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THE JOURNAL OF THE SRI LANKA DENTAL ASSOCIATION

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Editorial

Sri Lanka Dental Association

In keeping with the times, the members of the Ceylon Dental Association resolved at a special general meeting held in July 1978 to rename their Association, the Sri Lanka Dental Association. The Ceylon Dental Association was formed in 1932 and has functioned for nearly 46 years. At this stage it might be useful to review what Ceylon Dental Association has achieved, contributed and done for Dental surgery in Ceylon. But more important is to find out what lays ahead for our Association. The objects of the Ceylon Dental Association as laid down in the constitution are as follows:-

(*a*) The promotion of Dental and allied sciences.

(*b*) The maintainance of the honour and the interests of the dental profession by the aid of all or any of the following:- namely periodical meetings, publication of journals and other papers, grant sums of money for the promotion of dental sciences, maintainance of rights and privileges according to dental ordinance and acts, and the encouragement of a dental benevolent fund for the relief of necessitous members of the profession.

There is no doubt that in the past our Association has endeavoured to achieve its objects. The success of these cannot be expected to be anything remarkable when we find that only about one eighth of the profession are subscribed members of our association. Many of the members of our profession are often heard to ask the question "What has the Ceylon Dental Association done for us?" The answer to this question can be found in the third paragraph of the memorandum of the Association which clearly defines the objects of the Association. Neither the head or the tail of a body can do anything much by itself. The whole body must be actively engaged in its functions. The Ceylon Dental Association is its members! Exclusively the members of the dental profession. What they do for themselves is in short a reply to the question "what has the C.D.A. done for us". The members will have an Association they deserve.

It would be more prudent and appropriate for us to think and plan out how the objects of the association could be achieved for the maximum benefit of our profession for years to come.

Taking the first objective foremost that is the promotion of dental and allied sciences, our association has to take a lead in providing continuing education for our profession. Establishment of a library, organising seminars and scientific conferences local and perhaps international, lectures, discussions, demonstrations, scientific films are some ways of providing continuing education to the profession. Organised refresher courses by the university in various fields could be of immense value to the dental practitioner. Encouragement of research by granting sums of money, at least token amounts for a start, and by helping and co-ordinating various projects undertaken by members is a responsibility of the association. All these work needs organisation, management correspondence and funds. One cannot possibly do this without an office. Therefore the establishment of a Sri Lanka Dental Association office is a priority.

The 2nd objective is to maintain the honour and to promote the interests, welfare, rights and privileges of the profession. It is indeed a noble one. But unless the members of the profession make an effort to attend meetings and express their specific needs, not much can be done to promote this objective. There seems to be a general reluctance of the young dental surgeons to join the Association. It might be an interesting and useful effort to conduct a survey or a similar project to find out the reason for this reluctance. Then perhaps the Association could remedy this malady. Sri Lanka Dental Association is our representative National body. There are other Associa-

tions like the General Dental Practitioners Association, Government Dental Surgeons Association which look after their sectional interests. Nevertheless SLDA is representative of the dental profession as a whole. Hence it is the duty of the SLDA to sort out problems of the profession and to advise the administrative authorities on dental care of the community. It is essential that it is determined to make the soundest & most constructive advise at the policy makig administrative committees by selecting the best possible representatives of the profession to serve as their spokesmen so that it would benefit both the profession & the community it serves. Promotion of welfare schemes like medical sickness society assistance to set up private practice, Insurance schemes for accidents and disease have to be looked into by the Association.

It is refreshing to know that to coincide with the change of name many good things have been happening recently which makes us feel that there is fresh hope for Sri Lanka Dental Association. Urgent need of office space will be fulfilled when we get our space in the building of the organisation of professional associations. A grant for books from the Commonwealth foundation will help to make the start of the much needed library. Our association is providing continuing education by organising seminars, lectures, and clinical demonstrations. Invitation to a distinguished lecturer from London hospital sponsored by the British Council, and the planned first international dental conference are steps taken in the right direction. At a seminar held recently more papers were presented than earlier. The publication of the Journal "Dental Topics" from the Dental school Peradeniya is we hope the start of the university's participation in the continuing education for the Dental Profession.

Members have started to show an interest in their association. The Association is yours. Your unflagging interest will make the Association the healthy body it should be.

Presidential Address 1978

Sirilal A. Silva, L.D.S. (Cey.)

The first presidential address was delivered soon after the induction of the new president by the immediate past president Dr. V. S. Karunagaran who invested Dr. S. A Silva with the badge of office at the presidential meeting held on 23rd July 1978 at the Anatomy lecture theatre amidst a distinguished gathering. This was followed by a luncheon at Brighton Hotel.

Dr. Karunagaran, the immediate Past President, Members of the Council, Ladies and Gentlemen: Little did I dream when obtaining my professional qualifications from the university of Ceylon eighteen years ago, that I will be the first to be ceremonially inducted as President of the Ceylon Dental Association. I am indeed proud and very grateful to my colleagues that you have unanimously elected me as the President of an old and prestigious Association which was formed before I saw the light of day. I know that the profession has given me the highest honour that can be given to any of its members by appointing me as the leader of the dental profession in Sri Lanka—the Ceylon Dental Association being the supreme body incorporating detal surgeons in government service, dental surgeons on the University staff, dental surgeons in the armed services and dental surgeons in private practice. I do hope that my colleagues will find me a worthy successor to my distinguished predecessors.

While thanking the immediate Past President Dr. Karunagaran for the compliment he paid me, which is more than I deserve, I must pay a tribute to him for the work done during the past year as well as the contribution he has made to the welfare of the Association as an active member. It was mainly due to his interest and initiative that the Ceylon Dental Association sent a delegation to participate in a Dental Convention in Madras. This visit made us realise that the Association has a big role to play and for this purpose we need the active participation of all the dental surgeons. We have launched a drive to increase our membership. We need not only fee paying members but also members who will take an active interest in the Association.

This being the first occasion that our Association is having a ceremony of this nature, I will trace briefly the history of our profession and of this Association in Sri Lanka, Before outlining our plans for the future.

Teeth have been valued by man from early times when due to lack of other implements teeth were used to perform so many other functions. The earliest example of a prosthesis is a find in the cemetery of El Gizah near the great pyramids. It consisted of the lower left second and third molar teeth encircled by gold wire and dated about 2500 B.C. The Romans considered tooth loss a disaster and discoloration a grave misfortune. The Roman satirist Martial is reported to have asked this question about the first century A.D. How is it that Thais has black teeth and Laecaenia has white teeth? The answer "Thais has retained her own teeth whilst Laecaenia has purchased them." The same satirist martial made a reference to a prosthesis when he made this remark, "She at night lays down her teeth as does she her silken robe," It is remarkable that Gotfredson reports that as recently as sixty years ago in the country districts of Denmark it was customary for a girl to have all her teeth extracted and get a new set of dentures before marriage because her future husband was not expected to waste his money for this purpose.

The Ceylon Dental Association was founded in 1932 by twelve dental surgeons all of whom held British qualifications. The first President was Dr. Sydney Garne.

The first Dental School was opened in 1938 and here is an extract from the prospectus—"The idea of a Dental School for Ceylon originated in 1933 from the Ceylon Dental Association." This School came into being in February 1938 commencing with six post graduate medicals who were to receive a two year training with a view to granting a licence to practise Dentistry. It is intended later to give a full dental curriculum to students not already possessing a medical qualification."

The Dental School was made part of the Medical Faculty with the establishment of the University of Ceylon in 1942. The first batch of students then enrolled to follow a five year course passed out in 1947.

In 1927, unregistered persons were prohibited from practising dentistry and in 1933 the term Dental Surgeon was allowed to be used only by those possessing a recognised qualification. But it was only in 1947 that all aspects of dentistry were defined and dental practice in all its forms was restricted to those in the Dental Register. From 1947 with the increase of personnel the government dental services expanded.

There were others who also practised dentistry such as the Chinese who learned the trade by the apprentice system as well as surgery assistants and mechanics. No amount of legislation could deter them because of the paucity of qualified personnel.

Even now these unqualified persons carry on a flourishing trade specially in the provincial towns. This will stop only when there is sufficient personnel and when the general public is educated enough to see the danger of being treated by quacks. With the granting of permission for government dental surgeons to practise dentistry after hospital hours the quack may get pushed out of business. But it is deplorable that time and again the authorities ask the Association why they should not allow the unregistered the licence to practise in spite of repeated reminders by the Association that a University course will be redundant if this was allowed. Perhaps in the near future when we have our own Dental Council, this situation might not arise.

Nearly eighty percent of the dental surgeons in Sri Lanka are in the government sector. After a long and bitter struggle and thanks to the efforts of our Vice-President Dr. Goonetilleke, who is also the President of the Government Dental Surgeons Association, today, they are on a par with their medical colleagues. Due to the demand for tretatment and the lack of manpower and materials, the clinics, specially in the rural towns have become extraction centres.

The Ceylon Dental Association should play the role of an advisor to the government in the planning of the dental services. The Association has hardly ever been consulted. Moreover during the time of restricted foreign exchange for travel, when our Secretary wanted to attend at his own expense the sessions of the Federation Dentaire International, which is the governing body of all National Dental Associations, his request was turned down as being of no value, whereas a fair amount of government money has been spent on delegations attending minor conferences in other spheres.

The Ceylon Dental Association has failed to assist and advise the University on the training of dental surgeons. The staff of the Dental School is struggling against great odds to train the students with the minimum of facilities. The vote allowed to the Dental School is insufficient even to meet the basic requirements. With the increased intake, there are not enough dental chairs and a short supply of essential dental materials. It is the Dental Surgeon in practice both in the government and private sectors, who can advise the University the current demands of practice. We have given first priority to submitting a memorandum to the Ministry of Higher Education, after obtaining the views of the University Staff and the profession at large.

I belong to the group of dental surgeons in private practice or the General Dental Practitioners of Sri Lanka as we call ourselves. I am sure it will gladden the hearts of the General Dental Practitioners to know that the Royal College of Surgeons of England has recognised General Dental Practice as a speciality by the establishment of a post graduate qualification in this field called the Membership in General Dental Surgery of the Royal College of Surgeons of England. The air turbine handpiece which is a far cry from the old pedal machines has revolutionised dental treatment. In addition there is a vast range of new equipment and materials which is impossible to import because the cost is prohibitive with the prevailing exchange rates. The General Dental Practitioners are in the process of forming a co-operative to import at least part of their requirements and to equip a central dental laboratory because we do not have one to meet our needs.

I am glad that in the last issue of our journal Dr. Hilary Cooray spoke of the need for continuing education. The facilities available to us in Sri Lanka are quite inadequate. To quote Dr. Edward Samson, President of the British Dental Association in 1947, the dentist has to have manual dexterity, use intricate equipment expertly, be a practical psychologist, understand human behaviour, be sometimes a surgeon, sometimes a physician, sometimes an anesthetist, something of an artist, a designer of craftmanship, and his own radiographer and radiologist and if he still employs his own technician know more than a little about organising a small factory. To this I may add and sometimes an acupuncturist. Acupuncture is an old modality of therapy which has been given a new life. In a changing world of medicine it is best to keep an open mind and use all methods whatever their origin for the benefit of mankind. A couple of years back, the Asia Pacific Dental Federation held a three day symposium on Acupuncture in Taiwan. A university course can never give everything that is necessary to make a complete professional. Continuing education is a must. A young dental surgeon in the government sector has a chance of working under a consultant for even a short period of time. The university also conducts a course of one year's duration called the Higher Dental Diploma mainly to allow government dental surgeons to get to the next grade. The others have to go overseas for any type of post-graduate training. However there are quite a number who cannot afford to spend so much time away from their practice.

In this respect the Association has a responsibility towards the profession. I am thankful to Dr. Goonetilleke who started a series of clinical meetings. We hope to start a series of weekend lectures. But I think that like in other countries, it is the responsibility of the University to organise short refresher courses to help the busy practitioner to keep abreast with current development.

Our Association after so many years of existence lacks a library. Thanks to generous gifts by the Commonwealth Foundation and the British Council, we hope to have a well stocked library before the year is out.

The Ceylon Dental Association is a member of the Organisation of Professional Associations of Sri Lanka and we are waiting eagerly for the headquarters to come up so that we can house our own office and library there. Till such time, we will find temporary accomodation for our library.

The delegation from the Ceylon Dental Association which attended the Dental Convention in Madras, gave a pledge to organise a similar session in Sri Lanka. We hope to organise the Seminar in May 1979 and to expand the scope to include either countries within the Commonwealth or countries in South East Asia.

I hope you will bear with me if I speak for a short time on Dental Health Education which has been neglected by the profession and the Association. In Sri Lanka, the profession has failed the public at large by not educating them adequately on the importance of dental care. Modern dentistry is founded upon the prevention of dental disease and the retention of natural tooth substance rather than their replacement by artificial substitutes. In 1973, at the F.D.I. meeting in Mexico, Camara made this remark "To attempt to resolve the problem of caries by preparing and filling cavities is comparable to trying to resolve the problem of poliomyelitis by manufacturing more attractive and better quality crutches more quickly and more cheaply. In Sri Lanka as we all know the prime cause of tooth loss is periodontal disease and secondly caries. Perhaps this may be due to the fact that there is a fairly high concentration of fluorides in our water. We are also faced with the high incidence of oral cancer specially among the uneducated and poorer strata of life. The profession must not be content to treat only established disease. Otherwise what Professor S. B. Dissanayake said will come true, namely that our skill will be used to preserve the sick and not to prevent sickness and preserve the healthy.

The profession should mobilise the qualified ancillaries and other public health personnel to promote oral health education. Periodic examination is a must and owing to the shortage of manpower the public health personnel in rural areas should be trained to detect caries and early precancer-

ous leisions. The large extent of the leision when the patient is first seen is the main cause for the poor prognosis of oral cancer.

The School Dental Service is doing a good job of work in awakening a dental consciousness among the school going population. But they are completely isolated from the dental personnel in the government hospitals. More dental surgeons should be absorbed into the School Dental Service to play the proper supervisory role as well as to maintain the follow-up after the children reach the age of twelve.

We must organise an active educational programme to prevent caries. To put it in simple language a carious leision is produced by the interaction between the tooth, cariogenic bacteria and a fermentable substrate, otherwise known as the dental plaque. Periodontal disease is also largely caused by the accumulation of bacterial plaque and if the public at large is educated to prevent the accumulation of this plaque, tooth loss can be prevented. Plaque can be reduced by teaching the proper technique of tooth brushing as well as by rinsing with certain chemicals such as Chlorhexidrene. Trained personnel must continually remove calcified deposits from the teeth and encourage the patient in his efforts.

With the increase in the consumption of sugar, caries is on the increase because sucrose has been found to cause the most amount of damage. It is worth organising a campaign to ask manufacturers of sweets to use substitute sweetening agents which are not otherwise harmful to our health.

Fluoridation of water is much cheaper and more effective than the topical application of fluorides. There are of course more sophisticated and expensive methods of caries prevention such as the use of new fissure sealing materials.

We must persuade the administration that a continuous dental health education programme can bring about a substantial reduction in the incidence of dental disease.

I must thank you all for giving me a very patient hearing. I appreciate that all of you have come at great personal inconvenience to honour the Association and to honour me. I do hope that I have kindled in you an interest in the prevention of dental disease. I thank you all most sincerely.

A study of the Rugae Pattern and the shape of the Incisive Papilla in a Sri Lankan population

K. BAMBERADENIYA

Rugae — Plica palatina transversae — are the transverse ridges of mucous membrane occuring on the anterior part of the hard palate, behind the incisive papilla. The mucous membrane containing the rugae is tightly bound to the underlying periosteum. Rugae and Incisive papilla core contain dense connective tissue layers, with firmly interwoven fibres.[12].

Review of Literature.

As compared to other subjects in dentistry, the Rugae have been... "largely neglected in dental literature...[16]" The earliest account of Rugae was written by Winslow in 1753[19]. Lysell[10] states that Sappey (1857) made the first morphological study. Allen (1889)[1] was the first to relate them in reference to the teeth in the dental arch. Broomell (1902)[4] associated Rugae with the physical health of the individual while Goria (1911)[4], with mental health. Goria has claimed that Rugae in imbeciles and epileptics were primitive and less prominent than in normal people. Rugae form between 12-14 week of foetal life. Carrea (1937)[5] and more recently Leontsinis (1952) have stated that the Rugae pattern does not change throughout life. It is surprising however that the Rugae patterns have not been used in forensic work for identification of persons.

Peavy and Kendrick[13] have found a certain amount of stretching of the lateral ends of Rugae during tooth movement, the thinner Rugae stretching more than the heavier ones.

Murakami (1928)[10] studied the pattern of 100 Japanese, and comparing with other races found definite though minor racial differences.

Oshima (1937) investigated Rugae pattern of an unspecified number of Chinese. Nillest (1950) studied their morphology in a population of Germans, Saarlanders, Austrians and Swiss, (all Central European races).

Lysell (1955)[10] made a detailed research into a sample of 851 rugae of Scandinavians.

Of the more recent workers, the morphological and genetical study by Lysell, and the study of the effect of tooth movement on the Rugae by Peavy and Kendrick (1967) are outstanding contributions. Lysell was able to show that Rugae remained numerically unchanged upto the age of 23 years, after which they appeared to decrease in number. As age increased there was a tendency for the... "backward direction of the rugae to decrease..." Rugae could not be used to establish paternity, and there was no evidence of an inherited pattern. Nevertheless, Lysell stated that although less reliable than the fingerprint method, Rugae could be valuable in identification of persons, provided a detailed recording system was used.

There is no evidence of a study of Rugae undertaken in a Sri Lankan population.

The present investigation was carried out with a view to

(*a*) Comparison of Rugae pattern of the same individual after an interval of five years in order to ascertain whether the pattern could be employed for identification purposes.

(*b*) Comparison of rugae pattern of parents with those of children in the same family, in order to establish whether there are any features of the Rugae, or the shapes of Incisive papilae which are inherited.

(*c*) Ascertaining whether the pattern shows a sex difference.

(*d*) Ascertaining whether there are age differences.

(*e*) Assessing the contribution of the Premaxillary and Maxillary segments in the formation of Rugae in Cleft-palate patients.

Materials and method.

Students and members of the Dental School, and elderly persons who attended for Prosthetic Dental treatment (and were in good general health, and had healthy looking mucosa,) were selected for the study. There was also a group of cleft palate patients in one part of the investigation.

The impression of the upper jaw in all cases was taken in Alginate (Zelgan. Amalgamated Dental. London), and cast in Artificial Stone without delay, using a mechanical vibrator to eliminate air bubbles, and obtain a smooth surface.

Lysell's excellent descriptive recording system was used in this survey with some minor modifications. (Appendix I & II).

A hard lead pencil was used to outline the Rugae. They were traced into a tissues paper and transferred to a plain sheet, using carbon paper. All measurements were made on this tracing. The Protractor and Vernier-gauge were used. The same examiner interpreted, traced, transferred, and measured the rugae in every case. The results are tabulated.

(PART A) TABLE 1

CHANGES IN THE RUGAE PATTERN DURING AN INTERVAL OF FIVE YEARS

Subject	*No of Primary Rugae*	*Straight*		*Angular*		*Curved*		*Origin*		*Branched*		*United*		*Broken*		*No. of Secondary Rugae*		*No. of Fragment Rugae*		*Direction*	
		L	*R*	*L*	*R*	*L*	*R*	*L*	*R*	*L*	*R*	*L*	*R*	*L*	*R*	*L*	*R*	*L*	*R*	*L*	*R*
A	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
B	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	+	0
C	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
D	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	+	0	0	0	+	0
E	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
F	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	+
G	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	+

Total number of rugae considered — 23 (Left); 21 (R ght)
Mean interval between observations — 5 years.
Points of similarity — 52 (Left) 91.2%; 54 (Right) 94.7%
Mean lenguth/width Index — 0.6 (Left); 0.2 (Right)

(PART B) TABLE 2

SIMILAR FEATURES OF RUGAE PATTERN IN MORE THAN THREE MEMBERS OF EACH FAMILY

Family	*No. of Primary Rugae*	*Straight*		*Angular*		*Curved*		*Origin*		*Branched*		*United*		*Broken*		*No. of Secondary Rugae*		*No. of Fragment Ragaue*		*Direction Less than* 10 *Degrees*	
	L	*R*	*L*	*R*	*L*	*R*	*L*	*R*	*L*	*R*	*L*	*R*	*L*	*R*	*L*	*R*	*L*	*R*	*L*	*R*	*L*	*R*
A	0	+	0	0	.	.	0	0	0	+	.	.	.	.	0	0	0	0	0	0	0	0
B	0	+	0	0	0	0	0	0	0	0	0	0	.	.	0	0	0	0	0	0	0	0
C	0	0	0	+	0	0	0	+	+	0	0	0	0	0	0	0	0	0	0	0	0	0
D	0	0	0	0	0	0	0	0	0	0	0	0	.	.	.	.	0	0	0	0	0	0
E	0	0	0	0	.	.	0	0	0	0	0	0	0	0	.	.	0	0	0	0	0	0

Total number of rugae considered — 63 (Left)I 68 (Right)
Points of similarity — 1 (Left) 1.6%: 5 (Right) 2.4%.
Similarity in shape of Incisive paqillae in each family — nil.

(PART B) TABLE 3

SIMILAR FEATURES OF RUGAE PATTERN IN TWINS OF TWO FAMILIES.

Twins	*No. of Primary Rugae*		*Straight*		*Angular*		*Curved*		*Origin*		*Branched*		*United*		*Broken*		*No. of Second-ary Rugae*		*No. of Frag-ment Rugae*		*Direc-tion less than 10 Degrees*	
	L	*R*	*L*	*R*	*L*	*R*	*L*	*R*	*L*	*R*	*L*	*R*	*L*	*R*	*L*	*R*	*L*	*R*	*L*	*R*	*L*	*R*
A Female A' 25 yrs	0	0	0	0	0	0	+	+	0	0	+	0	.	.	.	.	.	.	0	0	0	0
B Male B' 8 yrs	+	0	0	0	.	.	0	0	0	0	0	0	0	0	.	.	.	.	0	0	0	0

(PART C) TABLE 4 SEX DIFFERENCES IN FEATURES OF RUGAE PATTERN

Number of male subjects = 15 Mean age = 26.67 years (S.D—7.37) (Rugae—101)
Number of female subjects = 15 Mean age = 24.20 years (S.D—2.81) (Rugae—92)

No.	*Features*	*LEFT*		*RIGHT*	
		M	*F*	*M*	*F*
1.	No. of Primary Rugae	46	50	55	42
2.	Straight	14	11	15	13
3.	Angular	0	1	7	4
4.	Curved	34	48	27	26
5.	Origin (R)	17	15	18	15
6.	-do- (M)	23	24	23	19
7.	-do- (L)	8	13	11	8
8.	Direction; Mean of sum	+63.4	+71.7	+43.1	+39.3
9.	Branched	8	6	7	15
10.	United	1	3	2	3
11.	Broken	0	3	0	0
12.	No. of Secondary Rugae	9	7	7	12
13.	No. of Fragmentary Rug.	10	14	13	9
14.	Mean length/width index	4o5	7o6	4o3	7o6

Coefficient of correlation r — 0.983
There is a correlation between Males and aemales
i.e. No sex difference

Coefficient of correlation r — 0.964
There is a correlation between Males and aemales
i.e. No sex difference

(PART C) TABLE 5

LENGTH OF RUGAE

MALE		*FEMALE*	
Left	*Right*	*Left*	*Right*
14.5	13.5	14.8	14.4
10.8	8.7	9.3	10.1
8.6	12.3	11.5	11.2
14.6	12.4	11.0	15.0
11.6	9.9	10.3	15.0
8.6	10.4	11.4	11.3
11.8	12.9	14.9	11.7
10.0	10.1	13.7	12.8
16.0	12.1	13.9	12.6
12.6	14.6	13.4	12.5
14.4	12.0	16.8	12.4
12.9	12.4	12.9	15.4
13.1	12.2	13.4	13.9
11.0	11.4	13.8	14.3
10.3	10.7	10.4	13.8
Mean Length 12.7	11.70	13.10	13.0

Mean Length — Male: Female Left Rugae 12.7: 13.1 (Not significant)
Mean Length — Male: Female Right Rugae 11.7:13.0 (Not significant)

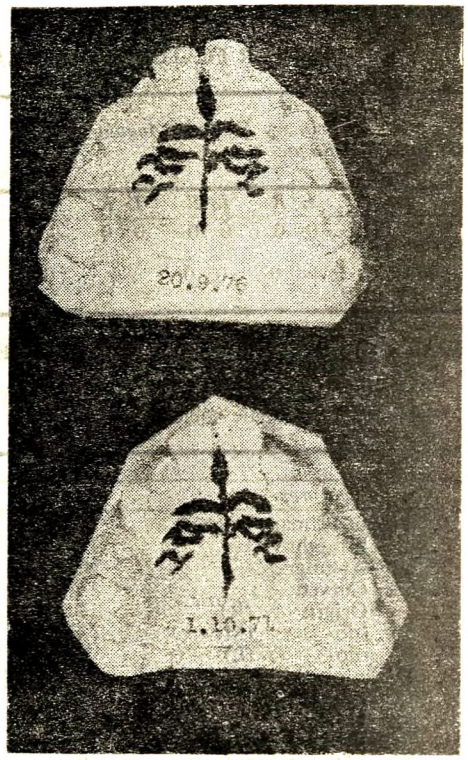
TABLE 6

SEX DIFFERENCE IN LENGTH/WIDTH INDEX OF RUGAE

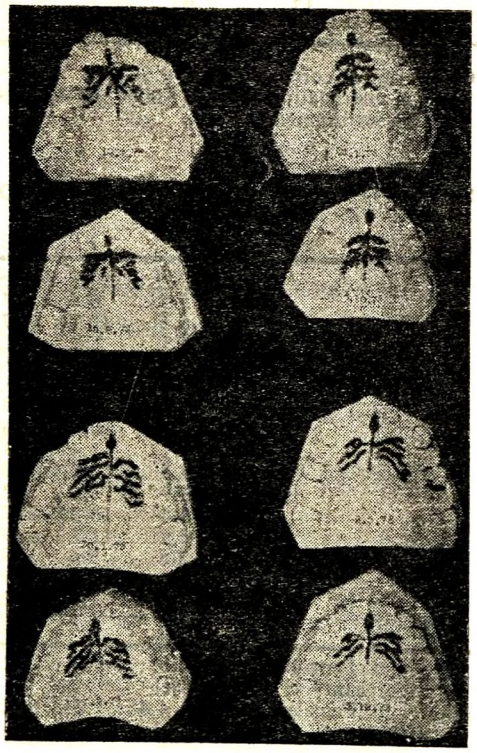
MALE		*FEMALE*	
Left	*Right*	*Left*	*Right*
6.0	5.5	6.6	4.7
5.5	5.5	10.7	7.2
9.7	7.5	2.6	1.0
2.9	2.6	2.3	4.3
3.2	2.7	7.0	5.9
3.5	2.8	9.9	9.1
5.6	5.3	10.4	6.7
4.3	2.1	3.4	3.6
7.9	7.1	12.8	10.2
4.8	3.7	11.6	12.9
2.2	3.4	16.3	17.8
4.6	2.9	11.7	19.0
2.9	2.8	9.6	9.7
5.3	5.1	9.7	9.7
3.4	3.3	3.9	2.9
Mean Index 4.8	4.5	8.9	8.3

Male and Female (Left — 4.85: 8.90 t—3.234
Significant at 0.01 level
Male and Female (Right) — 4.48: 8.31 t—2.948
Significant at 0.01 level

(PART A)



Photograph 1

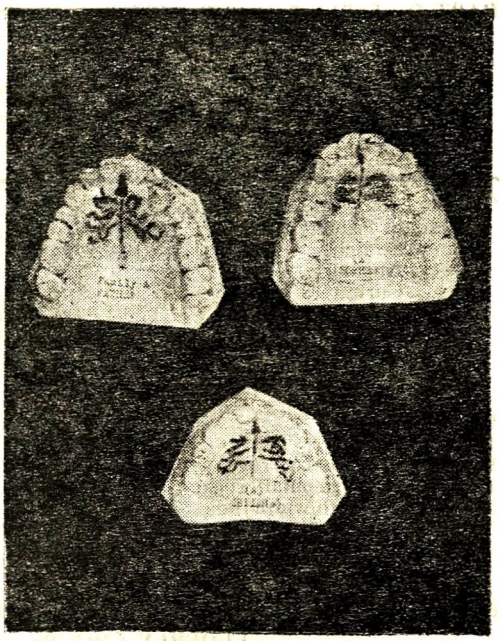


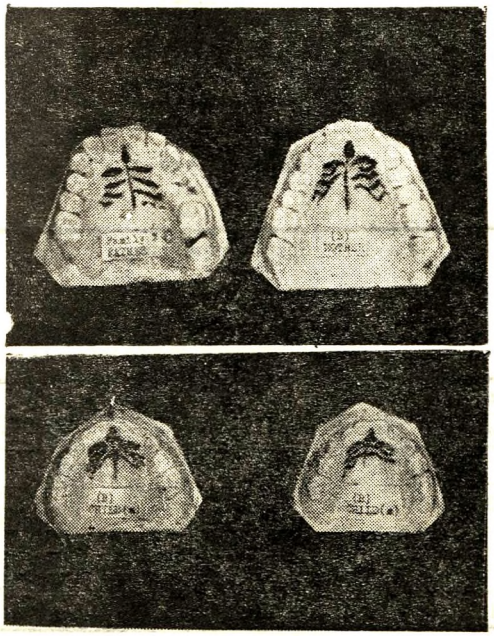
Photograph 2

Illustrating similar Rugae pattern after an interval of five years.

(PART B)

Photograph 3





Photograph 4

Illustrating the absence of any similarity of the rugae pattern in the members of family.

PART D TABLE 7 SEX DIFFERENCES IN THE FEATURES OF RUGAE PATTERN ELDERLY GROUP

Number of Males = 11 Mean age = 59.82 years (S.D. 6.68)
Number of Females = 10 Mean age = 57.30 years (S.D. 8.49)

		LEFT		*Right*	
No.	*Features*	*M*	*F*	*M*	*F*
1.	No. of Primary Rugae	38(3.45)	34(3.40)	33(3.0)	36(3.6)
2.	Straight	10(0.90)	12(1.20)	9(0.82)	8(0.80)
3.	Angular	3(0.27)	1(0.10)	2(0.18)	4(0.40)
4.	Curved	27(2.43)	22(2.2)	25(2.27)	24(2.40)
5.	Origin (R)	20(1.82)	18(1.8)	21(1.91)	15(1.50)
6.	-do- (M)	18(1.64)	13(1.30)	13(1.18)	10(1.0)
7.	-do- (L)	2(0.18)	2(0.20)	2(0.18)	6(0.6)
8.	Direction: Mean of sum	+38.4	+37.2	+14.2	1.5
9.	Branched	3(0.27)	4(0.40)	3(0.27)	4(0.40)
10.	United	1(0.09)	1(0.10)	0	0
11.	Broken	1(0.09)	0	0	3(0.30)
12.	No. of Secondary rugae	7(0.64)	2(0.20)	8(0.73)	3(0.30)
13.	No. of Fragmentary rugae	4(0.36)	3(0.30)	7(0,64)	3(0.30)
14.	Mean Length/width index	2.36	3.14	2.28	3.26

t — 0.322 There is no significant difference t — 1.456 There is no significant difference

PART D TABLE 8 COMPARISON OF THE FEATURES (MEAN VALUES) OF THE YOUNG AND ELDERLY GROUPS

Mean age of the Elderly group (21) — 59.06 years.
Mean age of the young group (30) — 25.43 years.

		LEFT		*RIGHT*	
No.	*Features*	*Young*	*Elderly*	*Young*	*Elderly*
1.	Number of lary Rugae	3.20	3.42	3.23	3.30
2.	Straight	0.83	1.05	0.93	0.81
3.	Angular	0.0	0.18	0.37	0.29
4.	Curved	2.73	2.32	1.75	2.32
5.	Origin (R)	1.06	1.81	1.10	1.71
6.	-do- (M)	1.56	1.47	1.40	1.09
7.	-do- (L)	0.70	0.19	0.63	0.39
8.	Direction: Mean of sum	+67.5	+37.8	+41.2	—7.8
9.	Branched	0.46	0.33	0.73	0.34
10.	United	0.13	0.09	0.16	0
11.	Broken	0.10	0.04	0	0.15
12.	Number of lary rugae	0.53	0.42	0.63	0.52
13.	Number of Frag. rugae	0.80	0.33	0.73	0.47
14.	Mean length/width index	6.07	2.75	5.9	3.04

t — 0.44 No significant difference t — 1.76 Significant difference

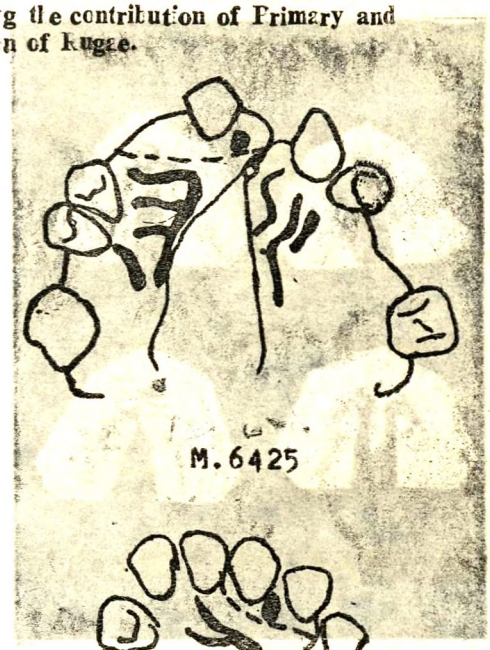
TABLE 9 SHAPE OF THE INCISIVE PAPILLA

	Part A		*Part B*		*Part C*		*Total*	*TOTALLY EDENTULOUS*									
								Partial		0—1*yrs*		1—2*yrs*		1—3*yrs*		—3*yrs*	
Shape	*M*	*F*	*M*	*F*	*M*	*F*		*M*	*F*	*M*	*F*	*M*	*F*	*M*	*F*	*M*	*F*
Long & Narrow	—	—	—	—	1	—	2	—	—	—	—	—	—	—	—	—	—
Spindle	1	3	2	2	3	10	41	3	2	—	—	—	—	—	—	—	—
Flame	—	—	—	—	3	2	10	—	—	—	—	—	—	—	—	—	—
Triangular	—	—	—	—	—	—	0	—	—	—	—	—	—	—	—	—	—
Pear	—	—	4	2	3	2	21.5	—	—	—	—	—	—	—	—	—	—
Oval	—	—	5	1	4	1	21.5	—	1	—	1	1	1	—	—	—	—
Round	—	—	1	1	—	—	4	1	—	—	—	—	—	—	1	6	6

(PART E)

Figure 1 **Diagram of Cleft Palates showing the contribution of Primary and Secondary palate to the formation of Rugae.**

T.1392



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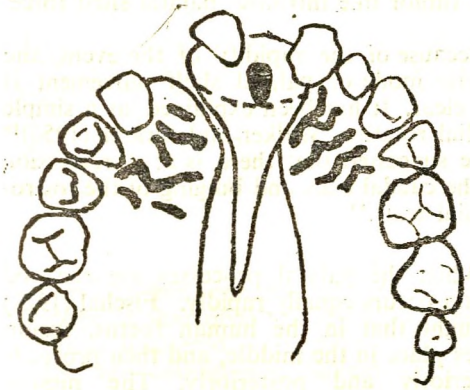

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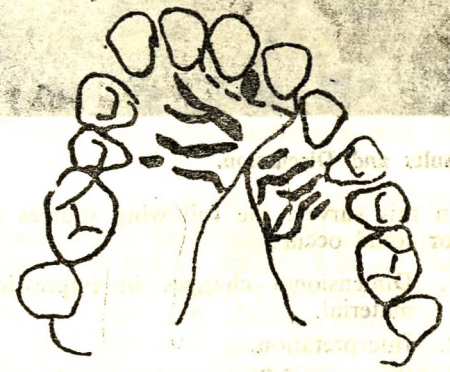

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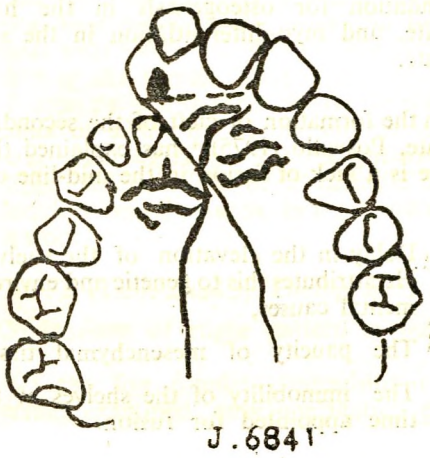

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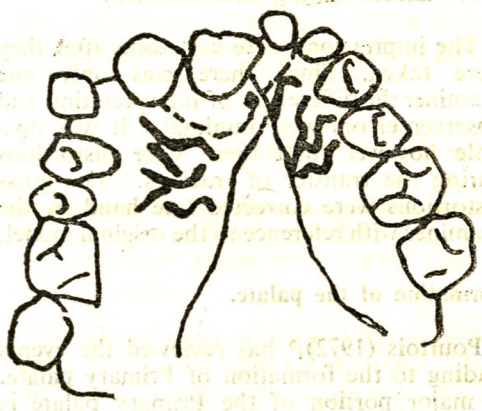
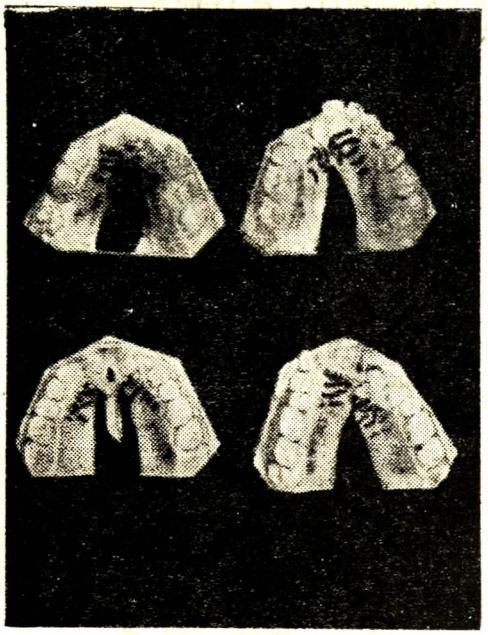


S.7300

Photograph 5



Results and Discussion.

In this survey, the following sources of error could occur:-

1. Dimensional changes in impression material.
2. Interpretation.
3. Drawing of Rugae pattern on the cast.
4. Distortion during the tracing and transferring to plain paper.
5. Errors during measurement.

The impressions were cast soon after they were taken. Since there was only one examiner the differences of interpretation and observer errors were minimal. It was possible however, that there were distortions during the transfer of tracings. The gross distortions were corrected free hand by the examiner with reference to the original model.

Formation of the palate.

Pourtois (1972)[15] has reviewed the events leading to the formation of Primary palate. A major portion of the Primary palate is formed by the fusion initially, of the Median and Lateral Nasal Processes.

Epithelial invagination of olfactory epithelium may play some part in the early establishment of an isthmus of mesenchyme across the mid-line. Later, the isthmus is supplanted by progressive contributions of mesenchyme, which penetrate into the centre of the maxillary process. Thus the initial separation of oral from the nasal cavity, is established, and the foundation of the upper lip and maxilla is laid.

At a slightly later stage, the Secondary palate formation occurs. There is no disagreement now about the general pattern. First, the paired shelves grow vertically downwards from the lateral roof of the oral cavity, to lie lateral to the tongue. About the 10-11 week I.U. the shelves assume a horizontal position over the tongue. This is thought to synchronize with 1. Extension of the head, 2. Opening of the mouth, 3. Combined effect of differential growth and turgor like intrinsic palatal shelf force.

Because of the rapidity of the event, the precise mode of palatal shelf movement is not clear. It has been explained as a simple medial rotation. Walker and Fraser (1956)[18] have suggested that there is a..."regression of the caudal ends and bulging of the rostromedial ends."

When the palatal processes are elevated fusion occurs equally rapidly. Fischal (1929) thought that in the human foetus, fusion takes place in the middle, and then proceeds anteriorly and posteriorly. The mesenchymal invasion occurs soon afterwards. The fused epithelial lining is breached by mesenchymal penetration which lays the foundation for osteogenesis in the hard palate, and myo-differentiation in the soft palate.

In the formation of cleft of the secondary palate, Poswillo (1975)[14] has explained that there is a lack of fusion in the mid-line due to:-

1. Delay in the elevation of the shelves. He attributes this to genetic and environmental causes,
2. The paucity of mesenchymal tissue,
3. The immobility of the shelves at the time appointed for fusion.

Kitamura (1977)[9] states however, that the shelves infact meet in the midline, but at the site of fusion epithelial cell proliferation occurs. Some of these epithelial clusters further increase in size, and form Cystic areas—"Epithelial pearls" He believes that the coalescence of these cystic areas causes the cleft palate. Kitamura compares this with a similar sequence of events in the fused epithelium of the eye-lid, which separates enabling the eye to open.

In the opinion of the writer the epithelial shelves move towards each other, in a horizontal direction, with the "tractive shelf force", meeting its opposite member and the nasal septum, in the midline. Their encounter causes strain "corrugations" to form on the epithelial envelope of the shelves. These corrugations are perhaps indelibly stamped with the invasion of the mesenchymal material. They are thought by the writer to be the future Rugae. The angles subtended by the rugae to the raphe of the palate, are determined by the positioning and orientation of the shelves, during their fusion with each other and the nasal septum.

PART A (Table 1) (Photograph 1 and 2)

(Comparative analysis for the Rugae pattern, after an interval of five years)

Five sets of models, cast from impressions taken at a five year interval, were examined. It appears that once established, the pattern remains unaltered. In the present sample, only one orthodontic patient showed an angular deviation of two left and one right ruga, of more than 10 degrees, during the five years under review. This is in consonance with the findings of Peavy and Kendrick, that during tooth movement the terminae near the tooth, move in the direction taken by the tooth. Despite this, the general rugae pattern remains clearly identifiable. Points of similarity in the sample studied were, Left=91,2% and Right=94.7%. Rugae pattern could be a valuable aid in identification of persons, in legal and forensic work.

PART B (Table 2 and 3) (Photograph 3 and 4)

(Comparison of rugae pattern in families)

Of the five families comprising twenty persons examined, only one feature out of fifteen were seen common to more than three members of one family. Chances of its occurrence will naturally be higher in larger families. The point of similarity on the left side in the total sample was 1.58%, while on the right the value was 2.35%. Of the two sets of dizygotic twins examined only one feature was found to be similar in one set. (Table 3). As Lysell pointed out the rugae pattern could not be used to prove paternity. There is no evidence that the features of the individual rugae, or the rugae pattern itself are inherited. The Rugae pattern like the thumb print, is specific for each individual.

PART C (Table 4, 5 and 6)

(Sex differences in the Rugae features)

The rugae of 15 males and 15 females, comprising 101, and 92 Primary rugae respectively were inspected and compared. The mean age of the sample was 26.67 years for males and 24.20 years for females. There was a strong statistical correlation between the sexes showing a lack of sex differences, with one exception.

It was found that though the lengths were similar, the length/width index in males was 4.85 and in females 8.90 on the left side, ($p>0.01$ level); and on the right side males 4.48 and females 8.31 ($p>0.01$ level). The male rugae were significantly thicker than the female rugae, as was observed by Lysell.

PART D (Table 7 and 8)

(Sex differences in the rugae pattern in elderly subjects and comparison with a younger group)

The rugae patterns of 21 elderly subjects, comprising 141 Primary rugae were studied for sex differences and compared with the younger group (in Part C). There were no statistically significant sex differences except that the male rugae were thicker than the female rugae.

In the comparison of age differences, however, it was seen that on the right side the differences were very significant. ($t=17.6$) In contrast to Lysell's findings, the rugae in the elder group tended to assume a more

backward direction. In our sample 70% of subjects were totally edentulous. It is possible that with the loss of teeth the constraints were removed, and the mucous membrance, at least superficially tended to expand fan-wise. Perhaps an investigation of a larger sample with teeth would give more conclusive findings.

PART E (Photograph 5) (Figure 1)

(Premaxillary and Maxillary components in the Rugae pattern)

Seven casts of cleft palate patients were examined for their Rugae pattern. The clefts were Veau type III and IV (Pre-alveolar and combination of Pre and Postalveolar types). In all the models examined, the Pre-maxillary component was remarkably free of Rugae.

It is the writers view that because the formative process of the Primary palate grows downwards, and at right angles to the palatal shelves, it is not subjected to the same stresses of the latter. This would perhaps explain the reason for the absence of Rugae in the Premaxilla.

Such a theory would lend support to Kitamura's finding of cystic degeneration at the line of fusion, as the cause of cleft palate. If this was so, the etiological factors would operate, not only during the first trimester of pregnancy, but even at a much later period.

Function.

The rugae of herbivorous animals are very prominent and stand out as raised ridges. They assist in grasping the grass blade against the tongue, before the leaf is incised. In man, however, the rugae are vestigeal. It is possible that they assist in the identification and discrimination of the texture of food, as this faculty decreases significantly when an acrylic plate is worn, covering the palate.[11]

Stereognostic ability also similarly decreases with dentures. [2 3]

Graber's (1950)[6], and Andersen's (1963)[21] theory of maturation of the infant swallow pattern, Hopkin & Evan's (1957)[8] and Straub's (1960)[17] analysis of tongue activity in relation to speech, and Havold (1966)[7] and others dependance on the behavioural activity for orthodontic prognoses, suggest that the correct tongue activity is an acquired reflex. Could it be that the Rugae play an important part in the orientation of the tongue, during the formative period of the adult swallow and speech?

Berry states (1966)[3] that little work has been done on normal sensory appreciation in the mouth. This statement is probably more applicable in regard to the reflex stimuli in the oral cavity. Since Rugae constitute a stable landmark in otherwise a continually changing "landscape" in the oral cavity, would this area be not a suitable starting base for such an investigation?

Incisive Papilla (Table 9)

The Incisive Papilla, like the Rugae are formed of dense connective tissue. It is said to contain the oral parts of the vestigeal naso-palatine ducts, (Stenson's organ — an auxilliary olfactory sense organ in most mammals). These are blind epithelial ducts of varying lengths. Frequently they are bordered by small irregular islands of hyaline cartilage—said to be the remnants of the capsule of Stenson's organ.[12]

In the present investigation the shapes of the Incisive Papillae in the younger age group were varied. However in the elderly edentulous sample, it was seen that gradually the Incisive papillae assumed a round shape, the longer the subjects remained edentulous.

It seemed as though the forces on the Incisive papilla which operated through the connective tissue, (rendering its shape,) appear to relax with the loss of the anterior teeth.

SUMMARY

The Rugae pattern and the shape of Incisive papilla of 80 individuals (comprising 557 Primary, 110 Secondary, and 118 Fragmentary rugae=Total 785 rugae) were examined using Lysell's recording system.

It was found that:- (1). Rugae pattern remained unaltered during an observed period of five years. (2). There were no

inherited features in their morphological pattern. (3). There were no statistically significant sex differences, except that male rugae were thicker than the female rugae. (4). In an elderly group consisting 70% edentulous subjects there were significant differences only on the right side, notably a backward directional trend of the primary rugae (5). It was observed that in cleft palates, rugae occurred only on the components of the secondary palate. (6). The shape of the Incisive papilla was varied in the young; in the elderly group the papilla tended to lose its shape and become rounded progressively, the longer they remained endentulous. A theory has been advanced for the rugae formation and their function.

ACKNOWLEDGEMENT

I am grateful to the staff of the Division of Prosthetic Dentistry for their help, and in particular, Miss M. B. Kurukulasuriya who helped me with the measurements with unflagging interest.

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REFERENCES

1. Allen H. The Palatal Rugae in Man, Proc. Acad. Nat. Sci (Philladelphia). **40** 254-273 1889. (quoted in 20).
2. Bamberadeniya K., Thiagendran A, An investigation into oral stereognosis Ceyl. Dent. Journ. **6.** 13-14. 1975.
3. Berry D.C., Mahood M., Oral stereognosis Oral ability in relation to Prosthetic treatment. Brit. Dent. Journ. **120**, 179 (1966).
4. Broomell (1902), Goria (1911): (quoted in 20)
5. Carrea J. (Buenos Aires 1937): Leontisinis S. (Beisileira 1952) (quoted in 20).
6. Graber T.M., The three M's, Muscle, Malformation, and Malocclusion, Amer. Journ. Orth. **36** 831 (1950).
7. Havold E.P., The role of function in aetiology and treatment of Malocclusion Amer. Journ. Orth. **54**, 883. (1965).
8. Hopkin G.B., McEwen J.D., Speech and the Orthodontist., Dent. Pract. & Dent. Rec. **7**, 10, 313 (1957).
9. Kitamura H., A consideration on the possibility of cleft lip or palate formation. A comparison of the palatal fusion with the Eye-lid fusion Bulletin of the Kanagawa Dental College **5**, 1.21-37 (1977).
10. Lysell L., Brayton V. (Translator) Plica palatinae transversae, and papilla incisiva in man. Morphologic and genetic study. Acta. Odont. Scand. **13**, 5-137. (Suppl. 18) 1955.
11. Murphy W.M. Morris R.A., O'Sullivan D.C., Effects of oral prostheses upon texture perception of food. Brit. Dent. Journ. **137**. 245. (1974).
12. Orban B., Oral Hitstology and Embryology. London. Henry Kimpton, 13.
13. Peavy Dan. C., (Jr.) Kendrick G. S., The effect of tooth movement on palatine rugae. Journ. Prosth. Dent. **18**. 6. 536. (1967).
14. Poswillo D., Causal mechanism of craniofacial deformities., Brit. Med. Bulletin, **31** 2 101-106 (1975).
15. Pourtois M., (1972) In Slavkin H, ed. Developmental aspects of Oral Biology. pp 81-108. Academic Press. London. (quoted in 14).
16. Salzamann J.A., Review of Lysell L. Plica pala tinae transversae and papilla incisiva in man. Amer. Journ. Orth. **41** 879-880 (1955).
17. Straub W.J., Malfunction of tongue. Amer. Journ. Orth. **47**. 404. (1960).
18. Walker B.E. Fraser F.C., Closure of secondary palaee in strains of mice. Journ. Embryol. Exp. Morph., **4**. 176-186 (1956)
19. Winslow J.B. Expositio Anatomea structurae corporis humanae. (quoted in 20)
20. Wood P. J., Kraus B.S., Prenatal development of the human palate. (Some histological observations) Arch. Oral Biol., **7**, 137-150 (1962).
21. Anderson W.S., The relation of tongue thrust syndrome to maturation and other factors. Amer. Journ. Orth. **49**. 4. 264 (1963).

APPENDIX 1

KEY TO CHART

(Adapted from:- Lysell L., Acta. Odont. Scand. **13**. 5-137. (Supp. 18) 1955.)

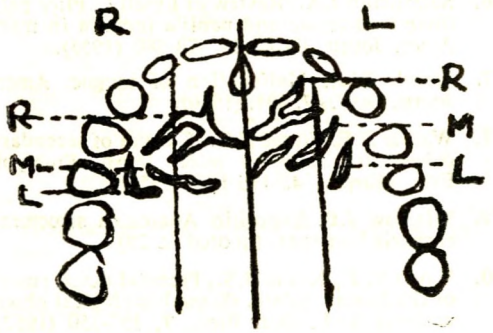
RAPHE=Midline of the palate, extending from the Incisive papilla along the mid-saggital plane.

PRIMARY RUGA=Visible on a soft or hard plaster model for at least 5 mm. irrespective of illumination.

SECONDARY RUGAE=All, except Primary rugae irrespective of width. Should be at least 3 mm. in length.

FRAGMENTARY RUGAE=Between 2-3 mm. in length. For a proliferation to be designated as a Ruga, it should be at least 2 mm. in length.

ORIGIN=One half of the palate, as divided by the raphe, is further subdivided by a line parallel to the raphe at a midpoint, between the raphe and the palatal margin of the permanent molar tooth.



R=A ruga that starts from the raphe.

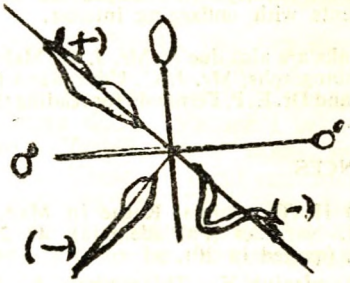
M=A ruga that starts from within the medial half.

L=A ruga that starts from within the lateral half.

SHAPE=

STRAIGHT **CURVED** **ANGULAR**

DIRECTION=Line joining the origin and the termination is extended to the raphe. The angle it makes as traced on plain paper.



Forward inclination=(+):
Backward inclination=(—)

BRANCHED=Origin in one stem, but divides into one or several branches.

UNITED=More than one origin, but they unite as they proceed laterally.

SHAPE OF THE INCISIVE PAPILLA=

FLAME **LONG & NARROW** **OVAL** **PEAR** **SPINDLE** **ROUND** **TRIANGULAR**

APPENDIX 2

RUGAE PATTERN

Index No: *Address*:

Name: *Impression taken*:

Age: *Model Examined*:

Sex:

1. PRIMARY	I		II		III		IV		V	
	L	R	L	R	L	R	L	R	L	R
(A) Straight										
(B) Angular										
(C) Curved										
(D) Origin										
(E) Direction										
(F) Branched										
(G) United										
(H) Broken										
2. SECONDARY										
3. FRAGMENTARY										

Left	Right
Length Index— Width	Incisive papilla shape—

Management of Oral Ulcers and Ulcerations

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Oral ulceration is commonly considered as a trivial disease entity by majority of patients in Sri Lanka. As a result many do not seek treatment and carry out various traditional remedies broadly based on ayurvedic teachings.

However, there are some forms of oral ulcers or ulcerations which are warnings of life threatening disease. It is important from a clinical point of view to distinguish trivial and self limiting oral ulcers from those that are more serious (Kramer[1]). The classification of oral ulcers (Lozdan and Duckworth[2]) presented below enables the clinician to identify lesions of differing aetiology with varying clinical presentation and prognosis.

1. Ulcers limited to oral mucosa at presentation.

1.1. Traumatic ulcer
1.2. Chemical burn
1.3. Recurrent apthous ulcer
1.4. Herpetic gingivo stomatitis
1.5. Herpes zoster
1.6. Acute ulcerative gingivitis
1.7. Cancrum oris
1.8. Squamous cell cancer

2. Ulcers associated with skin disease and other mucosae

2.1. Pemphigus vulgaris
2.2. Erythema multiforme
2.3. Stevens Johnson Syndrome
2.4. Behcet's syndrome
2.5. Reiters syndrome
2.6. Discoid lupus erythematosis
2.7. Erosive lichen planus

3. Ulcers related to systemic disease

3.1. Leukemia
3.2. Agranulocytosis
3.3. Cyclic neutropaenia
3.4. G.I. tract disease (Eg. Chron's).
3.5. Diabetes
3.6. Uraemia
3.7. Syphilis
3.8. Tuerculosis
3.9. Anaemia
3.10. Malnutrition
3.11. Vitamin deficiency

4. Ulcers related to drug therapy.

4.1 By interference with host defence (cawson[3])
- (*a*) Super infection (antibiotics)
- (*b*) Neutropaenia and bone marrow depression (phenothiazines, Phenyl Butazone, Carbamezapine, chloramphenicol).
- (*c*) Effects on immunological defences (Corticosteriod, Cytotoxic Agents).
- (*d*) Anti inflammatory. (indomethacin)

4.2. Lichenoid Reactions (antimetarials, gold and heavy metals).
4.3. Drug allergy and Erythema Multiforme. (sulphonamide and barbiturates).
4.4. Fixed drug reaction (phenolphthalein).

Although it is logical that patients with oral ulcers turn up for treatment to the dentist, it is clear from the above classification that during the management of oral lesions grouped in the second category referral to the dermatologist is imperative and patients belonging to third and fourth categories are best managed in consultation with the physician.

This paper reviews the present concepts of management of oral ulcers and ulcerative lesions limited to the oral mucous membrane, at the time of clinical presentation. Recent findings have greatly improved our understanding of the pathogenesis of some of these oral lesions.

Traumatic Ulcer —

Traumatic ulcer is the most common type of ulcer in the mouth and is caused by direct

trauma to the mucosa from various factors such as sharp cusps of teeth, jagged margins of carious teeth, malposed teeth, ill fitting dentures and other appliances and improper tooth brushing techniques. The diagnosis of the lesion is relatively simple as signs and symptoms of inflammation would be readily apparent.

The cause of the lesion should be identified so that this could be eliminated to prevent further recurrence. Relief from discomfort during the healing phase which usually takes about 5-7 days can be achieved by local therapy which includes the use of topical surface anaesthetics such as lignocaine oinment applied before meals and on retiring at night. Carboxy methyl cellulose paste (Orabase*) which adheres to moist surfaces and is mechanically protective has been found to be beneficial in the management of traumatic ulcers.

Ulcers which do not heal within the normal course of time (5-10 days) following removal of causative factors or traumatic ulcers with a history of long duration are best excised and several levels of the ulcer examined microscopically to rule out malignancy.

Chemical burn

Chemical burns may occur as a result of self medication by keeping asprin tablets close to the mucosa or accidentally by the dentist while using phenol, chromic acid, silver nitrate, paramonochloro phenol during various dental treatment procedures. Treatment of a burn includes palliative topical anaesthetic applications, protective coverings (orabase) or application of the appropriate antidote if available.

Recurrent apthous ulcer (RAU)

Clinically, recurrent apthous ulcers (RAU) can be classified into three categories.

(*a*) Minor aphtous ulcer (Mickulick's ulcer)

(*b*) Major apthous ulcer (Periadenitis necrotica mucosae recurrence)

(*c*) Herpetiform ulcers

Diagnosis of this type of ulcer is based on the history of recurrence and clinical presentation. Biopsy and histopathology is of no value as the microscopic features are non-specific.

As the management of these types significantly differs from each other, the recognition of each variety carries some importance.

Minor aphthous ulcers appear singly or in the form of crops of 3—4 at a time in non—keratinized surfaces of oral epithelium as shallow ulcers less than 5mm in diameter with the floor covered with a yellowish pyogenic membrane. They heal with in 10—14 days without any scarring.

Major apthous ulcer is diagnosed by its large crater type appearance being much larger in size and deeper in extension than the minor aphthous ulcer. It may affect both keratinized and non keratinized sites of the oral epithelium, generally occuring one at a time, takes 3—4 weeks to heal and produces scarring. Pain is considerable and may lead to depression.

Herpetiform ulcers (Cooke)[4], rather uncommon in its incidence, appear in clusters of pin head size with about 40 or more ulcers at a time.

Before outlining the management of RAU, the known aetiological factors underlying the clinical types should be listed as therapy is based on current findings.

The exact aetiology of RAU, is still unknown. However, several postulations have been made. The appearance of aphthous ulcers have been related to anxiety and emotional disturbances (Ship et al.[5]), Autoimmunity (Lehner[6]), heredity (Ship[7]), iron deficiency (Wray et al[8]) and vitamin B12 deficiency (Walker [9]). There is evidence to believe that RAU in females may be related to hormonal cycles as ulcers often appear premenstrually and disappear during pregnancy (Ship et al)[5].

As iron and vitamin B12 deficiencies have been implicated in the causation, a patient presenting with RAU should be investigated for Hb%, blood picture, and vitamin B12 assey. Wray et al[8] found in a haematologically deficient group of patients suffering from RAU, 65% showing a cure or remission of oral lesions following correction of blood status.

Where a systemic condition is not contributory, management of minor aphthae is limited to symptomatic treatment. Topical anaesthetic agent, (lignocaine oinment) gives relief before meals. Another preparation Bonjela* a topical analgesic with 8.7% choline salicylate has been found to be acceptable to many patients. As autoimmunity has been described recently as a possible causative factor topical corticosteroids have been used for clinical trials for RAU. Cooke and Armitage [10] have shown a reduction in the number of ulcers with this type of therapy. However, corticosteroid therapy should be limited to major aphthous type of RAU, where pain and discomfort are sometimes unbearable which may lead to depression and suicidal tendencies (Walker[9]).

Several corticosteriod preparations to be applied 2-4 times a day have been recommended (Kay [11])

(1) Hydrocortisone lozenges (corlan pellets 2.5 mg)
(2) Betamethasone lozenges (B corlan 0.1 mg)
(3) Betamethasone 17 vallerate (Betnovate .1 mg).
(4) Triamcinalone acetonide (Adcortyl 1%—A in orabase)

Lehner and lyne[12] investigated the adrenal function during topical oral corticosteroid therapy and found varing degree of suppression of adrenal cortical activity while using different topical preparations which are available in the market. The most harmful was found to be Betamethasone disodium phosphate when applied in dosage of .3-.4 mg daily, a potent preparation than other agents. However, any topical corticosteroid preparation should be used with caution. 1% Triamcinalone acetonide (Adcortyl A* Kenelog*) in the form of injectable topical corticosteroid preparations has been used for major aphthous lesions and some relief may be produced by intra-lesional, sub epithelial injections of 0.5-1.5 ml of this preparation to the surrounding mucosa using a syringe fitted with a 25 guage needle.

As hazards of systemic corticosteroid therapy are well known (Eggleston and Nally[13]) such treatment should be undertaken only for major aphthous lesions and when there is extreme distress and should be carried out preferably under the care of a hospital physician.

Use of the contraceptive pill or oestrogen therapy may benefit RAU, in a proportion of female patients where the ulcer incidence is related to premenstrual days. Several preparations are available and this type of therapy is best instituted in consultation with the family physician.

Herpetiform type of RAU, is benefited by tetracycline mouthwashes and a useful regime is to dissolve a 250 mg capsule in 100 ml of warm water and rinse the mouth t.d.s. for 3 days. Minor and major types of RAU are usually not benefited by antibiotic rinsing.

Herpetic Gingivo Stomatitis.

Diagnosed by characteristic vesicular lessions which lead to secondary ulceration giving rise to small ulcers (2-3 mm. diameter) particularly affecting gingiva, with marked sub mandibular lymphadenopathy, dribbling of saliva and fever. The diagnosis can be confirmed by the demonstration of multinucleated epithelial cells in a scraping from an ulcer which is not more than 3 days old or by a rise of antibody titre in the patient's serum against the herpes simplex virus.

Herpetic skin lesions may occur concurrently and the oral lesions are exquisitely painful. Management of this condition includes supportive therapy for pyrexia, sufficient fluids to prevent dehydration, and a tetracycline mouth wash t.d.s. to prevent secondary infection, of the lesions.

Iodoxy Uridine 0.1%, an antiviral agent used in the treatment of eye herpes has been tried for herpes labialis when ulcers characteristically appear above the muco-cutaneous junction of lip and recently Torezhalmy et al[14] found relief with a water-soluble complex of 600mg biflavonoid ascorbic acid administered in equal increments 3 times daily for recurrent herpes labialis.

Herpes Gingivo-Stomatitis is a self-limiting disease and patients fully recover in about two weeks. Recurrence of intra oral herpes is extremely uncommon.

Herpes Zoster

Oral ulcers of herpes zoster are anatomically related to the sensory nerve paths of maxillary and mandibular divisions of Trigeminal Nerve. Lesions are therefore easily diagnosed by their unilateral distribution along the affected nerve. A history of exposure to chicken pox may sometimes be obtained.

Management of oral lesions includes symptomatic therapy with Calamine lotion. The use of systemic hydrocortisone therapy remains contraversial (Kay [11]). Post-herpetic neuralgia develops in a proportion of cases and for the control of pain in such cases potent analgesics are required.

Vincent's gingivitis or Acute Ulcerative gingivitis (AUG)

This distressing oral infection commonly gives rise to oral ulceration affecting the gingival papillae in all quadrents of the mouth and in pericoronal flaps in the third molar region. Bleeding and halitosis are often associated with ulceration. Ulcer floor is covered with soft material consisting mainly of necrotic tissue.

Management of this ulcerative condition can be outlined under immediate measures to relieve pain and discomfort and delayed measures to prevent a recurrence.

Immediate local therapy to improve the oral hygiene with the use of a mouth rinse such as diluted H^2O^2 or Sodium perborate (Bocasan*) should be carried out. 10% Chromic acid which was used as a topical application is not recommended now as it leads to further gingival destruction. Scaling of teeth cannot be undertaken at this stage because of discomfort to the patient. Systemic antibiotic or antitrichromal therapy should be instituted. Fortified procaine penicillin (2 ml) 800,000 units IM followed by oral penicillin 250mg q.d.s. until systemic disturbances disappear or Metranidazole (Flagyl*) 200 mg. t.d.s. for 3 days has been found to be beneficial and the latter preparation is an useful adjunct in penicillin sensitive patients.

Once acute symptoms have subsided improvement of oral hygiene by prophylaxis, and correction of gingival contour by gingivoplasty should be carried out to prevent a recurrence; reduction of smoking is advisable as this appears to be a predisposing factor for vincent's infection.

Cancrum oris

Tissue destruction occurs in the oral cavity or para oral structures, in children leading to large crater shaped defects, with necrotic marginal tissue but structures adjacent appearing normal. This condition can be controlled with the use of correct antibiotic therapy, instituting a suitable diet, trimming of necrotic margins and is best managed in a hospital ward. Later management of the resulting deformity needs correction by plastic surgery.

Squamous cell Carcinoma.

Malignancy in the squamous epithelium manifests quite commonly in the form of an ulcer in the oral cavity. In Sri Lanka where oral cancer has a high incidence the possibility of a squamous cell carcinoma in over 40 age group should be borne in mind in patient examinations. Induration of the base and everted margins are characteristic features of this lesion and common sites of oral mucosa affected in Sri Lanka include the buccal sulci, retromolar region, lateral borders of the tongue and the alveolus. A suspicious lesion should be regarded as an urgent diagnostic problem and referral to a clinician specialized in this field is recommended. Several complaints may be associated with oral cancer, and these are related to the site & size of the lesion and rapidity of invasion of adjacent tissue. These complaints, are,

(*a*) Pain and discomfort
(*b*) Burning sensation,
(*c*) Excessive salivation
(*d*) difficulty in swallowing, speech or in opening the mouth
(*e*) Immobility of tongue
(*f*) Mobility of teeth in one quadrant.
(*g*) Unhealed extraction socket
(*h*) Mouth odour
(*i*) Denture too tight
(*j*) Nodes in neck
(*k*) Numbness in lip
(*l*) Anorexia, weight loss and weakness.

These complaints accompanied with a crater like defect in mucosa should be noted with caution. Biopsy of all suspected lesions is mandatory to establish a diagnosis early, and the critical feature of invasion should be looked into in the Histological material.

Treatment should be instituted preferrably at a specialized cancer treatment centre.

CONCLUSIONS

As there is an obligation to provide the most effective treatment possible, the recent developments in the understanding of oral ulcers should be taken into consideration in prescribing treatment. If oral ulcers persist despite local therapy beyond a 2 weeks periods a blood examination consisting of (1) Hb% (2) Blood Picture and (3) WBC / DC should be regarded as indicated to identify possible systemic background and if reported negative, a biopsy should be considered essential.

REFERENCES

1. Kramer, I.R.H. (1967) Ulceration of the mouth in children. Aust. dent. J., **12**, (2), 83-91.
2. Lozdan, J. and Duckworth R. (1972) Looking in the mouth. Dent. Update, June Issue, 1353—1363.
3. Cawson, R.A. (1972) Oral Ulceration; clinical aspects Oral Surg., **33**, 6, 912-921
4. Cooke, B.E.D. (1960) The diagnosis of bullous lesions affecting oral mucosa. Brit. dent. J., **109** (3), (4), 83-96, 131-138.
5. Ship, I.I., Merritt D. and Stanley, H.R., (1962) Recurrent aphthous ulcers. Am. J. Med., **32**, 32-43.
6. Lehner, T., (1964). Recurrent aphthous ulceration and autoimmunity. Lancet I, 1154-1155.
7. Ship, I.I. (1972). Epidemiologic aspects of recurrent aphthous ulcerations Oral Surg. **33**, (3), 400-406.
8. Wray, D., Mason, D.K., Dagg, J. and Ferguson M.M. (1974). Recurrent oral ulceration— associated haematological deficiencies and malabsorbtion syndrome. Proceedings of 1.A.D.R. Brit Div. April 1974.
9. Walker, J.E.G. (1973). Aphthous ulceration and Vitamin B12 deficiency. Brit. J. oral Surg., **11**, 165-170.
10. Cooke, B.E.D. and Armitage, P. (1960) Recurrent Mikulicz's aphthae treated with topical hydrocotisone hemisuccinate sodium. Brit. Med. J., i, 764-770.
11. Kay, L.W. (1972). Drugs in dentistry, 2nd Ed., John Wright and Sons Ltd., p. 99 and 108.
12. Lehner, T. and Lyne, C. (1969) Adrenal function during topical oral corticosteroid treatment Brit. Med. J., **4**, 138-141.
13. Eggleston, D.J. and Nally F.F. (1971) Hazards of Systemic hydrocotisone therpy Oral Surg., **31**, (5), 590-594.
14. Terezhalmy, G.T., Bottomley, W.K. and Pellen G.B. (1978) Use of water soluble ascorbic acid complex in treatment of recurrent herpes labialis. Oral Surg., **45**, (1) 56-62.

Some Electrophysiological Contributions to Unravel the Nature of Dental Pain

A mini — review.

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INTRODUCTION

Pain is a common experience and a few people have escaped pain from teeth. Sherrington (1900) described pain as a psychical adjunct to an imperative protective reflex and this is acceptable even today. One of the common sites where pain is experienced is the teeth. In addition, it has been postulated that the only sensation which can be evoked from stimulation of nerves in dentine or pulp is pain (Anderson, Hannam and Matthews, 1970), although the evidence from recent findings has raised doubts about this (Matthews, Baxter and Watts, 1976). Nevertheless, over the past few decades the teeth have been the focus of many investigations carried out on pain and this approach is still being used.

First of all we must look at the ways in which the pain sensation arising from teeth could be investigated. Of course, one can show the presence of nerve terminals and fibres by electronmicroscopy. Or else, one could utilise histochemical techniques to identify special types of chemical transmitters (such as substance or endorphin like chemicals) which could modulate the perception of pain. However, it is most appropriate to select a technique to study while the tissue is live. In addition, it is always better to stimulate the experimental stimuli as much to those which resemble natural stimuli; and this could be achieved in an electrophysiological experimental set up. Thus, electrophysiological techniques would be most appropriate to this type of investigation although it also has its own limitations. By this technique, the tooth, specially dentine, could be stimulated by mechanical, thermal, electrical or chemical means and recordings could be made from the relevant nerve fibres, or vice-versa.

Previous studies.

Pfaffman (1939) performed the earliest electrophysiological recording from the pulpal nerves of the upper canines of cats and stated that nerve impulses evoked by noxious stimuli had relatively slow conduction velocities compared with those evoked by pressure. In 1953, Brookhart, Livingstone and Haugen showed in cats, conduction velocities characteristic of a delta fibres. Wagers and Smith (1960) carried out experiments in dogs and found one fibre with a conduction velocity of 0.8 m/s and several A-delta fibres. Anderson and Perl (1974) also demonstrated A delta and C fibre afferents in feline teeth. From the few electrophysiological experiments cited above, it is evident that most of the fibres innervating teeth consist of A-delta and C fibres. However, most of these works are inconclusive. Therefore it is worthwhile to look at the results obtained by using more recent refined techniques utilised to explore dental innervation, and thereby try to explain dental pain.

Recent electrophysiological work.

The spread of the applied stimulus to adjacent tissues like gingiva and periodontium is the most critical factor in describing a certain nerve fibre as a pulpul or extra pulpul origin by electrophysiological means. In a recent study (Jiffry, 1977), the possibility of stimulus spread has been excluded. This study demonstrated that in the special type of incisor of rats (continuously erupting) only C — fibre action potentials could be recorded. However, these unmyelinated fibres were only found intra-pulpally, the extra-pulpal portion showed characteristics of A-delta fibre. (Jiffry, 1979). This observation had been confirmed by an electronmicroscopic study (Bishop, 1977).

The most promising evidence has been brought forward by Horiuchi and Matthews

(1974) on studies using artefact free dentinal electrodes. With this, these workers have been able to record electrical activity from the surface of cut dentine in the lower canine tooth of cats and they confirmed that the activity is wholy nervous in origin by single fibre preparations from the inferior alveolar nerve. Later Matthews (1977) showed that there were separate heat and cold sensitive fibres in teeth without having any specialised receptors. He is of the view that the odotcnblast is a passive transmitter of any external stimuli rather than an active specialised receptor. In addition, Matthews and Holland (1975) demonstrated the presence of coupling between nerve terminals in the pulp. Since it is a very recent concept, the following paragraph will be fully devoted to this subject.

Coupling.

When two cells are in close contact the cell membranes of these cells can have (*a*) a tight junction, (*b*) a gap junction or (*c*) an adherent junction. This classification fully depended on the degree of proximity between the two cell membranes. In places where gap junctions are found, the wave of electrical depolarization of one cell membrane could be easily transmitted to the other cell via the gap junctions. Holland (1976) demonstrated the presence of gap junctions between nerve fibre terminals in the pulp and also between odontoblast and nerve fibre terminals. Further-more, Matthews and Holland (1975) had shown that due to the presence of gap junctions in the pulp, a single stimulus applied in relation to a single or group of nerve terminals would tend to spread to the other nerve fibres via the available gap junctions. This will result in one single stimulus being capable of producing a continuous barrage of nerve impulses at different latencies.

Primary afferent depolarization (PAD)

A nerve fibre is electrically polarized at rest. When an impulse wave form passes a certain part of a nerve, this part is said to be depolarized. While a nerve fibre is depolarized, there cannot be any other impulse superimposed on the existing wave form and this phenomenon is called refractoriness.

Lisney (1978) had shown that when a upper canine of a cat is electrically stimulated while the vibrissae is being pulled at a certain frequency, the PAD caused by the latter at the main trigeminal sensory nucleus would inhibit the transmission of the impulses originated from teeth. This means, that the afferent conduction of impulse caused via the A-fibres of the vibrissae, had prevented the impulses of the tooth being (A delta and C fibre) transmitted to the higher centres.

Explanation of the pain phenomenon.

Let me summarise the current electrophysiological findings on dental innervation.

(*a*) Contrary to classical belief, pain is not the only sensation felt, on stimulation of dentine and pulp.

(*b*) There are warm and cold sensitive fibres in tooth pulp; but there are no specialised receptors. Even the odontoblasts show a passive function.

(*c*) Sensations arising from tooth pulp is transmitted via C and A delta fibres.

(*d*) Sensations arising from tooth pulp could be modulated by impulses carried by the extra pulpal afferents, to the trigeminal main sensory nucleus.

(*e*) Some of the tooth pulp afferents terminate even at the reticular system of the medulla, which is adjacent to the trigeminal nucleus (Shigenaga, Sakai and Okada 1976).

(*f*) Coupling between nerve terminals take place within the tooth pulp. Due to this phenomenon, a single stimulus would be projected as a continuous barrage of impulses to the higher centres.

(*g*) Some of the tooth pulp afferents show the characteristics of branched fibres, where its other branch/ branches supply adjacent gingiva and mucous membrane. (Lisney, 1977).

In the light of these finidings, any stimulus applied to the tooth could initiate either pain, or other sensation which

some authorities refer to as "pre-pain" (Shimizu, 1964). This sensation could be continuous, although the stimulus is of very short duration. This may be due to the coupling effect. It may be possible that, even at very high threshold stimulus, pain sensation may not be elicited, in the presenc of any other stimuli applied to extra-pulpal structures. This supports the gate-control theory of pain and also lends support to the acupuncture and electro-analgesic methods adopted in alleviating dental pain. The question of referred pain also could be explained by the presence of branched fibres and also by termination of dental afferents in the main sensory and spinal trigeminal nucleus where most of the oro-facial sensory fibres converge.

This is an attempt to point out the recent electrophysiological findings relevant to dental pain. However, the day to day advancement in pain research would place us at a more and more higher elevation to look at the very deep nature of dental pain.

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REFERENCES

Anderson, D. J., Hannam, A.G, and Matthews, B (1970) Physiological Reviews. 50:171

Bishop, M.A. (1979) Journal of Dental Research. 57 (in press)

Bookhart, J.M., Livingston, W.K., and Haugen, F.P. (1953) Journal of Neurophysiology. 16;4

Holland, G.R. (1976) Anatomical Record. 186;121

Horiuchi, H., and Matthews, B. (1974) Journal of Physiology. 243: 797.

Jiffry, M.T.M. (1977) Journal of Dental Research, 56 (Special Issue D).

Jiffry, M.T.M. (1979) Experimental Neurology. (submitted)

Lisney, S.J.W., (1977) In Pain in the Trigeminal Region, Anderson and Matthews (editors).

Lisney, S.J.W. (1978) PhD Thesis. University of Bristol

Matthews, B., and Holland, G.r. (1975) Brain Research, 98:354.

Matthews, B., Baxter, J, and Watts, S (1976) Brain Research 113:83

Matthews, B. (1977) Journal of Physiology. 264:641

Sherrington, C.S. (1900) Text Book of Physiology Vol. 2.

Shigenaga, Y, Sakai, A., and Okada, K (1976) Brain Research 103: 400

Shimizu, T (1964) Journal of Dental Research 43:467

Wages, P and Smith C (1960) Journal of Pharmacology and Experimental Therapeutics 130;89

Measurement of Study difficulty in Dental Students

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ABSTRACT

Study difficulty was measured among a batch of final year Dental students using the UCLSQ. Paper and pencil psychological tests seem to be acceptable to the students. Normative data is presented. The correlation matrix of the subscales was factor analysed. Three components emerged explaining 72% of the variance.

INTRODUCTION

Study difficulty among university students have been documented by Crown *et al* (1973). Few psychological tests have been done with our students. The reports available were those dealing with predictability of academic success, Kodituwakku and Mahendra (1978).

We wanted to obtain normative data regarding study difficulty for a population of final year dental students. Since very few tests have been done with our students we wanted to find out the acceptability of such an inquiry *i.e.* the use of questionnaires in gathering data. Our ultimate goal in developing psychological tests is for effective student counselling to be undertaken. Spielberger (1962) in a study with anxious college, freshmen showed that counselling was useful to them.

The study reported here is the first of its kind (known to the author) to systematically asses problems encountered by the way of study difficulty. This is a part of a larger study; details of which would be reported later. Study difficulty was assessed with the University College of London Study Difficulty Questionnaire (UCLSQ) Crown *et al* (1973), Lucas *et al* (1976). See also appendix I. We compared our findings with reported data.

* Crowns paper describes scales 1-7, syllabism (syllabus boundness) is described by Lucas.

MATERIALS AND METHOD.

University College of London Study Difficulty Questionnaire.

The UCLSQ is fully described and proved to be valid and reliable instrument Crown *et al* (1973), Lucas *et al* (1976). In its present form it incorporates eight scales: (1) anxiety (ANX), (2) obsessionality (OBS), (3) depression (DEP), (4) disorganisation (DIS), (5) low motivation (LM), (6) somaticism (SOM) (7) work satisfaction (WS), (8) sylbism (SYL)* The true/false format of the present UCLSQ was changed to agree/disagree as this was more acceptable to our students. Questions 4, 8 and 16 were re written to suit the local idiom without altering its meaning. Plus one was allocated to each statement marked agree. (''?'') and disagree being scored zero) The eight subscales were represented by questions 1 to 72 in that order. (e.g. anxiety was represented by question 1, 9, 17. . . etc.).

The questionnaire was administered during a lecture hour. (8-9 AM). The purpose of the study was explained. Students were allowed to clarify doubts regarding questions. The entire final year batch of dental students participated. There was a total 44 students. Although they had the option of not participating all of them participated. They were assured absolute secrecy. If they were interested in knowing their results and also wished the data to be used for further analysis they were requested to write their index number on the sheet. No time limit was imposed but all scripts were handed in completed in half an hour.

RESULTS

The means (X), standard deviations (SD) and alpha coefficiants (α) Cronbach (1951) are reported in Table 1.

TABLE 1

Means, standard deviations and alpha co-efficient of scores on UCLSQ (n — 44).

	ANX	*OBS*	*DEP*	*DIS*	*LM*	*SOM*	*WS*	*SYL*
Mean	4.2	4.4	3.2	2.8	3.9	1.8	5.5	4.3
S.D.	2.25	1.61	1.75	2.10	2.46	2.06	1.79	1.62
Alpha	0.69	0.78	0.45	0.71	0.75	0.77	0.45	0.29

Principal component analysis.

Factor analysis was carried out on the correlation matrix (of the subscales) with unity at its diagonals. Orthogonal factors were obtained which were rotated to simple structure (rotations were carried out to obtain purer factors) using varimax procedure.

Three components emerged with unrotated eigenvalues greater than unity. They explained 72% of the variance. The components were,

1. A component high on ANX, LM, DIS.
2. A component heavily loaded on SYL.
3. A negatively loaded component WS.

(See Table 2 for further details.

TABLE 2

Principal component analysis of UCLSQ after varimax rotation.

	Factor I	*Factor II*	*Factor III*
ANX	0.72	0.18	—0.22
OBS	0.05	0.30	—0.28
DEP	0.33	—0.01	0.13
DIS	0.81	—0.22	—0.12
LM	0.87	0.02	0.20
SOM	0.32	—0.02	—0.11
WS	0.02	0.04	—0.98
SYL	0.01	0.98	0.03
EIGEN values (unrotated)	3.88	1.40	1.01
Cumulative percentage of (EIGEN) value (Unrotated)	0.42	0.60	0.72

DISCUSSION

It was very interesting to note that all of the students had written their index numbers on the sheet. They were very enthusiastic about the study and their individual patterns. We could safely assume that psychological tests hold promise among our students.

It must be borne in mind that we had no external criteria to rate the normalcy of the group. However if any one student had any special problems, since the entire group was studied, it would not have significantly altered the scores.

Crown and Lucas (1974) have discussed the problems related to students mental health. Having discussed many different models he correctly states that any one model would predict only a small component of the behavioural patterns (p. 601). Our aim is to develop methods for effective student counselling and the work reported here is a small but significant contribution.

Although Lucas (1976) confirms the reliability of the UCLSQ the results of the present study must be viewed with caution since Lucas's study dealt with British University students. However our local universities (and the educational system) has been patterned on the British model.

In Table 3 we see the different figures of mean values obtained in 3 different studies.* This could well be due to random effects of distribution (Tests for statistical significance between different of means could not be made as the S.Ds of the other two studies were not available) and also the different samples used in those studies.

Factor analysis was used to study the underlying inter-relationships between the subscales and also to make cross cultural comparisons with the other studies. Principal components was used as this method had been used in the previous studies. It was also readily available at the local computer centre. Everitt (1975) and Kennard (1978) have written on the various tenets to be observed when employing multivariate techniques. We were guided by these principles in our study.

* These are the only figures that the authors could obtain.

TABLE 3.

Mean score for study difficulty in the 3 different studies (normal subjects).

	ANX	*OBS*	*DEP*	*DIS*	*LM*	*SOM*	*WS*	*SYL*
Crown etal	3.49	5.63	3.53	3.73	5.59	1.89	8.41	—
Lucas *et al*	1.45	3.05	1.85	2.04	2.62	0.78	4.45	2.7
Present study	4.20	4.39	3.16	2.77	3.89	1.75	5.52	4.3

We obtained a three factor solution explaining 72% of the variance and they make psychological sense. It is important that the factors should make psychological sense else they may arise due to statistical artefacts. Eysenck (1976). Everitt states that factors to be significant must be repeatably demonstrable. The component indicating LM and DIS emerges in all of the studies. However in our study the LM, DIS component has also ANX on the same axes. (Although in previous study ANX was an unrelated component see Lucas (1976). The heavy negative loading on the WS scale is similar to that obtained by Lucas. This indicated a threat of work dissatisfaction among students. Although Lucas found this was positively related to syllabus boundness, SYL emerged as an orthogonal factor in our study. However the alpha reliabilities were low on the WS and SYL scale, hence more work is required before arriving at any firm conclusions. Also as Everitt (1975) states factors to be significant should be isolated repeatedly in independent studdies.

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REFERENCES

CRONBACH L.J. (1951) Co-efficient alpha and the internal structure of tests. *Psychometrika* 16 297-334.

CROWN, S., LUCAS, C.J. and SUBRAMANIUM S. (1973). The delineation and measurement of study difficulty in university students. *Br. J. Psy chiat.* **123**, 381—393.

EVERITT, B.S. (1975). Multivariate analysis the need for data and other problems. *Br. J. Psychiat.* **126** 237—240.

EYSENCK, H.J. (1976) Psychometricks. The statistical basis for the study of individual differences. In-: *A Text Book of Human Psychology* (Eds. H.J. Eysenck and G.D. Wilson) MTP Press: London

KENNARD, D. (1978). Identifying dimensions or perceived patient change: some problem in the use of factor analysis. *Br. J. Med. Psychol.* **51**, 42-52

KODITUWAKKU, P.W. and MAHENDRA, B. (1978). Factors in medical school performance: a further report. *Proc. Kandy Soc. Med.* **1** 16-1.

LUCAS, C.J. and CROWN, S. (1974). Concepts and methods in students mental health. *Br. J. Psychiat.* **125** 595—603

LUCAS, C.J., CROWN S., STRINGER, P' and SUBRAMANIUM, S. (1967). Further observation in study difficulty in university students including 'syllabus boundness' *Br.J. Psychi t* 129 598-603.

SPEILBERGER, C.D., WEITZ, H. and DENNY, P.J. (1962). Group counselling and the academic performance of anxious college freshmen *J. Counsel Psychology.* **9** 195-2404.

APPENDIX

Index No.....

Below is a list of feelings or reactions which students sometimes experience in relation to study. Please indicate how you stand in respect of an each item by circuling A (Agree) mainly true (or agreed). D (Disagree) mainly false (or disagree). Only if you are not quite sure circle "?". Answer each question. There are no right or wrong answers.

1. I can't stop thinking about work even when trying to relax A ? D
2. I go over work again and again even when I know it A ? D
3. When I start a piece of work I feel inadequate and incapable of doing it A ? D
4. I keep losing the hang of things A ? D
5. I just can't get down to working as much as I should A ? D
9. Thinking about work can make me feel physically ill A ? D
7. I enjoy tackling a difficult topic or problem A ? D
8. I prefer cramming (doing set work) to research A ? D
9. I always feel I have to hurry through my work A ? D
10. I hate to hand in an untidy piece of work A ? D
11. My tutors over estimate my abilities A ? D
12. I keep changing from one topic to another A ? D
13. I get bored quickly A ? D
14. I often get headaches when trying to study A ? D
15. Some aspects of my subjects are really exciting A ? D
16. I do my set work equally well whether it interests me or not A ? D
17. Sometimes when studying I get downright panicky A ? D
18. I spend too much time on unimportant detail A ? D
19. I often can't be bothered to respond to a question even when I know the answer A ? D
20. I often make silly mistakes A ? D
21. I always feel I can't catch up with my work A ? D
22. I often can't fall a sleep for thinking about work A ? D
23. I often study purely for pleasure A ? D
24. I prefer to limit myself to reading recommended texts A ? D
25. I feel guilty unless I am working A ? D
26. I rarely complete my work to my satisfaction A ? D
27. If I get good marks I feel I don't deserve it A ? D
28. I get interested in a topic but soon lose interest A ? D
29. When working I continually break off to smoke or drink coffee or walk about to talk to someone A ? D
30. My hand gets stiff and clumsy so that I can't write properly A ? D
31. I enjoy discussing work topics with others A ? D
32. I consider the best way of learning is by completing the set work and doing the required reading A ? D
33. I get anxious (worried when I hear others talking about work A ? D
34. I am always planning out work schedules A ? D
35. I feel I out not to be taking up a place in the faculty A ? D
36. I frequently misplace my notes or text books A ? D
37. I don't worry enough about work A ? D
38. I am frequently distracted by aches and pains A ? D
39. I look forward to lectures or classes A ? D
40. I find a systematic presentation of a topic more usful than discussion A ? D
41. As soon as I start one task I feel I should be doing something else A ? D
42. I like doing things thoroughly, or not at all A ? D
43. My thinking about work matters seems very slow A ? D
44. My notes get in to a muddle A ? D
45. I keep wanting to sleep all the time A ? D

46. I am often handicapped by sheer physical tiredness A ? D
47. I like reading around my subject A ? D
48. I find it difficult to tackle something unles I know just what is expected A ? D
49. When I try to revise my work, my mind goes blank A ? D
50. I spend a lot of time on making preparations to work A ? D
51. I am often too depressed to concentrate properly on my work A ? D
52. I forget to go to tutorials or lectures A ? D
53. I read automatically without taking things in A ? D
54. I get a feeling of nausea and sickness when there is a lot to do A ? D
55. I would like to continue postgraduauate study or research A ? D
56. I don't let myself get diverted onto something that is not strictly relevant to the course A ? D
57. I am afraid of panicking (getting frightened) in exams A ? D
58. I find it difficult to decide which parts of my work are most important A ? D
59. I often feel that others know more A ? D
60. I keep getting out books but never really read them A ? D
61. I often think another subject would be more interesting A ? D
62. I suffer from eye strain when working A ? D
63. I believe in knowledge for its own sake A ? D
64. It isn's often I try to think of doing something differently from the way described in the lecture or book A ? D
65. When I am asked a question about work I dont know what to say. A ? D
66. I force myself to work even if I dont feel like it A ? D
67. I fear exams will expose all my weakness A ? D
68. I work in fits and starts (on & off) A ? D
69. I seem to have no real drive (enthusiasm) to work A ? D
70. I never seem to be able to get comfortable when trying to study A ? D
71. My interest in my subject grows continuously A ? D
72. I like to feel everything important is contained in my notes A ? D

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ASSOCIATION NEWS

SRI LANKA - INDIA DENTAL CONVENTION 1979

at The Sri Lanka Foundation Institute
106, Independance Square, Colombo

Date: **Friday 4th May at 3.30 p.m.**
Inauguration by Hon. **Ananda Tissa de Alwis** *Minister of State*

Saturday 5th May 8.30 a.m. to 4.30 p.m.
SCIENTIFIC SESSION —

Dinner Dance at Hotel Lanka Oberoi at 9.00 p.m.

Scientific Session — There will be a presentation of about sixteen to twenty papers from India and Sri Lanka delegates.

Childrens Dental Health Day in connection with the International Year of the Child in Sri Lanka sponsored by the Sri Lanka Dental Association. Friday 25th May, 1979.

67th Congres Dentaire Mondial. FDI
20th to 26th October, 1979 in Paris

for information write to:-
Dr. Alain Deyrolle
Secretary General of the F.D.I., World Congress
19 Bis Rue Legendre 75017
Paris. FRANCE.

Asian Pacific Dental Federation
9th Dental Congress 23rd to 27th April, 1979. in Malaysia.

The 1st International and 34th National Dental Conference organised by the Indian Dental Association will be held in Bombay in January 1980.

For information:-
Dr. G. R. Bhat, Hon. Gen. Secretary
Indian Dental Association
17, Purasawalkam High Road
Madras 600 007.

The Organisation of Professional Associations of Sri Lanka

Professional Centre:

The O.P.A., has obtained the approval of the grant of One Acre, of land in the Colombo Race Course Area, for the construction of the Centre. Tenders are called and construction work should start soon.

Member Associations requiring office space in the Professional Centre for their exclusive use, should pay 25%, before end of May 1979. The Sri Lanka Dental Association has requested office space of 200, square feet, at an approximate cost of Rs. 20,000/-.

Representatives of the S.L.D.A., in the O.P.A.

Dr. L. S. W. Dassanayake, *Vice-President.* Dr. S. A. Silva, *Asst. Secretary.*
Dr. M. M. Mukthar, *Forum Member.*

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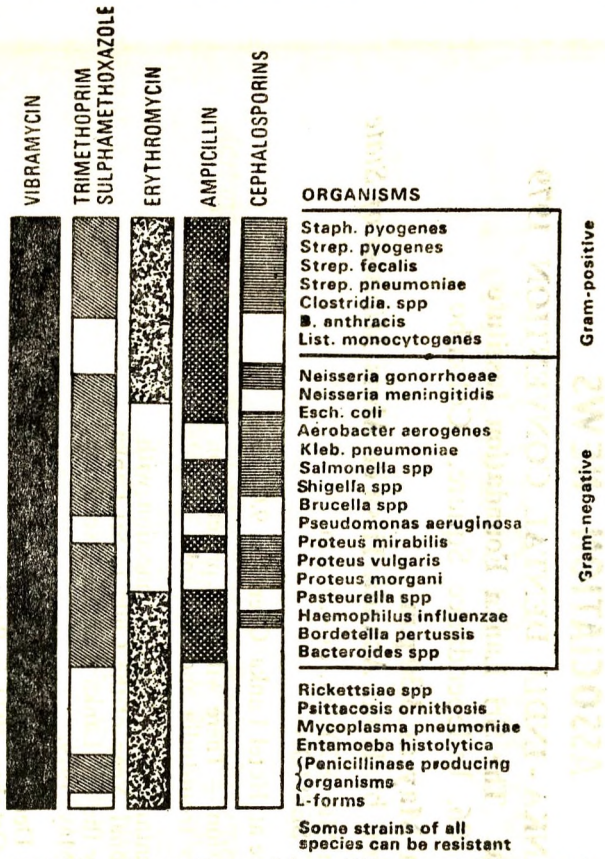

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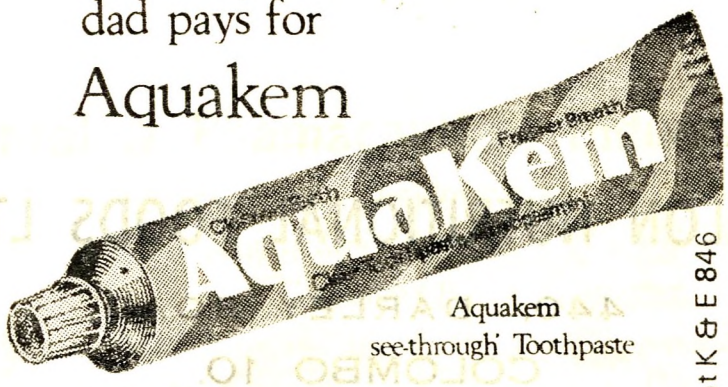

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