appraisal of Unicystic Ameloblastomas
My supervisors are Professors M. Shear and G. Kuriem.

I will be grateful for your permission to reprint the diagrams in figure 4 in the following reference:-
Furuki,et.al.A radiographic study of recurrent unicystic ameloblastoma following marsupialization. Report of three cases Dentomaxillofacial Radiology, 1997, 26, 214-218.

Please send your reply to ...
Dr Suvir Singh
2nd Floor
City Park Chambers
87 Loop Street
CAPE TOWN 8001
SOUTH AFRICA TEL: 27214806252 FAX: 27214264084

Thank you for your generosity.
Yours sincerely

.....
DR SUVIR SINGH
26 May 2000

NATURE PUBLISHING GROUP grant permission for reproduction of the attached material with the following conditions:
(a) the source of the material be acknowledged by a full reference to the original article
(b) permission to reproduce the material is obtained (as far as possible) from the author(s) of the original article(s).

SFindlay **DATE:** 1.6.00

SHARYN FINDLAY, Permissions Co-ordinator
NATURE PUBLISHING GROUP
Houndmills, Basingstoke RG21 6XS UK
Tel: +44 (0) 1256 329242 Fax: +44 (0) 1256 384018