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Sri Lanka Dental Journal

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EDITORIAL

FIFTH TRIENNIAL MEETING OF THE COMMONWEALTH DENTAL ASSOCIATION

The fifth triennial meeting of the Commonwealth Dental Association (CDA), is scheduled to be held in Colombo, Sri Lanka from 01st of December to 3rd of December 2006. As editor of the *Sri Lanka Dental Journal*, the official publication of the Sri Lanka Dental Association, I feel it is incumbent on me to pen a few lines on this significant event and ponder its implications and ramifications for us in Sri Lanka and the rest of the Commonwealth.

The Commonwealth Dental Association consists of countries that once formed part of the fabled glorious British colonial empire where the sun never set. The inception of the CDA was the year 1991 and the charter of the CDA spells out its noble philosophy as "lending a helping hand to countries in need." With the prime objective of uplifting dental and oral health status, the CDA does yeomen service to the closely knit Commonwealth fraternity that is diverse in social, economic and cultural parameters, comprising both rich and poor countries and accounts for a staggering 1.8 billion people spread through 53 countries of the world.

Development and introduction of effective and novel primary health care strategies, technical

other auxiliary oral health care workers, delivery of cost effective oral health care programmes and facilitating, continuing dental education programmes, distance learning programmes, training, and sharing expertise are some of the services rendered by the Commonwealth Dental Association. The Commonwealth Dental association is also responsible for enhancing the importance of oral health within the Commonwealth bodies of countries and acts as a strong lobby that influences government policy makers and decision makers on the need to promote oral health and primary care strategies for the benefit of the people, especially in the less developed countries of the Commonwealth.

This triennial conference will strengthen the links and affiliations between the Commonwealth states as well, and all this, translates for the benefit of the Commonwealth that is fast becoming a very influential world body and augurs well for the future. We in Sri Lanka extend our fullest support and blessing to this event and wish its deliberations all success.

Upul B. Dissanayake
Editor, SLDJ

Message: chairman organizing committee of the fifth triennial meeting of the Commonwealth Dental Association

The Commonwealth Dental Association was formed on 25th April 1991 in Kuala Lampur, Malaysia. The decision to form such an association was taken at a meeting of the Commonwealth Oral Health Initiative Conference in London which was supported by the Commonwealth Foundation.

As the Vice President of the Commonwealth Dental Association it is my honour and privilege to have the opportunity to participate at the 5th Triennial Meeting as its Chairman of the Organizing committee. The other meetings were held in Trinidad, Bournemouth, New Delhi and Nairobi.

The objectives set out in the association are very noble, important and practical suggestions to bring about better oral health to the people of the Commonwealth countries in particular and the world in general.

The first objective is to develop and provide primary oral health care strategies, using appropriate technology with an emphasis on the prevention of oral and dental diseases. The Commonwealth Dental Association closely collaborates with the respective national organization in achieving this objective.

The 2nd objective of the Commonwealth Dental Association is to provide technical cooperation and advice to member association of countries within the commonwealth. Particularly in the training of a professional and auxiliary oral health work force and the organization of a cost effective delivery of oral health care. The Commonwealth Dental Association has arranged a symposium in oral health workforce in the Commonwealth-priority issues, which will deliberate on issues such as, training needs of various types of dental professionals, continuing professional development, demand for the work force and how they could be retained in their own country. These deliberations will give us an insight into how improvements could be achieved in these areas.

The discussions that could revolve round the ethics would make us comply with the objective of promoting the ethical practice of dentistry among member associations. Amongst the other objectives is that the Commonwealth Dental Association to facilitate continuing dental education programs including distance learning for the dental surgeons and oral health care personnel working in the Commonwealth countries. The Sri Lanka

Dental Association has arranged a comprehensive scientific program as a continuing professional development for the Dental Surgeons in the Commonwealth.

The Commonwealth Dental Association has sponsored the participation of five eminent international speakers including the President of Federation Dentaire International, Dr. Michelle Aerden.

I hope that all these efforts by the Commonwealth Dental Association and Sri Lanka Dental Association would percolate to the community to give them a better oral health care service. I take this opportunity to thank all those who have demonstrated their corporate social responsibility by assisting in this endeavour. We are grateful to M/s Unilever (Signal)who are the principal sponsors, the other sponsors, members of Commonwealth Dental Association executive and Sri Lanka Dental Association organizing committee.

The Commonwealth Foundation has played a significant role in this conference by sponsoring the visits of the Commonwealth Dental Association executive and some speakers to this event. They deserve a special word of thanks.

Hilary Cooray

Leading Article

Sri Lanka Dental Journal 2006; 36(03)110-114

Prevention of maxillo-facial injuries

N.A. de S. Amaratunga

Introduction:

Causation of maxillo-facial injuries (MFI) is related to social, economical, and political activities of a particular society. The pattern of these injuries including the site, severity and concomitant injuries would to a large extent depend on the cause which in turn is strongly associated with the afore mentioned activities of the society. The whole pattern could undergo change when these activities change. A classical example is the changes that were observed in the pattern of MFI in Sri Lanka after the liberalization of its economy in the late seventies. These economic measures had a far reaching impact and repercussions on most aspects of our culture. Consumerism well and truly took hold of the society and income generating activities had to enhance to support the newly created wants.

Mobility of the people, competition and rivalry among them, industrial and sports activities had to keep pace with the rapid changes that were taking place. These economic, political and social implications make the causation of MFI highly complicated. For instance alcohol restriction measures have to contend with the fact that alcohol is one of the highest revenue earning items and the fact that liquor manufacturers have a heavy political clout. Another aspect often not taken into serious consideration is the spiritual decline that accompanies growth of consumerism and its effect on the causation of MFI.

Complexity of the aetiological pattern

As mentioned above the aetiology of MFI would depend on the social, economic and political activities of a given society.[1,2] When these activities undergo major changes the aetiological pattern of MFI would show changes and as a result the incidence and severity of injuries too would change. A classical example of this phenomenon is the changes in MFI patterns observed after the liberalization of the economy with the change of governments in 1977. Falls were the commonest cause of MFI before these economic changes were adapted and road traffic accidents (RTA) were the next common while assault came third.

By mid eighties this pattern had changed and, RTA had become the number one causative factor while assault had advanced to second position and falls which was the number one factor prior to the economic liberalization had fallen to number three position. More importantly there had been a three fold increase in the incidence of MFI in the year 1990 compared to 1975.[1] Significantly the influence of alcohol on all three major causative factors had risen during this period. The economic changes mentioned above had apparently resulted in an increase in the mobility of people, interaction between people and consumption of alcohol. The complexity of the aetiology of MFI has to be taken into consideration when planning their prevention.

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In the final analysis development of the economy sans its ill effects must be the goal which seems to be elusive in most of the developing countries.

Prevention of road traffic accidents

What has to be done to prevent RTA is well known but effective implementation and enforcement of preventive measures and legislation are difficult to achieve in developing countries. Preventive legislation such as speed limits, alcohol restriction, helmets, seat restraints, improved road construction and safety measures in vehicles such as safety glass, stronger frames, collapsing steering columns, air bags etc. have been effective in reducing the incidence and severity of RTA injuries.[3]

Yet non compliance is the main reason for the difficulty in preventing RTA to a more satisfactory degree even in the developed countries.[3,4,5,6,7] Provision of seat restraints is ineffective unless its use is made compulsory by law.[3] Enactment of seat belt law alone is ineffective unless police surveillance is developed to a satisfactory level. When all these factors are taken care of there is the problem of young adults who are reluctant to use seat restraints.[3]

In Sri Lanka legislation pertaining to most of the contributory factors in the causation of MFI is either absent, inadequate or their enforcement is ineffective. A seat belt law is still not in place though most of the vehicles now have seat belts. The law against driving under the influence of liquor is in place but the police are ill equipped to enforce the law to a satisfactory degree. Same could be said about speed limits.

The volume of traffic on the roads is said to be a factor in the causation of MFI and it is directly related to RTA. This was the reason for the rapid increase of RTA caused by MFI after the import restriction on vehicles was lifted in the late seventies. Vehicular mass and velocity are the more important factors in determining the severity of MFI.[8,9] When the number of vehicles on the roads increase without a corresponding expansion of road network RTA are expected to rise. However under these conditions high speed may not be possible as is being experienced on some roads in Sri Lanka. Though the number of accidents has increased the severity of injuries has lessened because high speed was not possible when traffic volume is large on narrow roads.

Road construction with improved safety measures have not kept pace with the rise in traffic volume. Separation of pedestrians from traffic stream, dual carriage, multi lane highways etc. have to be introduced if modern vehicles are to make full use of the sophisticated facilities fitted to these vehicles specially the speed and acceleration options. It is ironical that Sri Lanka can afford to buy such modern vehicles in large numbers, but cannot afford a thorough modernization of the roads. This kind of mismatch contributes to chaos rather than development which is being experienced on our roads at present.

In Sri Lanka, unlike in developed countries pedestrians are the most commonly affected road user in RTA.[1] and this could be attributed to lack of pedestrian safety measures. Protected pedestrian pavements, crossings, warning signals, etc. are not in place except in few towns Street hawkers appear to have political clout and audacity to construct permanent structures on the pavements meant for pedestrians. The police have to turn a blind eye as they cannot prosecute the offenders, nor could they stop the "jay walkers". Thus the situation that exists on Sri Lankan roads is a classic example of chaos due to living beyond the means which has come about due to the society moving away from spirituality towards

consumerism. This aspect would be further discussed later on.

Prevention of Assault

The increase in the incidence of assault after the liberalization of the economy is often attributed to the increase in interpersonal rivalry which is a result of competition which in turn is a phenomenon observed in market economies.[10,11] This is rather a superficial analysis lacking in historical perspective and human behavioral considerations. Historically the Eastern societies in the medieval times were not based on the economy but on spirituality. The western societies on the other hand were based on economy. The former system was designed to nurture cooperation while the latter promotes competition and conflict (Fig. 1)

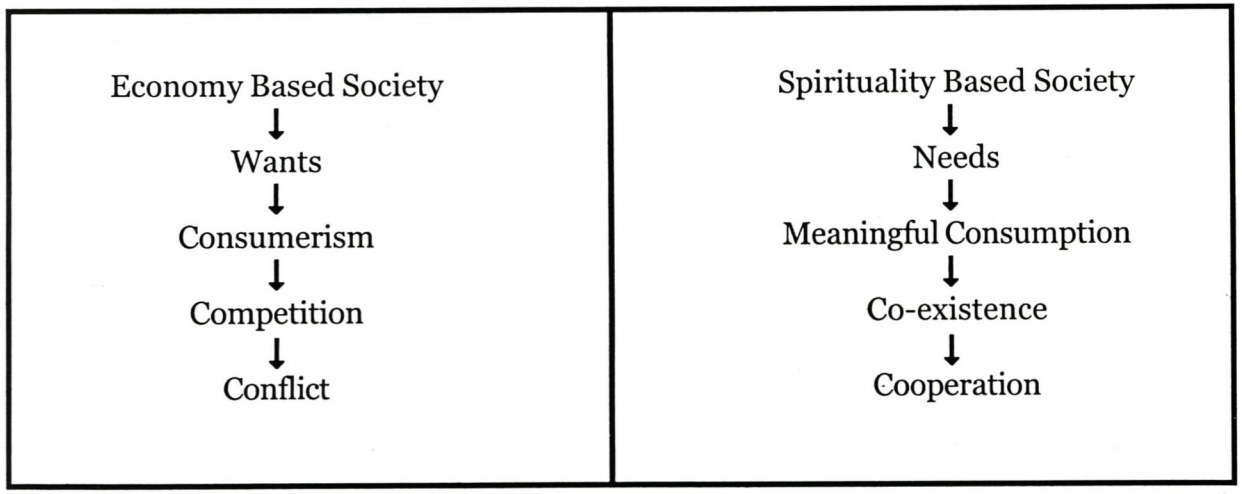

Figure 1.

In western countries assault commonly occurs in public houses under the influence of liquor. Assailant and the victim apparently in most instances have had no past animosity towards each other. It is alcohol intoxication that lead to an altercation and assault.[10,11] This shows how rivalry that lies dormant in the subconscious is aroused when inhibitions are removed due to intoxication. Reasons for assault in Eastern countries are not so trivial. Assailant and victim had been long time enemies. Alcohol may play a part but it is not the main precipitating factor as in the west.

Thus it is clear that assault could be prevented only by the practice of activities that enhance the spirituality of a society. The western model (Fig 2) is not suitable for this purpose. Obviously animosity, hatred and conflict are more likely to develop in a society where consumerism and acquisitiveness have displaced human values.

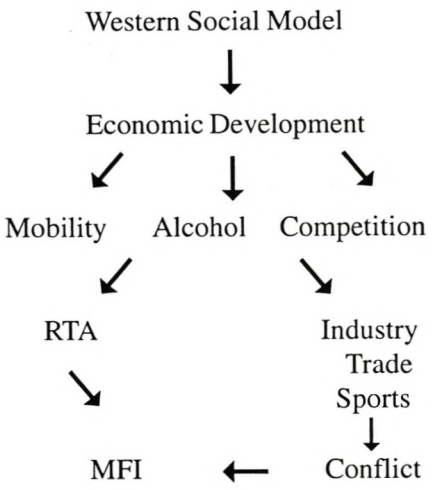

Figure 2.

Role of alcohol

Alcohol is one of the common addictive substances in use. It is also one of the highest revenue earning market items for governments in most of the countries. Also alcohol production and marketing is extremely lucrative. Because of this combination of factors alcohol restriction is a difficult proposition. Founders of ancient religions probably knew about its ill effects and had advised people to refrain from consuming alcohol and thus it forms one of the five precepts in Buddhism. Yet alcohol consumption is on the rise in most of the countries.[3,7,10,11]

As mentioned earlier alcohol has an adverse effect on all the causes of MFI. Incidence of RTA, assault, falls, sport and industrial accidents could rise due to the influence of alcohol.

In the western countries influence of alcohol on the incidence of MFI caused by assault seems to be greater than that of other causes. This seems to be the reason for MFI caused by assault to gradually increase and in some countries like Finland assault has became the number one cause of MFI.[10]

While the ban on drunken driving has resulted in a reduction in the incidence of MFI caused by RTA where the drivers had been under the influence of liquor, no decrease in MFI caused by assault due to intoxication has been observed.[7,10,11]

In these countries no action has been taken to reduce social drinking though there is legislation to restrict alcohol in sports and industrial activities.

Social structure

In capitalist societies the basic social structure is economic or materialistic which determines the spiritual. This is in stark contrast to the system that was in operation in the middle ages in Eastern countries where the structure of society was based on spirituality which determined the material needs. Economy developed on the basis

of material needs. Under capitalism the economy assumes primary importance. Economy determines the material needs and also the spiritual needs. If spirituality is allowed to rise up above materialism the economy would suffer. Capitalism cannot allow that to happen. Therefore it has to suppress spirituality and allow consumerism to be the driving force in the life of human beings. Under these conditions less people practice their religions, there is more competition leading to conflicts, there is more stress leading to heavier dependence on alcohol and other stimulants, there is greater mobility as people run about in search of money leading to more accidents. The cumulative effect of all these ills is discontent, disease and trauma. Thus it is difficult if not impossible to curb the social ills that contribute towards the occurrence of injuries by the introduction of safety measures and legislation alone. A return of the social structure to its spiritual base is also necessary.

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Review Article 1

Sri Lanka Dental Journal 2006; 36(03)115-124

Globalization health and dentistry

A.N.I. Ekanayaka

"Globalization" is a word that is on everyone's lips nowadays. It is a phenomenon that evokes strong feelings and has polarized world opinion either for or against globalization. However, whatever our attitude towards it the very existence of such heated debate and widespread concern suggests that globalization is both real and ubiquitous. Clearly something has changed and something is happening in our world which has profound implications for every aspect of human existence. In the face of this reality it is a pity that there has been hardly any serious analysis of the impact of globalization on dentistry. This may be predictable considering the narrow technical and mechanical focus as against a broad holistic perspective on health that has been the bane of dentistry throughout history.

A cursory search of any electronic research database reveals the existence of many thousands of publication on globalization. On the other hand a Medline survey of papers pertaining to globalization and health found 882 publications which included only about a dozen publications on globalization and oral health of which the excellent analysis on economic globalization and oral health by Hobdell[1] seems to be the most pertinent. The implications of globalization for dentistry arise out of the broader effects of globalization on health, which in turn are grounded in an understanding of the nature and manifestations of globalization itself. Accordingly this review will firstly aim to clarify our understanding of globalization, its effects, and the issues surrounding it. Secondly we will examine the relationship between globalization and health, and thirdly identify some of the specific implications of globalization for dentistry.

The nature of globalization

Taken to the extreme globalization involves a set of processes leading to the creation of the world as a single entity (or at least an "inter - connected world") relatively undivided by national borders or other types of boundaries.[2] Globalization has also been described as "the flow of information, goods, capital and people across political and economic boundaries."[3] Definitions and descriptions of globalization are replete with expressions like "intensified human interaction", "economic interdependency", "shared identity", "shared society", "interdependence of nations", "global cosmopolitan society", and such expressions give us some idea of the general direction in which a globalized world seems to be heading. In an article in the New York Times Crossette[4] pointed out that " globalization means many things to many people. It is not simply the greater movement of goods, jobs and capital across borders but also includes equally important cultural, environmental and political components".

What it all means is that in our modern world notwithstanding national boundaries nations have become increasingly interdependent and the

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lifestyle and happiness of their subjects increasingly bound up with what happens elsewhere in the world. Whether we like it or not this is the reality and we must come to terms with it and make the best of it. As Sir Anthony Giddens the distinguished director of the London School of Economics observed in the first of his 1999 BBC Reith lectures on globalization " ... globalization is not incidental to our lives today. It is a shift in our very life circumstances. It is the way we live now."[5] Not surprisingly the famous lecture series (which can be heard on the web) was entitled "Runaway world". While Tony Blair[6] has estimated the extent of this transformation in terms of the "fragility of our frontiers in the face of the world's new challenges" others like the Japanese business writer Keniche Ohmae have been reported to euphorically proclaim the era of the nation state as being already finished with nations becoming mere fictions.[5] To the historian Eric Hobsbawn[7] one of the great achievements of the 20th century was the virtual annihilation of time and distance. The Canadian scholar Marshall McLuhan coined the metaphor "global village" as epitomizing the coming together of people of different nations and cultures through technological advances.[2]

If globalization represents a "shrinking" world and greater interactions across cultures there is a sense in which globalization is not new. Human beings have fanned out across the earth from time immemorial exploring, trading, forming alliances and conquering. The advent of steam ships, aeroplanes, printing, radio and telephones accelerated this process and yet all this took place a very long time ago. Cynics have argued that the globalization of disease may have begun in 1492 when the Europeans discovered America and inflicted genocide on the native Indians while introducing smallpox, yellow fever and measles as well as firearms !. Nevertheless what is unique and exciting about globalization in our day and age is the sheer scale and pace of change. For example in 1950 there were 2 million international airline passengers. By 2000 this had risen to 1.4 billion. The first communications satellite was launched about 35 years ago. Today there are more than 2000 such satellites in orbit. It was 40 years before radio in the USA extended to an audience of 50 million. By contrast 50 million Americans were using the internet just four years after it was introduced. It has been estimated that today more than a trillion dollars are turned over in currency markets daily which is a huge increase compared to 10 years ago.[5] Clearly the phenomenon of globalization has overwhelmed human societies in our day and age in a manner and to an extent with which past trends do not bear comparison.

Globalization has at least six main dimensions of which the most obvious is *economic* globalization. It involves the inescapable economic interdependence of modern States amidst the huge transactions on global currency markets, the massive proliferation of global trade in an era where the free market has become the dominant economic ideology, and the power of international monetary organizations and the structural adjustment policies they impose on poor countries. It is interesting to note that the World Bank has how overtaken the WHO as the largest donor of foreign aid for health care. At the end of 1999 the Melinda and Bill Gates foundation committed $6 billion for vaccines. This contrasts with WHO's annual budget which is less than $1 billion and reflects the growing role of public private partnerships as a dominant player in the global economy.[8]

Globalization is also *environmental* because the harmful environmental impact of economic growth and rising affluence in some parts of the world affects all humanity globally. The terrors of environmental pollution, deforestation, desertification, climate change, ozone depletion, and global warming transcend national boundaries and threaten the very sustainability of life on the planet.

Globalization is *cultural* because the diffusion of western lifestyles in so many areas including sexual morality, dietary practices, fashion, entertainment and culture has broken down barriers and induced cosmopolitan lifestyles. The popularity of Coke, American fast food products and western videos and pop music in the remotest parts of the world is testimony to this trend.

Globalization is *political* because global economic interdependence has severely reduced the power of sovereign governments to control the destinies of their nations.

Globalization is *technological* because of the rapidity with which communication technology is permeating to every nook and cranny of the world enabling the constant exchange of information and bringing people closer. Amidst the marvels of fax, the internet, and satellite TV, in 2001 traffic on international switchboards topped 100 billion.

Finally globalization is *ethical and judicial* because of the increasing pressure for nations to conform to global standards of excellence and civilized norms of human conduct that are universally accepted.

This is the multidimensional framework within which globalization has transformed the way we live. It has brought both benefits and disadvantages. From a philosophical perspective it epitomizes the great ideal of a common humanity. Openness to foreign trade and investment can trigger global affluence increasing incomes and accelerating economic growth and development in poor countries. India, Vietnam, China and Uganda have been cited as examples.[9]

In an interconnected globalized world there are liberal enlightened global values and standards of law, governance and social justice that all civilised States are expected to observe. These include freedom, democracy, transparency, media freedom, human rights including the rights of women and children, protection of the environment, and health promotion. Governments can no longer tyrannise their own population with impunity, hide the facts, and taking cover under the antiquated dogma about non interference in the internal affairs of a country expect the rest of the world to turn a blind eye when crimes against humanity are perpetrated on their own people. In his Reith lecture Anthony Giddens explained that globalization was an important factor in the collapse of the autocratic communist regimes in the Soviet Union and Eastern Europe. He argued that "the ideological and cultural control upon which communist political authority was based could not survive in an era of global media."[5] The global spread of technological advances is a further benefit of globalization. In particular the communication revolution through the global dissemination of information technology has vastly expanded the scope for education, culture, and entertainment, thereby enhancing the quality of life of millions throughout the world. Finally globalization is credited with spurring the revival of local cultural identities (for example in the countries of the former Soviet Union) a process that has paradoxically gone hand in hand with globalization's push towards a global cosmopolitan society.

However globalization has negative features and its detractors have been scathing in their criticism.[10] Mahathir Mohamed former Prime minister of Malaysia has warned developing countries against embracing globalization and has been quoted as saying that the " .. blind acceptance of an ideology that to date stands as just that – an ideology – is unacceptable, naïve and downright dangerous", while President Musaveni of Uganda has said that globalization was "the same old order with new means of control, new means of oppression, new means of marginalisation."[10] The main thrust of such criticisms is that globalization is a modern form of economic and cultural imperialism by powerful industrialised countries, and that it involves the subjugation of the economies of developing countries to the interests of industrialised countries

and powerful transnational corporations, and the substitution of traditional culture, values, and lifestyles by a global western culture. However, the most important debate about whether globalization is "good" or "bad" for mankind centres around the question whether it has been responsible for dramatically increasing social and economic inequalities both within and between countries.[11] As Anthony Giddens[5] puts it " Globalization, some argue, creates a world of winners and losers, a few on the fast track to prosperity, the majority condemned to a life of misery and despair." And so the debate continues.

Unraveling the issues involved goes beyond this review. However both globalizers and anti globalizers agree that vast social and economic inequalities are an unpleasant reality which must be addressed. The statistics are both prolific and alarming. 20% of the world's population live in absolute poverty with an income of less than $1 per day.[8] Absolute poverty is increasing with about 600 million children living in families unable to afford the very basic necessities of life while women comprise 70% of the absolute poor. 90% of the disease burden exists in developing countries yet they benefit from only 10% of the resources allocated to health.[12] However, while all parties concede the existence of gross inequalities there is debate whether or not such disparities are increasing and if so whether the blame must go to globalization as against other factors that may be responsible for such trends.

David Dollar[9] has argued that more open international trade has in fact narrowed the gap "between" rich and poor countries. As for inequities "within" countries, he concedes that some poor families are impoverished in the short run by trade liberalization making it necessary for open trade policies to be complemented with effective social support involving unemployment insurance and food for work schemes. China is cited as an example of a country where the increase in inequality has been quite steep despite a massive decline in rural poverty overall from 250 million in 1978 to 34 million in 1999.[9] On the other hand in other globalizing countries like Vietnam, Malaysia, and the Philippines there has been either no change in household inequality or even a modest decline. Such contradictions may highlight the need to disentangle globalization as a cause of rising social and economic inequalities in developing countries from corruption, waste, excessive bureaucracy, too much state power, poor governance, demographic changes, and the AIDS epidemic that may be the real factors responsible for growing poverty and inequality. Nevertheless the possible contribution of globalization to growing social inequity is a question that will continue to trouble the world especially in view of the recent convincing evidence about the impact of such inequalities on population health.[13]

Globalization and health

Given the nature of globalization and the known determinants of population health it should come as no surprise that for better or for worse they are strongly interrelated. The impact of globalization on health has been analyzed in terms of three types of changes that have swept through human societies in recent decades and the multiple health implications of such changes.[14] They include firstly *spatial changes* where globalization affects how we perceive physical or territorial space in a world of intense movement of people, other life forms, information, capital goods and services across national borders. Secondly, there are the *temporal changes* involving how we perceive and experience time in a high pressure world of modern communication and rapid transportation that is "hooked on speed."[15] Finally, there are the *cognitive changes* involving how we see ourselves and the world around us in a connected world where our cultures, knowledge, needs, values, beliefs, knowledge and aspirations are shaped by a myriad influences that battle for our hearts and minds.[14] More recently Huynen *et al*,[16] proposed a comprehensive conceptual framework which explains the numerous direct

and indirect ways in which globalization affects population health.

The conceptual model integrates the four major ecological, socio cultural, economic and institutional determinants of population health with the six main pillars of globalization namely, new global governance structures, global markets, global communication, global mobility, cross cultural interactions and global environmental changes. Within this framework the chain of events linking population health and globalization are considered to take place at three hierarchical levels of causality. They include proximal causes (meaning direct and immediate effects on health eg. through the workings of health services), distal or intermediate causes, and further back contextual determinants which are the macro level conditions that shape the proximal and distal determinants.[16] While the specific ways in which globalization can have an impact on health may be deduced within such a conceptual framework they may be itemized as follows for clarity,

- The spread of diseases like HIV/AIDS and SARS caused by the intensified migration of people throughout the world, and the economic burden to richer countries of unhealthy populations migrating from poorer countries.[17] The speed of modern transportation can further contribute to the rapid global spread of infectious diseases across national borders as with the SARS epidemic of 2002-2003.[18]

- The loss to poorer countries of skilled professionals including health professions migrating to "greener pastures" in the highly industrialized countries

- The globalization of the tobacco and alcohol industries, and the global popularization of fast foods, sugar products, and related western dietary patterns through intensive international marketing may promote unhealthy lifestyles in poorer countries causing diseases of affluence, dietary excess and sedentary living.

- The globalization of trade and food production which has led to the transmission of food born diseases[19] and trade in infected biological products which can contribute to the spread of HIV/AIDS and Hepatitis B.[20]

- The increased opportunities for trafficking in illicit drugs, and smuggling of substances harmful to health in a globalized world, where national borders have become increasingly permeable.

- The impact on national health policies of the emergence of global governance structures like WHO, the World Bank and the World Trade Organization. It has been argued that already "the centre of power for global health governance has shifted from WHO to the WTO."[21]

- Poverty is a major determinant of poor health. Poverty affects nutrition, housing, and sanitation as well as access to care.[1,22] Globalization can affect health for better or for worse depending on whether it reduces or increases poverty and inequality – an issue that has already been touched on. Economic globalization has enabled organizations like the IMF to enforce structural adjustment policies on poorer countries. These include cutbacks in public spending, privatisation of government enterprises, less government interference in the marketplace freezing of wages and freeing of prices, increased taxes especially sales tax, increased production including food for export, reduction or

abolition of exchange controls, and priority to the productive sector over the social sector. The health impact of such policies is a mater of debate. However, they can potentially increase poverty and accentuate health inequalities through reduced government spending on health resulting in fewer doctors in the state sector and cutbacks in curative and primary health care services. Moreover with the introduction of "user charges" health services become more market driven reducing access to the poor who will make extra demands on the already weakened state services. Essential drug policies may tend to get sidelined and amidst irrational prescribing practices and rising drug prices in an increasingly market oriented health service public health will be further undermined.

- It is universally accepted that social and psychological stress is an important social determinant of health.[23] Stress can damage health by depressing the immune system and also by inducing compensatory risk behaviours like smoking and drinking. People may also try to cope with stress by frequent sugar consumption.[1] The poor health of populations living under conditions of relative poverty (as against absolute poverty) is one of the dominant themes of contemporary public health[24] and it has been postulated that the stress induced by relative poverty is what damages health.[13] Economic globalization may be an important factor in this cycle if it increases relative poverty.

- Instant global connectivity resulting from the electronic revolution, especially the internet has created phenomenal opportunities for medical education through the dissemination of knowledge and the exchange of medical information. The increasing export of medical education, and the proliferation of split training pathways, exchange programmes, foreign elective programmes, international scientific meetings, and medical journals - reflect the internationalization of medical education in an increasingly connected world.

- The global connectivity of international health organizations coordinated by WHO enables the early detection of new infections and rapid and informed response to potential global health emergencies. Telemedicine has opened up new horizons in health education, medical diagnosis and treatment. On the other hand such advances have raised new ethical, and regulatory issues and questions about privacy of patients. Meanwhile the frenetic marketing of drugs through the internet may compromise rational prescribing encourage drug abuse and increase the likelihood of drug resistence.[8]

- The liberalization and privatization policies accompanying globalization have various implications for food and water security

- Global mobility, the ease of global communication, and heightened cross cultural interactions have the potential to either increase or decrease the likelihood of human conflicts with their terrible toll on human health. "International terrorism" is one of the depressing realities of a globalized world.

- Global trade agreements mediated by WTO and other related international covenants governing intellectual property

rights like the TRIPS agreement may put poorer countries at a disadvantage. On the positive side global covenants like the WHO framework convention on tobacco control and the United Nations Kyoto protocol on climate change, are examples of globalization helping to create an international consensus on issues that have a bearing on health.

- There are also the global environmental consequences of globalization and their impact on health and life itself. These include the environmental effects of the deregulation of trade and frenzied unsustainable economic growth spurred on by the insatiable demand of modern societies for ever more comfort, luxury, and affluence.

The above are just some of the ways in which globalization may impact on health. However, the associations are much too general and a lot remains to be learnt about the specific impact of globalization on health. It has been rightly asserted that " Despite some empirical research efforts indicating the links between globalization process and specific health impacts, the present weakness in empirical evidence on the multiple links between globalization and health is still a problem."[20]

Globalization and dentistry

The impact of globalization on dentistry can be deduced from an understanding of its impact on general health. This is to be expected considering that oral health is an integral part of general health. In addition to the well known oral manifestations of systemic disease recent research has shown that oral disease can compromise general health in various ways.[25] Furthermore as populations age worldwide dentists will have increasing responsibilities for the treatment of the elderly many of whom might be medically compromised. Moreover today both the prevention of disease and the promotion of health are approached through a "common risk factor" approach which again compels a closer integration between oral and general health and their respective workers. Accordingly many of the implications of globalization for health have direct or indirect relevance to dentistry. A good example is the foregoing discussion about the impact of poverty on health which is directly applicable to oral health. Hobdell[1] has reviewed the relationship between oral cancer, dental caries, periodontal disease and cancrum oris and social deprivation. Social deprivation is a major threat to oral health through its manifold effects on oral habits, diet and nutrition, access to care, attitudes and various psychosocial pathways. Consequently the on going debate regarding the impact of globalization on poverty and inequality is highly relevant to the future of oral health and the practice of dentistry.

One of the most vivid examples of globalization's influence on oral health is etched into the very history of dentistry and goes back to an age many decades ago even before globalization came to be recognized as a distinct entity . The export of dental caries from highly industrialized countries to parts of the world where the disease was previously unknown in indigenous populations which had hitherto consumed a coarse natural diet, is one of the best known historical studies in dental epidemiology.[26,27] More recently the popularization of sugar rich dietary patterns as part of the global permeation of western lifestyles, supported by the powerful advertising and marketing of sugar products worldwide, has contributed to the increase of dental caries in several developing countries.[28]

Meanwhile there are concerns whether the worldwide popularity of acidic carbonated soft drinks amongst young people may increase the incidence of dental erosion which may emerge as a new public health problem.[29] On the positive side global marketing and the unified

recommendation of the dental profession worldwide has made fluoride toothpaste a household word throughout the world. It has been responsible for the dramatic decline of dental caries in the past and is the single most important factor on which the potential for controlling dental caries in the future is based.

Global dental partnerships like WHO, FDI, IADR, IDM, IFDEA are another aspect of globalization that is influencing dental research, practice, education, and policy worldwide. To take an example from dental education, a WHO Expert Committee in 1990 formulated a general set of aims and objectives for a first course for oral health professionals suitable for the early 21st century in any part of the world.[30] The WHO Oral Health Programme[31] contributes to the implementation of the Global Strategy on Diet, Physical Activity and Health.[32]

Even more important is the global imperative of evidence based dentistry threatening isolationism and obsolescence in dental practice and compelling the adoption of rational therapeutic and prevention regimens that stand up to the scrutiny of science. The lack of evidence based support for rigid six monthly dental recall intervals, frequent scalings, chairside dental health education, the removal of asymptomatic third molar teeth, invasive treatment modalities with a strong technical bias - and conversely the emergence of a basis of scientific evidence favouring the least invasive biological and behavioural management of dental caries, the more conservative management of periodontal disease, the acceptance of a functional dentition in older individuals, and cost effective treatment modalities – have shaped the character of modern dental care across different cultures.

Dentistry has also benefited from the development of standardized survey methods[33] enabling the documentation of global oral disease patterns.[34] Such information, the guidance provided by global oral health goals[35] and the shared experience of nations with different systems of oral health care provision[36] have made it possible for countries to plan their oral health systems on a rational basis with the valuable hindsight of past global experience.

In our globalized world changing disease patterns, the dazzling scientific and technological advances of recent decades, and the rising expectations of modern sophisticated consumers have given dentistry a radically new attractive image. Dentistry today at its best is a complex cosmetic art capable of dramatically transforming facial appearance and oral function virtually in any age group, thereby magnifying personality and self confidence – values which are highly cherished in modern materialistic competitive societies. Consistent with recent advances in dentistry the workload of the dentist of the 3rd millennium will ideally be dominated by advanced diagnostic procedures and complex cosmetic and rehabilitative care. He/she will be firmly grounded in evidence based dentistry and will be skilled in the art of empowering patients in an efficient and cost effective practice where the dentist will manage a team and work with auxiliaries. He/she will be knowledgeable in investigating and controlling risks and integrating oral health with general health in patient care. While forming useful alliances with other health workers there will be involvement in advocacy and education. Such a "new age dentist" functioning in an active market environment will be rich and fulfilled and more correctly merit the title of "oral physician".

What is depressing is that this scenario may be a reality for only a small fraction of a globalized world in which total per capita spending on health ranges from International $ 5711 in the USA to $ 121 in Sri Lanka![7] The modern dentist is the creation of modern dentistry, and modern dentistry is the invention of affluent industrialized societies whose populations are able to afford the superlative treatment modern dentistry affords at the price tag that it carries. Globalization has

poor countries whose weak economies cannot sustain dental practice at that level, except for a minority of patients who can afford to pay the high costs involved, and a minority of dentists who have the financial strength to acquire the costly practice infrastructure required. This will only further exacerbate health inequalities in the population and practice inequalities in the profession. Hobdell[1] has succinctly encapsulated the potential predicament that faces dentistry caught up in this contradiction in a globalized world " For those dentists serving population groups whose socioeconomic status is improved by economic globalization, the practice of elective dental care as sophisticated as anywhere in the world will become possible. For those dentists serving population groups who become impoverished by the process of economic globalization, unless they adapt their dentistry and develop new ways of meeting the growing and possibly changing oral health needs, there is little to look forward to".

Clearly the tide of globalization cannot be turned back. Countries both rich and poor have no alternative but to look upon globalization as an opportunity, extract the best that it offers and judiciously adapt and harness it for the health and happiness of their people. This will require a level of good governance that has proved elusive in Sri Lanka as in many other poor countries.

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Review Article 2

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Application of molecular biology techniques in dentistry

P.R. Jayasooriya, B.R.R.N. Mendis, T. Lombardi

Abstract

Integration of genetic information to our understanding of growth of the cranio-facial complex in health and diseases will greatly change management of dental patients in the near future. This genetic information is mainly obtained using molecular biology techniques by analyzing DNA, RNA and proteins in tissues. One of the most revolutionary of these techniques is the polymerase chain reaction (PCR) which relies on exponential amplification of specific DNA segments, resulting in millions of copies that can serve as templates for different kinds of analysis. At present molecular biology techniques have contributed to dental research and show potential to be used for prevention, diagnosis and treatment of many simple as well as complex hereditary oral conditions. Therefore, the purpose of this review is to provide an overview of important molecular biology techniques and to summarize current and possible future applications in dentistry.

Key words: Molecular biology, Polymerase chain reaction, simple and complex hereditary oral conditions

Introduction

Molecular biology, the science that deals with cell development and control is largely based on investigation and analysis of DNA and RNA. Recent advances achieved in molecular biological techniques have permitted insights into many developmental, infectious and neoplastic diseases. In addition, it is also now known that genes play an important role in the aetiology of disease.[1] Although, molecular biological techniques are not used routinely in dental practices at present, integration of currently available knowledge to the development of cranio-facial complex may provide new approaches to prevention, diagnosis, risk assessment and management of both common and rare dental disorders in the near future.[1,2,3] As such, dental surgeons will require knowledge of human genetics and the application of new molecular based diagnostic and therapeutic techniques in the future.

Therefore, the aims of this review are to

1) provide an overview of important molecular biological techniques such as the Polymerase Chain Reaction (PCR), Hybridization techniques namely Northern, Southern and Western blotting and DNA/

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c-DNA micro-arrays and their current applications in dentistry.

2. summarize the knowledge gained with reference to
   a) complex hereditary oral conditions such as dental caries, periodontal disease,oral cancer and odontogenic tumours.
   b) simple hereditary conditions such as developmental disorders of teeth using molecular biological techniques.

Overview of techniques

1) Polymerase chain reaction

PCR, the enzymatic amplification of a specific DNA sequence *in vitro* is based on the replicating capacity of DNA.[4,5] The initial step in any PCR reaction is the isolation of DNA. DNA can be extracted from many different samples such as blood, saliva, sputum. In addition to fresh tissue specimens, PCR can be used to amplify relatively degraded DNA extracted from formalin fixed-paraffin embedded tissue specimens.

A single DNA strand is used as the template for the synthesis of new complementary chains under the action of DNA polymerase enzyme, which is able to bind nucleotides present in reaction to the template. However, DNA polymerase requires a starting point in the template which will direct the addition of subsequent nucleotides. This starting point is provided by an oligonucleotide that hybridizes to the template strand, which is known as the primer. (Both original strands of DNA can act as templates when specific primers are provided to each strand) (Fig1).

As such PCR requires

* a DNA template, which contains the region of the DNA fragment to be amplified
* two primers, which determine the beginning and end of the region to be amplified
* a DNA polymerase enzyme, which copies the region to be amplified
* Deoxynucleotides-triphosphate, from which the DNA Polymerase builds the new DNA
* a buffer, which provides a suitable chemical environment for the DNA Polymerase.

Each PCR cycle contain 3 steps namely template denaturing, primer annealing and elongation.

* denaturing: the double-stranded DNA has to be heated to 94-96°C in order to form single strands
* annealing: after separating the DNA strands, the temperature is lowered to 55-65°C so the primers can attach themselves to the single DNA strands
* elongation: new DNA strands are formed by the activity of DNA polymerase

This process is repeated and with each cycle, there is an exponential increase in the quantity of DNA such that after 'n' cycles the amount of DNA increases by 2^n. After amplification the products are size-fractionated by electrophoresis on an agarose gel stained with Ethidium Bromide. Products appear as a single band corresponding to the size of the amplified sequence, which exhibits fluorescence when illuminated with UV light (Fig 2).

2) Reverse transcriptase PCR (RT-PCR)

RNA is used as the template when information is needed on gene expression or to detect RNA viruses. However, RNA is very unstable and has a short half life and techniques such as Northern blotting which uses RNA as its template are is very difficult to perform. These difficulties can be overcome by using RT-PCR. In this technique, DNA that has been synthesized from messenger-RNA (m-RNA) by reverse transcriptase enzyme, known as c-DNA is used as the template as c-DNA is more stable than RNA. Thereafter, c-DNA template can be used to produce multiple copies of a specific DNA segment.[6,7]

3) Hybridization methods

Pairing of complementary RNA or DNA strands

molecule is referred to as hybridization. Southern blotting refers to the process in which DNA is analyzed while Northern and Western blottings refer to analysis of RNA and proteins respectively. As the principles behind all three processes are the same, Southern blotting is taken as the example to describe the hybridization techniques.

Southern blotting

Initially DNA is cleaved into several pieces using restriction endonucleases. The resultant DNA fragments are size separated by agarose gel electrophoresis. After separation, the DNA is transferred to a nylon or nitrocellulose membrane. Finally, separate DNA fragments can be identified by hybridizing the membrane with labeled complementary DNA probe[8] (Fig 3).

***In situ* Hybridization**

Both hybridization techniques and conventional PCR techniques the isolation of DNA/RNA from the cells as the first step. However, in *In situ* hybridization technique, specific DNA/RNA sequences can be identified within the cell, without causing destruction to the cell morphology. The principle is based on using a complementary probe directed against specific DNA/RNA sequence.(A probe is a specific sequence of nucleotide bases which is complementary to the DNA or RNA of interest). The probe is labeled enabling it to be detected by using an antibody directed against the label. This complex is then visualized by means of a fluorochrome or peroxidase reaction of a substrate[9] (Fig 4).

4) Micro-arrays

Micro-arrays are the most recent developments in the molecular biology field and have greatly facilitated the quantitative study of m-RNA. It can be used to analyze differential gene expression profiles between diseases and healthy tissues. Micro-array is based on hybridization techniques. The technique uses probes which are c-DNA fragments labeled with florescent markers. c-DNA produced from the test sample is spotted on to the DNA chips containing labeled probes. Probes and spotted DNA hybridizes revealing varying red/green and yellow fluorescent emission corresponding to the gene expression levels of a specific gene within the tested sample.[10]

Advantages and disadvantages of each method[11,12] mentioned above are summarized in Table 1.

Applications

1. Microbiology

PCR/RT-PCR have been used to identify periodontal pathogens, species associated with dental caries and to characterize micro-flora associated with endodontic infections. It can also be used to study microorganisms associated with malignancies.[12, 13] An advantage of PCR over traditional detection methods of micro-organisms such as culture, protein analysis or direct microscopy is that DNA or RNA of the infectious organism can be detected in test material even when the organism number is very low. In addition, archival material can be used to detect organisms when PCR is used.[14] In situ hybridization has been used to detect Epstein Barr viral particles (EBV) in oral hairy leukoplakia, a lesion seen in individuals infected with Human immunodeficiency virus (HIV).[12]

Human papilloma virus (HPV), Epstein Barr virus and Hepatitis-C virus have been identified in oral squamous cell carcinoma specimens. High risk HPV types 16, 18, 31 in combination with existing chemical carcinogens have been shown to contribute to the development of some oral squamous cell carcinomas by inducing genetic instability. However, though detected in some tumour samples, Hepatitis C and EBV viruses have not been implicated in the pathogenesis of oral squamous cell carcinoma.[15] Molecular techniques such as RT-PCR have been used to detect Hepatitis C virus in Oral Lichen planus

samples. As such, though not well proven, Hepatitis C virus has been implicated in the aetiology of Oral Lichen planus.

2. Human Genetics

Oral disorders that have a genetic basis can be basically divided into two categories, namely simple and complex conditions. Simple conditions are those resulting from mutations in a single gene that has a major effect, while complex conditions result from a collection of altered genes interacting with environmental influences. Major oral disease burden result from complex diseases that involve infectious microbial aspects coupled with hereditary and environmental risk factors.[1,2] Molecular biological techniques have played an important role in mapping and sequencing of the human genome which facilitated the identification of numerous specific genetic changes (mutations) responsible for simple Mendelian genetic conditions. With reference to Dentistry, these advances have resulted in identification of some mutations present in simple hereditary conditions such as hypodontia, amelogenesis imperfecta and dentinogenesis imperfecta. However, not all the mutations causing different types of amelogenesis imperfecta and dentinogenesis imperfecta are known to date. Therefore, further research is necessary to complete our understanding of these disorders.[16]

A genetic test is a test based on analysis of DNA, RNA, chromosomes or proteins in order to detect heritable disease related genotypes, mutations, phenotype or karyotype for clinical purposes. This type of test can be undertaken to predict risk of disease, identify carriers, and to determine the prognosis and response to therapy. It will be an important component of health care in the future. Therefore, dental surgeons should have the knowledge to guide their patients with reference to genetic tests in at least the genetic conditions with dental importance such as Papillon-Lefevre syndrome, Trico-dento-osseous syndrome, Amelogenesis imperfecta and Dentinogenesis imperfecta.

Congenitally missing teeth.

Hypodontia is the most common simple hereditary trait affecting the oral cavity. Missense mutations in MSX I gene, with an autosomal dominant pattern of inheritance has been shown to cause missing lateral incisors, second premolars and third molars. In addition, mutations in PAX 9 gene with an autosomal dominant pattern of inheritance have been shown to cause congenitally absent mandibular incisors, premolars and molars. However, as the prevalence of hypodontia has been shown to be different between races and also depend on the specific tooth that is missing more mutations may be discovered in the future. In addition, individuals with missing teeth can receive genetic testing to determine if molecular basis of their condition is known to establish the mode of inheritance and recurrences in offspring.[17,18]

Amelogenesis imperfecta

Amelogenesis imperfecta represents a group of hereditary conditions that manifest enamel defects without evidence of generalized or systemic disorders. Fourteen clinically and genetically distinct subtypes have been recognized to date. Autosomal dominant, recessive as well as X-linked inheritance patterns have been reported. However, molecular defects remain unknown for many forms of amelogenesis imperfecta at present. Twelve different mutations have been identified in AMEL X genes that code for amelogenin the most abundant enamel matrix protein. However, phenotypes resulting from these mutations are diverse ranging from enamel hypoplasia to hypomineralization. Understanding the genotype/phenotype relationship of different

amelogenesis imperfecta conditions will allow clinicians to accurately diagnose different types of amelogenesis imperfecta, predict prognosis and select the best treatment modality depending on the tissue defect.[19,20]

Dentinogenesis imperfecta

Dentinogenesis imperfecta (DI) is subdivided based on its association with osteogenesis imperfecta or being associated with Brandywine isolate. DI has been attributed to mutations in any one of the following three genes namely, Collagen 1A1(chromosome 17), collagen 1A2 (chromosome 7), dentinosialoprotein (chromosome 4). The molecular defects in Osteogenesis imperfecta include mutations in pro alpha chain of collagen type I. DI types II has been associated with silaophosphoprotein gene. Molecular differences seen in DI type I and II can be used to differentiate families that may have mild form of Osteogenesis imperfecta with DI from those with DI type II.[21,22,23]

Dental caries

Dental caries is the commonest dental disease. Although, dental caries is an infectious disease, there are numerous host resistance and risk factors that are genetically determined. Hereditary factors that contribute to many risk factors include pit and fissure morphology, enamel structure, tooth eruption time, salivary flow and composition, arch form, spacing immunologic function and dietary preference. Identification of the genetic factors that contribute to caries risk and resistance will provide clinician with new tools for targeting individuals for more efficient and effective preventive therapies. Numerous research papers exist on identification of pathogens involved in dental caries. Igarashi *et al,*(1996)[24] reported three identification methods namely dex A hybridization analysis, PCR analysis and electrophoretic profile of dextranase. Out of these methods, PCR method could potentially replace conventional identification methods such as biochemical and immunological tests for identifying mutans streptococci species. A study by Saarela *et al*, (1996)[25] using arbitrarily primed PCR (AP-PCR) indicated that the technique is suitable for epidemiological studies on mutans streptococci. In a study by Rupf *et al*,(1999)[26] a competitive PCR method determined the amounts of *S. mutans* in a sample and it can be used as a tool for evaluating the caries risk in patients and to determine the effectiveness of preventive and therapeutic measures. A another study by Igarashi *et al*,(2000)[27] using a specific primer for *S. sobribnus* indicated a relatively higher prevalence of *S. sorbinus* (83%) compared to previous studies. These results suggested that the described PCR method to be suitable for specific detection of cariogenic bacteria namely *S. sorbinus* and *S. mutans*. In 2002 Richard *et al,*[28] used specific PCR primers and DNA probes to differentiate *L. rhamnosm* from other *lactobacilli* species. These techniques may become useful to identify this species and to localize it, thereby helping to determine the progression of the carious process. Therefore, PCR and RT-PCR has been used to study epidemiology and to identify the cariogenic strains of bacteria.[1,2,13,24]

Periodontal disease

Periodontal diseases are a group of diseases characterized by inflammation and destruction of periodontium. While the importance of microbial aetiology is well known and specific microbes appear to be associated with certain forms of periodontitis, in most cases micro-organisms alone are not sufficient to cause the disease. Significant data now supports an important role for hereditary susceptibility in a variety of types of periodontitis. Microbial aetiology in chronic periodontitis is well established. Species more commonly found in subjects with chronic periodontitis than in healthy subjects include *T. denticola, Eubacterium saphenum, Porphyprymonas Gingivalis, T.forsythensis, Fillifactor alocis, Prevotella denticola, Cryptobacterium curtum,*

Treponema medium, T. socranskii and Actinomycoses nalsundi. Therefore, PCR has contributed to the knowledge of periodontal pathogenesis by enabling the diagnosis of pathogens. In addition, it also contributes to the identification of new pathogens. As the amount of microorganisms in plaque can be low, traditional culture methods may give false negative results and PCR has an advantage over culture as it has the ability to detect even small amounts of microbial DNA. Takeuchi *et al,* in (2001)[29] used PCR to identify, *T. denticola* and *P. gingivalis*. Their results indicated that these pathogens caused an inçrease in severity of periodontal tissue destruction. The involvement of viruses in periodontal disease has also been studied. In 1996, Parra and Slots[30] determined the prevalence of Human cytomegalovirus virus (HCMV), Epstein Barr virus, Herpes Simplex virus, Human papilloma virus and Human immuno deficiency virus in crevicular fluid of individuals with various forms of periodontal disease. Their results revealed the presence of viruses in many advanced periodontal lesions. Saygun *et al,* in (2002)[31] confirmed the presence of HCMV and EBV in periodontal lesions and showed a strong relationship between presence of HMCV and EBV with the attachment loss. In addition to microbial factors, hereditary factors are also considered to play an important role in the etiology of periodontal disease. Although, major genes and the total number of genes involved in chronic periodontitis are unknown, several gene products that may modify the clinical expression of periodontitis such as interleukin 4, polymorphous FC gamma receptor gene is known. Rather than mutations, naturally occurring genetic variations (genetic polymorphisms) are likely to be involved in the aetiology chronic periodontitis. The role of chemokines in periodontal disease has been studied by Garlet *et al,* in (2003).[7] Their results revealed a higher expression of protein 1-alpha and interferon gamma inducible protein 10. Receptors CCR5 and CXCR3 were seen in aggressive peridontitis. Monocyte chemo-attractant protein-1 and its receptor CCR4, IL-10 were seen in chronic periodontitis. In contrast, in aggressive periodontitis 1L-10 expression was low compared to chronic periodontitis. Results of studies of periodontitis in human twins suggest approximately 50% of susceptibility may be attributable to genetic factors. Therefore, chronic periodontitis is thought to result from additive effect of multiple genes, which may contribute to disease susceptibility through interactive effects with other gene products and through modulation of environmental factors such as smoking. In contrast to chronic periodontitis, a direct cause effect relationship for a specific gene defect has been identified in several syndrome forms of periodontitis. Mutation in Cathepsin C gene is responsible for Papillon-Lefevre Syndrome. CHS gene mutation cause Chediak-Higashi syndrome. Identification of these gene mutations demonstrates how different gene defects, influence disease susceptibility. Identification of such gene mutations permit genetic testing and are important to arrive at a definitive diagnosis of these conditions.[1,2,13]

3) Tumour biology

PCR has revolutionized the study of cancer and has provided greater insights into pathobiology of neoplasia. PCR has been used to detect mutations in cancer associated oncogenes such as ras, tumour suppressor genes p53, p16, monoclonality in B and T cell lymphomas, chromosomal translocations such as Philadelphia chromosome t(14:18) in chronic myelogenic leukaemia. PCR has also been used as a screening technique for detection of malignant cells in human secretions. As PCR is suitable to study low numbers of unique DNA fragments, it has been applied to the detection of malignant cells in urine, sputum and saliva.[11,12,13]

Oral cancer

Head and neck squamous cell (HNSCC) carcinomas are characterized by genetic alterations in somatic cells such as loss of heterozygosity (LOH), mutations, over-

rearrangements. A number of environmental factors such as tobacco, alcohol, HPV are important in HNSCC risk assessment. While these agents have been clearly associated with the development of HNSCC, not everyone exposed to these agents develop cancer and as such genetic factors appear to modulate cancer risk. Telomerase activity is associated with most malignant human tumours, but is not detected in normal somatic cells. A study by Lee *et al*, (2001)[32] revealed that the detection of H-TERT expression to be a useful diagnostic marker for early detection of OSCC and to distinguish healthy tissue from neoplastically transformed tissue.

Reis *et al*, in (2002)[33] indicated a worse prognosis and poor survival in patients with relative copy number loss of marker D225274 involving DIAI gene mapped on 22q13. As in chronic periodontitis, HNSCC risk is not determined by the presence of a single gene mutation, but rather the cumulative effect of multiple different gene polymorphisms. The presence of specific alcohol dehydrogenase gene polymorphisms have been determined to confer differential susceptibility to the effect of alcohol risk in certain populations. Genetic polymorphism of several enzymes including cytochrome P450 1A1 (CYPIAI), glutathion–S- transferase gene (GSTMI and GSTTI) and UDP–glucuronosyl-transferase 1A7(UGTIA7) genes may confer increased risk for tobacco related HNSCC.[34] Mutation in p53 gene has been reported in HNSCC. This gene may be affected by HPV which appear to have a casual association with a subset of HNSCC.[35] Vitamin E may help to inhibit the cancer formation by stimulating the expression of cancer suppressor gene p53. Altered expression of several matrix metalloproteinase and their inhibitors may be important determinants of the invasiveness and ability to metastasize in HNSCC. Cyclin D1 is a cell cycle regulatory factor that modulates a critical step in cell cycle control. CCNDI is over expressed in a proportion of HNSCC and has been correlated with aggressiveness, early recurrences and poor prognosis. Over expression of CCNDI has also been correlated with radio-sensitivity and may be a useful predictor of the effectiveness of radiotherapy on HNSCC. Antisense cyclin D1 may be useful in combination therapy with cisplatin in treatment of HNSCC.[36]

In the future, genetic characterization of OSCC can be combined with histological staging to develop classification of better clinical utility. Identification of genetic alterations that occur in specific forms of HNSCC may provide specific and sensitive diagnostic tests and such tests may be useful to identify recurrences before lesions become histopathologically visible.

Although changes that occur in cancer are complex, techniques such as micro arrays can be used to detect expression profiles of many genes at different stages of malignancy.[37]

Odontogenic tumours

Odontogenic tumours are lesions derived from the remnants of tooth forming apparatus and are found exlusively in jaw bones. A series of genetic and molecular alterations appear to promote development and progression of tumours via multiple steps. Although, the aetiology of odontogenic tumours remains unknown, recent studies have identified various molecular alterations responsible for development and progression. Oncogenes such as Ras, Myc, Fos; tumour suppressor genes p53 and APC, oncoviruses HPV and EBV, telomerase, cell cycle regulators such as Cyclin D, regulators of tooth development such as amelogenin, enamelin, ameloblastin and hard tissue related proteins such as osteonectin, BSP, osteocalcin, osteopontin and BMP are molecules possibly associated with tumorigenesis and or tumour cell differentiation. Molecules possibly associated with tumour progression are cell adhesion molecules, E-selectin, E-cadherin, integrins, matrix degrading proteins, MMP-1,2and 9, angiogenic factors VEGF and osteolytic cytokines, IL-1, TNF. However, further molecular studies such as genomic and protein based profiling are required

to clarify the aetiology and pathogenesis of odontogenic tumours. In addition, these molecular mechanisms may find applications such as prediction of course of disease, patient tailored therapy in the future.[38]

Conclusion

Knowledge derived from the study of proteomics and gene expression will further advance the diagnosis and treatment of cranio-facial pathology in the oncoming years. Therefore, the future dental surgeons will require a basic working knowledge of molecular biology and genetics to diagnose, refer for further evaluation and apply molecular based treatment modalities to their patients. As such, this review was written to summarize the molecular based techniques and their current applications in dentistry. In addition, possible future applications are also summarized.

Table 1. Advantages and disadvantages of molecular biological techniques

Method	Advantages	Disadvantages
PCR	* Sensitive/ specific * Can perform with very small amount of DNA (pico gram quantities) * Partially degraded DNA can be used as the template	* False negative/ false positive results due to contamination * Technically demanding * Expensive
Reverse transcription PCR	* Sensitive/ specific * Amount of RNA required is less compared to Northern blot * Less technically demanding than Northern blot	* False positive * Expensive * Time consuming * Technically demanding
Southern Blot	* DNA size information * Quantification of gene copy number possible	* Time consuming * High quality DNA is needed * Requires hybridization
Micro-array	* More than one gene can be analyzed at a time * Gene expression levels in health and disease can be analyzed	* Very expensive * High quality RNA/ DNA is required * can only analyze previously characterized genes that are available on the DNA chip and not novel genes

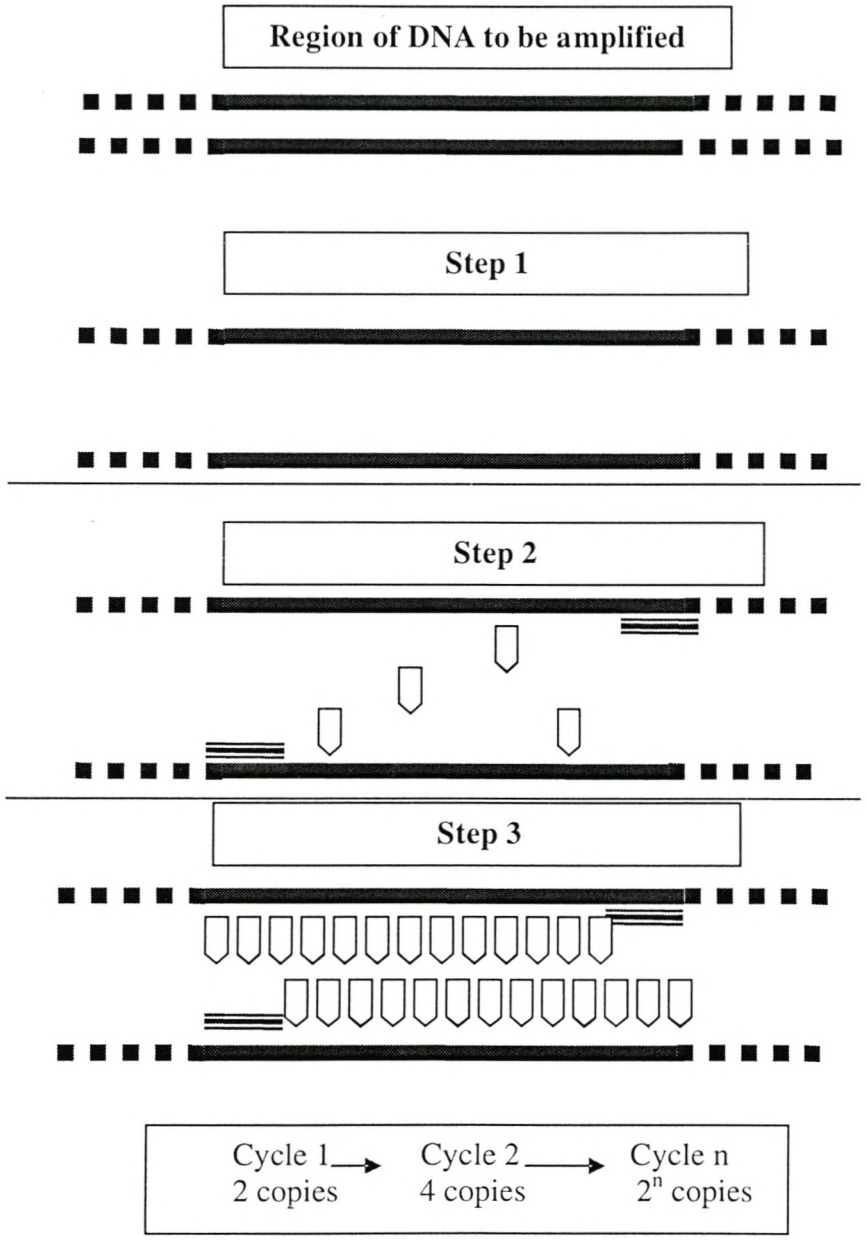

Figure 1. Schematic diagram of PCR to show the three steps in each cycle. Step 1: Denaturing, Step 2: Primer annealing, Step 3: Elongation

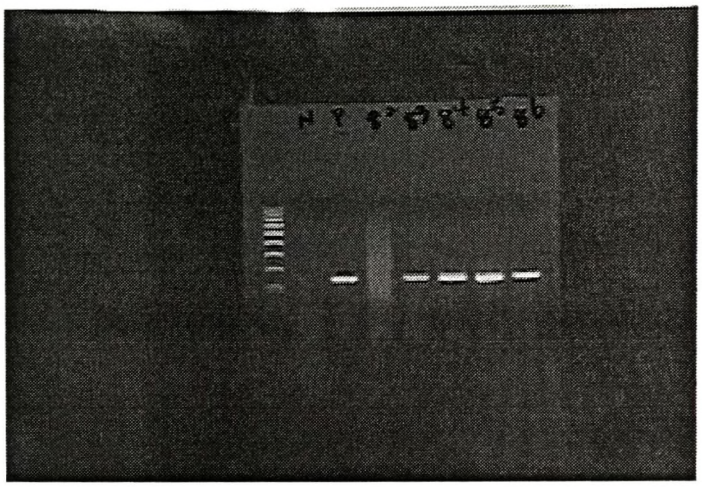

Figure 2. Ethidium bromide stained agarose gel showing results of PCR amplification. A single DNA band of the predicted size (150bp) was obtained using consensus primers of the target organism, Human papilloma virus. DNA was extracted from formalin fixed paraffin embedded oral squamous cell carcinoma tissue samples.

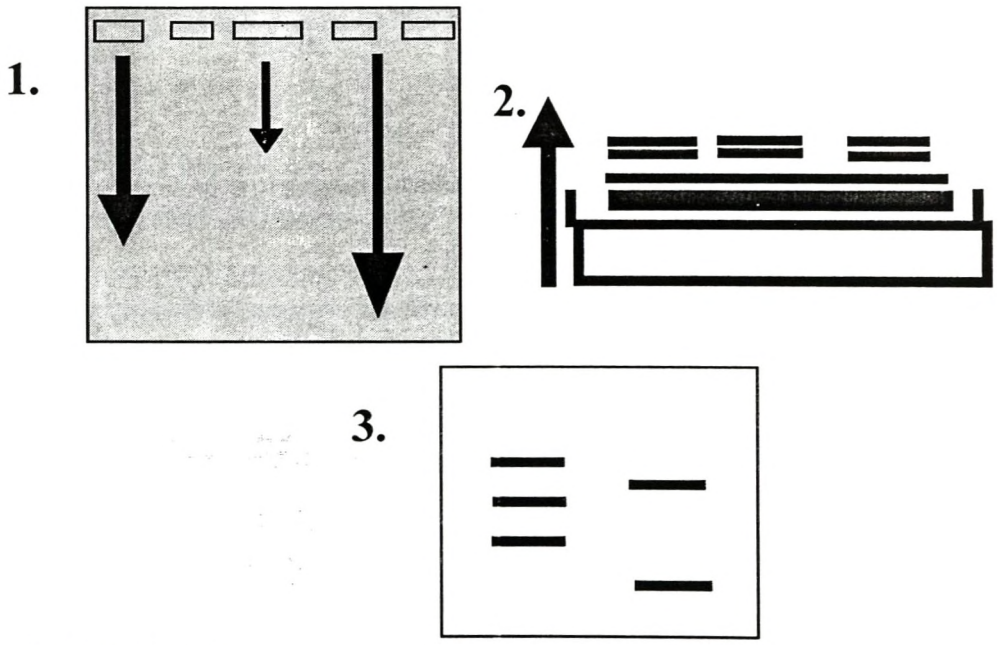

Figure 3. Southern blotting.
Step 1: Size separation by gel electrophoresis, DNA or RNA that has been previously cut by restriction endonucleases.
Step 2: The size fractioned DNA or RNA is transferred on to a nylon or nitrocellulose membrane from the gel.
Step 3: Following immobilization of DNA/ RNA fragments on the membrane, it is hybridized with labeled DNA or RNA probe to demonstrate the specific bands of genetic material that are present.

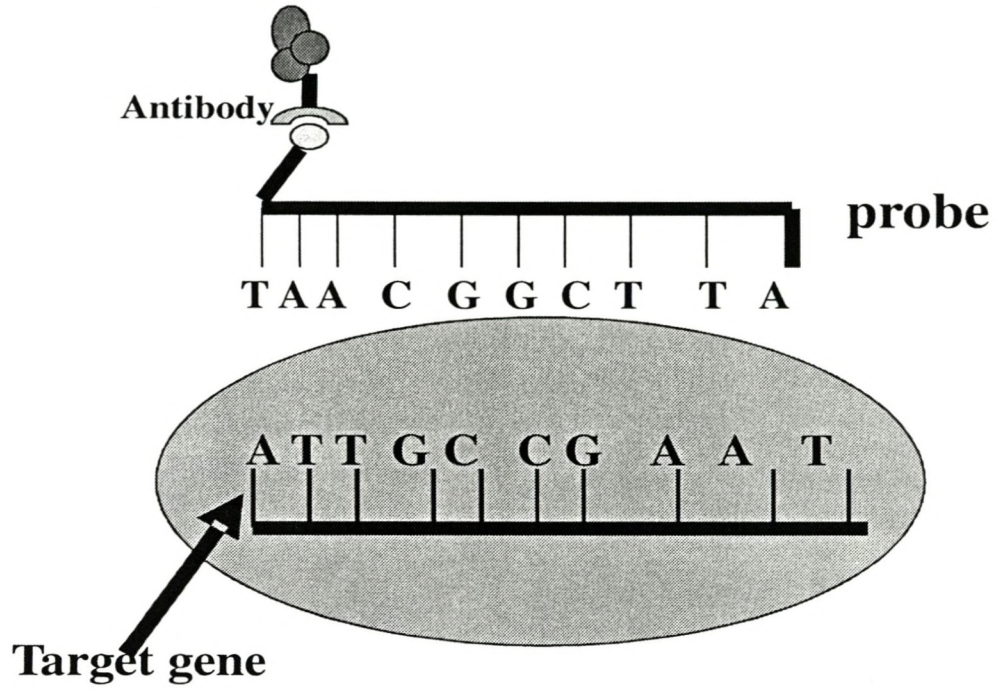

Figure 4. *In situ* hybridization. DNA/ RNA in a cell is identified in situ by using complementary probe of RNA or DNA.

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Research Article 1

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Use of electromyography in predicting muscle pain related to Temporomandibular Disorders

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Abstract

Objective: The aim of the present study was to identify the electromyographic (EMG) parameters that best predict the severity of muscle pain in muscle-related temporomandibular disorder (TMD) patients.

Material and methods: Thirty-eight muscle-related TMD patients (16 males and 22 females; mean age 29±10.3 years) participated in the study. The EMG activities of masseter and temporalis muscles of both sides were recorded at the rest position of the jaw, maximum opening of the mouth and maximum voluntary clenching, using a portable EMG machine. At the maximum voluntary clenching, the bite force was controlled using a custom made Bite Force Transducer. Severity of muscle pain perceived by patients was assessed using a 100 mm Visual Analogue Scale.

Results: EMG activities recorded at rest were the only significant predictors of the severity of pain expressed over the masseter (R^2=0.13, F=5.48, P=0.025) and temporalis (R^2=0.23, F=10.88, P=0.002) muscles. EMG activities recorded at maximum voluntary clenching and maximum mouth opening did not show any significant association to the severity of pain (P>0.05).

Conclusion: The present findings suggest that only the EMG activity recorded at rest position of the jaw would be useful in predicting and objectively measuring muscle pain in TMD patients in research and clinical practice. It may also be used to substantiate the subjective measurements of muscle pain.

Key words: Temporomandibular disorders, masticatory muscle pain, electromyography, myogenous, masticatory muscles, pain, bite force

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Introduction

Electromyography (EMG) has been a popular tool in objectively measuring muscle function in many investigations since it was first introduced to dental research by Moyer in 1949.[1] Among the two basic techniques of EMG, fine-wire (needle) EMG has limitations to the practical application owing to the cost and level of specialisation required. In contrast, surface EMG became the popular mode as it was non-invasive and convenient. Since then, multitudes of EMG parameters have been used as objective measurements of muscle activity mainly in relation to muscle dysfunctions including temporomandibular disorders (TMD)[2-5] Originally it was hypothesized that the hyperactivity of masticatory muscles is the major cause of TMD and hence, investigators assumed that the EMG activity would be invariably high in TMD patients. However, findings have been inconsistent,[6-7] and use of unreliable and invalid EMG parameters have been held partly responsible.

Muscle hyperactivity is broadly defined as abnormally prolonged and/or elevated levels of muscle activity beyond the normal requirement for function and postural maintenance.[8] Such alterations in postural muscle activity (postural tone) is believed to be brought about by many factors such as life stress,[9] oral para-functions for example day-time clenching and nocturnal bruxism,[10-13] and micro and macro trauma[14] etc., probably initiating or precipitating muscle related TMD.

However, with the understanding that the TMD is a multifactorial and heterogenous group of disorders, investigators observed marked differences in muscle activities with regard to various sub-groups of TMD. Basically, changes in EMG activities of masticatory muscles have less frequently been observed in joint-related TMD than in muscle-related TMD.[3,15-17] Therefore, experimental models and methodology required to be developed considering the diagnostic heterogeneity excluding inappropriate sub-groups of TMD in study samples.

Changes in electrical activity of masticatory muscles in patients with muscle-related TMD were later attributed to the presence of pain and were thought to change with the intensity of pain.[8] Particularly, the muscle hyperactivity observed at the rest position of the jaw has been a very consistent observation, which has been partly attributed to the presence of pain.[3,4,18] The higher EMG activities of masticatory muscles observed in TMD patients during mouth opening have been ascribed to a reflex mechanism known as protective co-contraction (protective muscle-splinting). Moreover, the EMG activities recorded during clenching have been observed to be lower as a result of dysfunction or frail function of muscles in the presence of pain.[4,19] Thus, it was clear that in this heterogeneous group of disorders, the muscle activities vary widely depending on the involvement of muscles, presence of pain, and jaw positions at which the EMG is recorded.[3,4,18]

Therefore, the objective of the present study was to ascertain the EMG parameter/s that best predict/s the existing intensity of muscle pain in muscle-related TMD patients. The null-hypothesis tested was that the severity of TMD related muscle pain measured in terms of a Visual Analogue Scale (VAS) would not be significantly predicted by EMG activities of masseter and temporalis muscles recorded at rest, maximum mouth opening and maximum clenching.

Material and methods

Thirty-eight muscle-related TMD patients (16 males and 22 females; mean age 29 years, SD = 10.3 years) were recruited from patients who sought care at the Oral Medicine Clinic of the Dental Hospital of the University of Peradeniya, Sri Lanka. Two examiners screened and selected the patients in agreement as a convenient consecutive sample, according to the criteria established by American Academy of Orofacial

Pain.[20] Duration of TMD related pain and dysfunction reported by patients varied between two months to one year. Exclusion criteria were presence of disc-interference disorders without pain in masseter and/or temporalis muscles, presence of a medical history of established psychological disorders or treatment for psychological conditions, presence of trauma associated with head injury, or neurological disorders. Patients with degenerative joints were excluded following confirmatory radiographic investigations. Patients with myositis, myospasm and contractures did not report to the clinic during this period.

Procedure

The study protocol was approved by the Research and Ethical Committee of the Faculty of Dental Sciences, University of Peradeniya. The nature and the objectives of the experiment were explained to the selected subjects before obtaining informed consent. Following an initial screening, the patients were examined using a specially designed index protocol adopted from the one used by Liu and others.[19] This standardized the clinical examination and further, the final selection of patients was based on these data. Pain intensities reported over the muscles were evaluated using a Visual Analogue Scale (VAS) as described below. EMG activities of left and right masseter and temporalis muscles were recorded using a portable EMG scanner. A single examiner performed these procedures for all subjects following a written operational manual.

EMG recording

A battery operated, portable EMG machine (MS-100 EMG scanner, Myotronics Inc., Seatle, WA) was used for the purpose of recording surface EMG activities. The post-style, Ag-AgCl reusable electrodes (diameter 10 mm) mounted on the EMG scanner were employed. Distances between the two recording electrodes and between a recording electrode and the reference electrode were 10mm and 5mm respectively. These inter-electrode distances were constant for all patients as the electrodes were mounted on the machine. This EMG scanner records only the integrated EMG activity, and the reading is presented on a liquid crystal display, which could be frozen and recorded at the press of a button. The accuracy of MS-100 EMG scanner was tested against a conventional EMG machine. A high Intra-class Correlation Coefficient (γ =0.9, $P<0.001$) was observed between the two sets of readings obtained with the two machines.

Measures were taken and instructions were given to keep the subjects in a relaxed state. During EMG recording, each subject was comfortably seated in a chair without supporting the head and with both hands resting on the lap. Skin surface was thoroughly cleaned with 95% alcohol and a special conductivity gel (Myo-gel, Myotronics Inc.,) was applied on the recording surface of the electrodes to minimize the skin impedance. The electrodes were placed parallel to the direction of muscle fibres and perpendicular to the skin surface following the guidelines for placing electrodes.[21] The reading at the eighth second was always recorded as specified by Myotronics Inc. In every subject, the EMG activities were recorded in the order of right masseter, left masseter, left temporalis and right temporalis.

The EMG activities were recorded at three jaw positions: at rest (EMG-RP), at maximum opening of the mouth (EMG-MO) and at maximum voluntary clenching (EMG-MVC). Bite force at the MVC (BF-MVC) was controlled and recorded using a custom made Bite Force Transducer (BFT). The BFT was produced by using a plate of stainless steel that can withstand the bite force of humans within the elastic limit of the material and a strain gauge to measure the tension. The thickness (height) of the bite piece of the BFT was maintained at 14 mm based on previous research.[22-23] The resistance produced in the strain gauge of BFT during biting was converted to a voltage and amplified ten times

using a bridge circuit and an amplifying circuit. This was connected to a digital multimeter through which the visual feedback was established. Reliability and reproducibility of this BFT were tested in separate preliminary experiments and observed to be very satisfactory.

Assessment of muscle pain

A 100 mm horizontal line was used as a VAS for rating pain intensities. The anchor words, "no pain at all", and "the worst imaginable pain" were written at both ends of the line and were verbally explained to all the patients.[24] Patients were asked to draw a mark on the line at the point that corresponds best to the degree of pain they perceived at the time of palpation. The distance between the starting point and the mark was measured in millimeters and recorded as the intensity of pain. The palpation was performed by the examiner according to the method described by Langemark and Olesen. [25] The pain over right and left masseter and temporalis muscles were evaluated, and when the pain was present on a particular muscle of both sides, the highest rating for that muscle was always recorded.

Data Analysis

As this was a part of a large study that used a comprehensive index protocol for clinical examination and data collection, only the data related to the present objective are analyzed and presented. In the presence of unilateral pain EMG activity of the muscle of the respective side was used, and when bilateral, the EMG activity of the muscle of the side that reported the highest pain was used for the analysis. Data were examined using explorative statistical procedures and the ability to use parametric tests was assessed with the Kolmogorov-Smirnov test before testing the null-hypothesis. EMG-RP and EMG-MO were used for the analysis without any transformation. For EMG-MVC, *EMG activity to BF ratio (EMG-MVC/BF-MVC)* was calculated for each patient. Pain intensities of masseter and temporalis muscles were used as outcome variables and EMG measurements were employed as predictor variables. The associations of EMG parameters to pain intensities were explored using two independent stepwise multiple regression analyses in relation to the two muscles. Statistical significance was accepted at a level of 0.05. The data were analyzed using the Statistical Package for Social Sciences (SPSS) for Windows version 11 (SPSS Inc., Chicago, IL, USA).

Result

Mean intensities of pain and EMG activities recorded over masseter and temporalis muscles are illustrated in Table 1. Multiple linear regression analyses revealed that only the EMG activities recorded at rest position of the jaw (EMG-RP) were included in models with respect to masseter and temporalis muscles, and the EMG-MO and EMG-MVC/BF-MVC were excluded (Table 2 and Table 3).

Discussion

The results of the present study indicate that the integrated EMG activity at rest position is the only EMG parameter that can satisfactorily reflect the pain intensities of both masseter and temporalis muscles. EMG activities at maximum opening of the mouth and maximum voluntary clenching did not show any association with the degree of existing pain in respective muscles.

The observation that the resting EMG activity of masticatory muscles is proportionately associated with the severity of existing muscle pain has consistently been corroborated by many previous investigators.[8,26] Numerous studies support the fact that resting EMG activity goes down when pain is relieved by successful treatment of the condition.[27] This observed muscle hyperactivity at rest may also be an outcome of any existing pain. However, Liu and colleagues[4] have failed to observe any association between resting EMG activities and the severity of muscle pain. Having

a five point scale to grade the severity of pain may have masked the available inter-group variation and the presence of a mixed group of patients as the sample may have attenuated the effect.

Although it has been shown that the integrated EMG activities tend to be less in maximum voluntary clenching[4] and higher in maximum mouth opening in TMD patients than in healthy individuals,[4,19] these parameters were not effective in predicting the severity of existing muscle pain. Perhaps such changes either do not proportionately fluctuate with the changing levels of pain or have a higher variability (Table 1). Hence, caution is necessary when using such parameters as objective measurements of muscle pain or dysfunction, except in occasions where the investigator intends to study muscle activity irrespective of the inherent degree of pain.

The present research design nullifies the effects of other biological variables such as age, sex, weight, skeletal patterns and psychological status, which may possibly act as confounding factors in a case control design.[28] However, exclusion of patients with disc-interference disorders without pain in masticatory muscles can be considered a limitation of the present study. Although, it has been observed that EMG changes are less frequent in this group of patients, theoretically, it is possible that mechanisms such as protective co-contractions may operate so that EMG activity at maximum mouth opening could be affected.

Subjected to aforementioned limitation, the results of the present study suggest that the pain intensities of masticatory muscles in muscle-related TMD patients can reliably be predicted only by the resting EMG activities. Further studies including all sub-groups of TMD are recommended to confirm these results, so that the clinicians and researchers could use resting EMG activities of masticatory muscles as an objective measurement of muscle pain. It is also suggested that a multi-channel EMG machine be used for such studies so that the above findings can be validated and confirmed.

Table 1. Pain intensities and EMG activities recorded in relation to each muscle

	MasseterMean ± SD	Temporalis Mean ± SD
Pain intensity	39.70 ± 18.67	33.91 ± 19.11
EMG-RP (μV)	5.43 ± 1.74	4.97 ± 1.45
EMG-MO (μV)	7.44 ± 2.97	6.11 ± 2.22
EMG-MVC/BF-MVC (μV)	5.98 ± 2.67	5.52 ± 2.48

EMG-RP (EMG activity at rest position), EMG-MO (EMG activity at maximum opening of the mouth), EMG-MVC/BF-MVC (EMG activity at maximum voluntary clenching /Bite force at maximum voluntary clenching)

Table 2. Association of EMG parameters to muscle pain in masseter muscle

Variable	Coefficient (B)	Standard Error	95% confidence Interval	*P*
Variables included				
Constant	1.86	0.95		
EMG-RP	0.39	0.17	0.05 to 0.72	0.025
Variables excluded				
EMG-MO	-0.06			0.72
EMG-MVC/BF-MVC	0.04			0.81

Stepwise multiple linear regression analysis, Dependent Variable: VAS recording of masseter muscle. Predictors in the Model: (Constant), EMG-RP (EMG activity at rest position), EMG-MO (EMG activity at maximum opening of the mouth), EMG-MVC/BF-MVC (EMG activity at maximum voluntary clenching / Bite force at maximum voluntary clenching), Model: R^2=0.13, F=5.48, P=0.025

Table 3. Association of EMG parameters to muscle pain in temporalis muscle

Variable	Coefficient (B)	Standard Error	95% confidence Interval	*P*
Variables included				
Constant	0.24	0.99		
EMG-RP	0.63	0.19	0.24 to 1.02	0.002
Variables excluded				
EMG-MO	-0.19			0.19
EMG-MVC/BF-MVC	0.03			0.85

Stepwise multiple linear regression analysis, Dependent Variable: VAS recording of temporalis muscle. Predictors in the Model: (Constant), EMG-RP (EMG activity at rest position), EMG-MO (EMG activity at maximum opening of the mouth), EMG-MVC/BF-MVC (EMG activity at maximum voluntary clenching / Bite force at maximum voluntary clenching), Model: R^2=0.23, F=10.88, P=0.002

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Preliminary investigation revealed no evidence of hepatitis C virus infection in Sri Lankan oral lichen planus patients

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Abstract

Objective: The objective of this study was to determine the prevalence of hepatitis C in Sri Lankan patients with oral lichen planus.

Material and methods: Thirty-five consecutive patients referred to Oral Medicine clinic, Faculty of Dental Sciences, University of Peradeniya, Sri Lanka during the period from March to December 2004 and ten controls were enrolled for this study. Detection of anti HCV antibodies (HCV-Ab) was performed using rapid immunochromatographic assay kit (Advanced Quality ™ Rapid HCV Test).

Results: None of the patients or the controls was positive for the HCV-Ab.

Conclusion: Failure to detect HCV antibodies in any of the OLP subjects strongly suggests the lack of association between OLP and HCV infection in this group of Sri Lankan patients. However, further studies using a larger sample would be needed to confirm this finding.

Key words: oral lichen planus, hepatitis C virus infection, HCV antibodies, Sri Lanka

Introduction

Lichen planus (LP) is a well known muco-cutaneous condition of unknown aetiology.[1] It occurs in the skin, mucous membranes or both and shows a female predilection from forth to fifth decade of life.[2] Oral lichen planus (OLP) manifests in six different morphological forms namely reticular, papular, plaque like, atrophic, erosive and bullous forms (3). Generally, affected individuals with oral lichen planus may be asymptomatic or may complain of roughness, mild discomfort or pain.

Several reports in the recent past have proposed a possible association of lichen planus with hepatitis C virus (HCV) infection (Table 1). The prevalence of anti-Hepatitis C antibodies varies from 0-65% in patients with cutaneous LP and /or OLP or both.[4-39] The majority of studies were reported from southern European countries and Japan. However, the studies in the United Kingdom[20,22,24] Netherlands[23] USA[30] Nepal[31] and Serbia[38] have failed to substantiate such an association. No reports of such data are available from Sri Lanka

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to date. Therefore the objective of this study was to determine the prevalence of hepatitis C in Sri Lankan patients with oral lichen planus.

Patients and methods

Thirty five consecutive patients who were referred to the Oral Medicine Clinic, Faculty of Dental Sciences, University of Peradeniya, Sri Lanka from March to December 2004 were enrolled for this study. The diagnosis established was based on standard clinical and histological criteria. Patients with the possibility of lichenoid reactions were excluded from the study.

The study group consisted of 16 males and 19 females with a mean age 43.6 years (SD ±12.82). Ten age and sex matched healthy volunteers were used as controls for the preliminary study. All the patients were informed about the study and informed consent was obtained. Five milliliters of venous blood was drawn from all the patients and volunteers (for control) and immediately stored at -70^{0}C until the assay was performed.

Virological assay

Detection of anti HCV antibodies (HCV-Ab) was performed using rapid immunochromatographic assay kit (Advanced Quality™ Rapid HCV Test).

Results

Demographic and clinical data are given in the table 2. None of the patients or the controls was positive for the HCV-Ab.

Discussion

The present study was designed to ascertain the prevalence of HCV infection among a group of OLP patients in Sri Lanka. The general method of detecting infection with HCV is to observe the presence of antibodies to the virus by enzyme-linked immunosorbent assay (ELISA) followed by recombinant immunoblot assay (RIBA) or polymerase chain reaction to confirm the results.[12,17,18,23] However, in the present preliminary study we used the rapid HCV test as a screening method which is cheap and less time consuming.

Thirty six studies have been reported between 1992 and 2005 with reference to the association between lichen planus and hepatitis C (4 -39). A control group was used to compare the results in only 21 studies.[6,9,11-15,17,18,20-22,25-27,32-34,36,38,39] A significant positive association has been found in southern European countries[4, 7, 12, 18, 19, 24] and Japan.[10] In contrast, studies from UK,[20] Netherlands,[23] Serbia[38] and Nepal[31] revealed no such association between hepatitis C infection and lichen planus. Such a variability of the prevalence of hepatitis C virus among lichen planus patients led to the controversy regarding the cause and effect relationship.

It is estimated that nearly 170 million people are infected with HCV worldwide and the prevalence of carriers estimated to average 3% with a range from 0.1-10%, depending on the country affected.[40] HCV infection is relatively uncommon in Sri Lanka. In a study De Silva and colleagues[41] revealed 15% HCV positive cases among alcoholic cirrhosis patients. Fernando and colleagues[42] revealed 33% and 10% HCV positive cases among Hemophiliacs and Thalassemics respectively. However, no positive cases were found among the otherwise healthy adults.[43]

None of the oral lichen planus patients was positive for hepatitis C antibodies in the present study. As HCV infection is uncommon in Sri Lanka, lack of association between lichen planus and hepatitis C could be suggested in Sri Lankan patients. However, a further study is needed with a larger sample to confirm this finding. In conclusion, failure to detect HCV antibodies in any of the OLP subjects strongly suggests the lack of association between OLP and HCV infection in this group of Sri Lankan patients. However, further studies with larger samples would be needed to confirm the findings.

Table 1. Summary of reported studies: association between HCV infection and OLP (1992-1995)

Author	Country	Year	Number of cases OLP/CLP/Both	Cases (positive) n	(%)	Controls (Number) n	Controls (positive) n	(%)
Rebora et al., [4]	Italy	1992	50/0/0*	2	(4)	0	0	(0)
			29/0/0**	19	(65)	0	0	(0)
			46/0/0***	11	(24)	0	0	(0)
Divano et al., [5]	Italy	1992	0/46/0	15	(32.6)	0	0	(0)
Criber et al., [6]	France	1994	4/48/0	2	(3.8)	112	3	(2.6)
Bagan et al., [7]	Spain	1994	187/0/0	28	(15)	0	0	(0)
Gandolfo et al., [8]	Italy	1994	105/0/0	10	(9.5)	0	0	(0)
Tanei et al., [9]	Japan	1995	28/8/9	17	(37.8)	45	3	(6.7)
Nagao et al., [10]	Japan	1995	45/0/0	28	(62)	0	0	(0)
Bellman et al., [11]	USA	1995	0/30/0	7	(23)	41	2	(4.8)
Carrozzo et al., [12]	Italy	1996	70/0/0	19	(27.1)	70	3	(4.3)
Sanchez –Perez et al., [13]	Spain	1996	22/22/34	16	(20)	82	2	(2.4)
Chosidow et al., [14]	France	1997	102/0/0	5	(4.9)	306	14	(4.5)
Imhof et al., [15]	Germany	1997	22/62/0	13	(16)	87	14	(1.1)
Grote et al., [16]	Germany	1998	24/0/0	1	(4.2)	0	0	(0)
Bagan et al., [17]	Spain	1998	100/0/0	23	(23)	100	5	(5)
Mignogna et al., [18]	Italy	1998	263/0/0	76	(28.8)	100	3	(3)
Dupond et al., [19]	France	1998	28/0/0	8	(29)	0	0	(0)
Ingafou et al., [20]	UK	1998	55/0/0	0	(0)	100	0	(0)
Ilter et al., [21]	Turkey	1998	0/75/0	0	(0)	75	0	(0)
Tucker and Coulson [22]	UK	1999	13/32/00	0	(0)	32	1	(3.1)
Ven der Meij&van-derWaal[23]	Netherland	2000	55/0/0	0	(0)	0	0	(0)
Roy et al., [24]	Scotland	2000	6/0/0	0	(0)	0		(0)
Kirtak et al., [25]	Turkey	2000	27/46/0	5	(6.8)	73	1	(1.36)
Erket et al., [26]	Turkey	2001	0/54/0	7	(12.9)	54	2	(3.7)
Beaird et al., [27]	USA	2001	0/24/17	7	(17)	20	1	(5)
Chainani-Wu et al., [28]	USA	2001	31/0/0	14	(45)	0	0	(0)
Mignogna et al., [29]	Italy	2002	600/0/0	165	(27.5)	0	0	(0)
Eisen D [30]	USA	2002	195/0/0	0	(0)	0	0	(0)
Garg et al., [31]	Nepal	2002	14/35/15	0	(0)	0	0	(0)
Daramola et al., [32]	Nigeria	2002	0/57/0	9	(15.8)	24	0	(0)
Figueiredo et al., [33]	Brazil	2002	63/0/5	6	(8.8)	898	6	(0.6)
Campisi et al., [34]	Italy	2003	847/0/0	236	(27.9)	822	151	(18.4)
Klanrit et al., [35]	Thailand	2003	60/0/0	5	(8.3)			
Gimenez-Garcia and Perez-Castrillion [36]	Spain	2003	101(not specified)	9	(8.9)	99	2	(2.02)
Ghodsi et al., [37]	Iran	2004	146(not specified)	7	(4.8)	NA	NA	(0.1)
Bokcor-Bratic M [38]	Serbia	2004	48/0/0	0	0	66	0	(0)
Giuliani et al., [39]	Italy	2005	76/0/0	9	(11.4)	466	25	5.3

Table 2. Demographic and clinical characteristics of experimental sample and the controls

Characteristic	Cases (n=35)	Controls (n=10)
Age (years)	43.6 (12.8)	37.7 (13.7)
Male: Female ratio	16:19	4:6
Duration of symptoms (weeks)	25.1	-
Asymptomatic	8	-
Roughness	5	-
Burning sensation at meal times	22	-
Skin lesions		
No	32	-
Yes	2	-

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Crown lengthening and aesthetic recontouring of upper central incisors

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Introduction

Patient awareness and expectations of facial aesthetics have increased significantly in the recent past leading to a situation where less than optimal aesthetics is no longer acceptable.

Facial aesthetics depends on a number of factors where teeth, gingiva and lips play an important role. The importance of correct size, shape, number and position of teeth is frequently stressed.[1] Guidelines for anterior aesthetics have been proposed and documented.[2] For instance teeth should be well proportioned, with the central incisor having a width to height ratio of about 0.8.

When the width of the incisors is to be increased, increasing the height should always be performed whenever possible to maintain the shape of the tooth. Restorations can be done at the incisal edge to achieve this, only when occlusion and aesthetics permit. Increase in crown height can also be achieved by crown lengthening gingival surgery in selected patients.[3] Crown lengthening involves the surgical removal of soft and hard periodontal tissues to gain supracrestal tooth length, resulting in longer clinical crowns and reestablishment of biological width.[4] A high lip line, causing a gummy smile can also be improved with crown lengthening surgery.[5]

Proper assessment of the patient is mandatory for achieving successful results in crown lengthening surgery. Forced eruption of teeth by orthodontic treatment is indicated prior to crown lengthening surgery when the gingival margins of teeth are already at an ideal relationship with the adjacent teeth.[6] This will ensure that the final gingival margin remains at original position. Teeth with a minimal tapering in the coronal third of the root achieve better results. An excessive tapering in this region results in restorations with a poor emergence profile or unaesthetic black triangles following crown lengthening. If the width of the keratinized gingiva must be preserved or augmented, then the flap may be apically repositioned at or slightly apical to the alveolar crest. Coronal or apical migration of the gingival margin may occur during subsequent months. In cases with a thin periodontium, some apical migration may be expected. Younger patients show a greater tendency for coronal migration of the margin, post surgically than their adult counterparts.[7]

The apical extension of restorations should not interfere with the biological width. The concept of the biological width derives from the histological description of the dentogingival complex described by Garguilo *et al*,.[8] Accordingly the mean sulcus depth is considered to be 0.69 mm, epithelial

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attachment 0.97 mm and supracrestal connective tissue 1.07 mm. Therefore the total length of the dento-gingival complex is 2.73 mm. Hence it is recommended that 3 mm, of supracrestal tooth structure be obtained during surgical crown lengthening. Although the new junctional epithelium is formed in 2 weeks, the biological width at treated site is reestablished to its original vertical dimension by 6 months.[9]

Surgical crown lengthening is also indicated when restoring teeth with

- Root caries
- Subgingival fracture margins
- Dentinal hypersensitivity
- Inadequate crown height

The following disadvantages are associated with crown lengthening surgery;

- Treatment delay (especially for wound healing and orthodontic extrusion)
- Discomfort
- Considerable added cost
- Increased crown / root ratio
- Loss of tooth structure
- Risk of exposing furcations, root concavities and developmental grooves

Case report - 1

A 23 year old female who attended the Advanced Restorative Clinic, Department of Restorative Dentistry, Faculty of Dental Sciences, Peradeniya, Sri Lanka complained of discolored composite veneers on upper central incisors.

These teeth had fractured several years ago and root canal treatment had been done, followed by light cured composite (LCC) crown restorations. She had not experienced any symptoms following treatment. The patients medical history showed no significant diseases and her general appearance was healthy. She requested a treatment with short duration, to improve her aesthetics as she was to get married in a few weeks time.

The patient's oral mucosa appeared normal. She had the full complement of teeth. 11 and 21 had discolored unaesthetic LCC veneers (Fig. 1). They were proportionately too broad and had over hanging margins. There were 3 mm, deep periodontal pockets on the labial side of 11 and 21. The cemento enamel junction could be felt at a depth of 1.5 mm. The width of the attached gingiva was about 4 mm, in the upper incisor region. The gingival margins of 11 and 21 were at a lower level than normal when compared with 12 and 22. Gingivae in other areas and other teeth were healthy.

Endodontic treatments of 12 and 21 were radiographically acceptable. Both teeth also had adequate alveolar bone support. Considering the above clinical findings, the following treatment plan was suggested to the patient.

1. Apical repositioning of gingivae on 11 and 21.
2. Replacement of LCC veneers.

The treatment plan was accepted by the patient. Apical repositioning of gingiva by 3 mm was planned on a study model and an appointment was given for surgery.

During surgery a crevicular incision was made in relation to the labial side of 11, 12, 21 and 22 under local anesthesia. A mucoperiosteal flap was raised and the gingival flap was mobilzed by cutting the periostium horizontally. The sulcular epithelium was trimmed with the scalpel. Thereafter the flap was apically repositioned and sutured after contouring the marginal alveolar bone (Fig. 2 and 3). The patient was advised to refrain from raising the upper lip. Ibuprofen (200mg tds for 3 days) and chlorhexidine 0.2% mouthwash were prescribed.

Sutures were removed after one week (Fig. 4). The patient was requested to wait for one month for gingival stabilization. Light cured composite veneers were done on 11 and 21 using shades A1, A2, A3 and opaquer. A better contour was obtained by replacing the previous veneer (Fig.

5). The patient was extremely happy about the final aesthetics (Fig. 6). She was given a review appointment after six months.

Case report - 2

A 20 year old male who was referred to the Department of Restorative Dentistry complained of spacing between upper front teeth. He had lost his upper central incisors about 12 years ago following an accident and had been without any prosthesis since then.

The patient's medical history revealed no significant diseases and his general appearance was healthy. The oral health of the patient was satisfactory.

A 6 mm space was present between 12 and 22. Gingival margins of the 12 and 22 were lower than those of 13 and 23. Three millimeter gingival pockets were seen on the labial aspect of 12 and 22. Clinical crowns of 12 and 22 were shorter than their anatomical crowns. Both 12 and 22 were vital when tested with an electric pulp tester and no pathology was observed on the periapical radiograph. After assessment on study models the following treatment plan was suggested to the patient.

1. Crown lengthening surgery on 12 and 22 with gingivectomy and alveolar bone recontouring on labial aspect.
2. Composite build up on mesial surfaces of 12 and 22 for partial closure of the space as an interim measure.

After obtaining the patient's consent, surgery was performed. He was asked to rinse the mouth with a 0.2% chlorhexidine for one minute prior to giving infiltration anesthesia. After obtaining local anesthesia, a crevicular incision was made in relation to 13,12,22 and 23. The labial bone was recontoured in relation to12 and 22 reducing the height by 1 mm. Thereafter the gingiva was trimmed by 2 mm, in relation to 12 and 22. Finally the flap was replaced and sutured with simple interrupted sutures. Antibiotics (Amoxycillin 250mg 8 hourly and metronidazole 200mg 8 hourly for 5 days) and an analgesic (Ibuprofen 200mg 8 hourly for 3 days) were prescribed with 0.2% chlorhexidine mouthwash three times a day for one week. As the wound healing was satisfactory when examined after one week, sutures were removed. Recontouring of 12 and 22 to simulate 11 and 21 was performed with composite resin restorations (shades A2 and A3), 2 weeks after suture removal. The patient was satisfied with the final aesthetics

Discussion

Surgical crown lengthening is an invaluable treatment adjunct available to overcome the problems related to retention and shape of a future restoration. However performing gingival surgery with predictable results is clinically challenging. Moreover obtaining a biological width of 3.0 mm, following crown lengthening surgery is not routinely achieved.[10] The acceptable final results achieved in management of these two cases could be attributed to proper treatment planning, surgical techniques and patients' cooperation.

On a previous occasion the diastema of the first patient had been closed by broadening the teeth 11 and 21. This had altered the shape of the incisors. Square shaped teeth with inadequate scalloping at the gingival margin can result in delay in passive eruption.[2] As the cemento enamel junction of the 11 and 21 were observed at a depth of 1.5 mm within the gingival sulcus, this was unlikely to be the case in this patient. Over eruption of 11 and 21 due to loss of tooth contact with the opposite teeth, following fracture and subsequent restoration could have been a possible cause. As the patient was young and was to get married soon, it was decided to improve the aesthetics of upper centrals as much as possible in the shortest possible time. Porcelain fused to metal crowns was not selected as the treatment option because of the long waiting list for such procedures in the clinic. As it takes several months to establish the

also logical to postpone placing crowns for several months.[9] The presence of 3 mm, periodontal pocket on the labial surface of 11 and 21, accompanied with a 4 mm, width of keratinized gingiva led to the decision of apical repositioning rather than gingivectomy.

In the second patient, the space between the upper lateral incisors was inadequate to accommodate two normal size central incisors. It was also too broad to be closed by recontouring of lateral incisors to simulate central incisors. Partial approximation of 12 and 22 by orthodontic means would have improved the final outcome but the patient was not willing to wait that long. Therefore a compromise treatment plan was formulated although the final results would be less than ideal. Crown lengthening was performed to maintain the proportions of the crowns when recontouring the lateral incisors. In contrast to the first patient, the second patient had two lateral incisors with short clinical crowns due to delayed passive eruption.[2] Crown lengthening, therefore, needed to be accompanied with gingivectomy to obtain optimum results.

As discoloration of composite restorations is a well-known problem, porcelain veneers or porcelain fused to metal crowns are considered to be ideal treatment options for tooth recontouring. Due to the inadequate tooth substance remaining as a result of tooth fracture in the first patient, and inadequate stability of the gingival margins in both patients, it was decided best to avoid porcelain veneers. However, porcelain fused to metal crowns can be considered together with cast metal dovel cores at a later stage in the first patient. Porcelain veneering would be the ideal treatment for the second patient when the gingival margin has stabilized.

Although a remarkable improvement in aesthetics was achieved in a short time, meticulous plaque control with proper brushing technique is a must to ensure long lasting periodontal health and aesthetics of both patients.

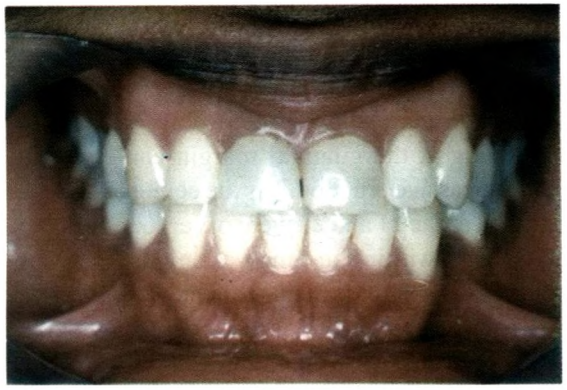

Figure 1. Pre operative appearance.

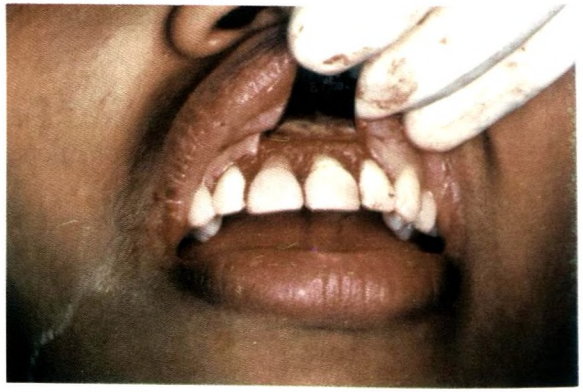

Figure 2. Crestal bone had been re-contoured in relation to11 and 21 after raising a mucoperiosteal flap.

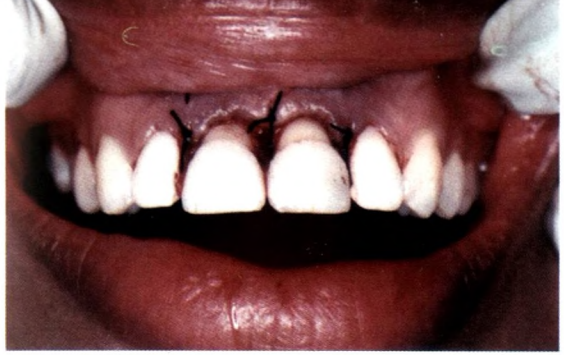

Figure 3. Immediately after apical repositioning of flap.

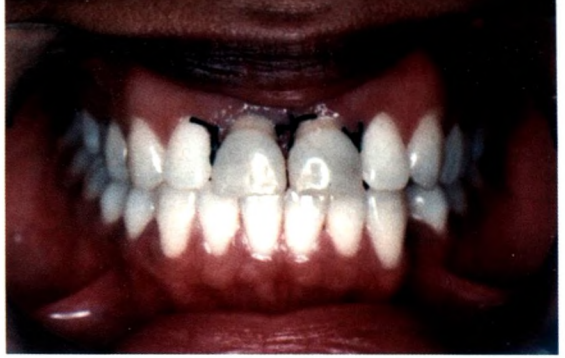

Figure 4. One week after the surgery (just before suture removal)

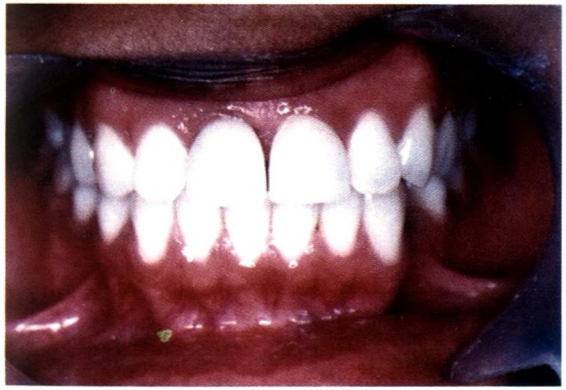

Figure 5. Final appearance with new composite veneers on 11 and 21.

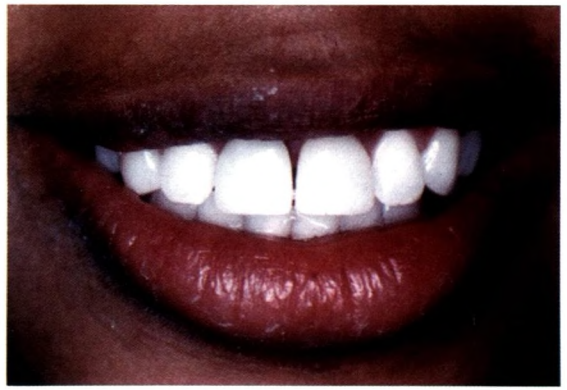

Figure 6. Post operative smile.

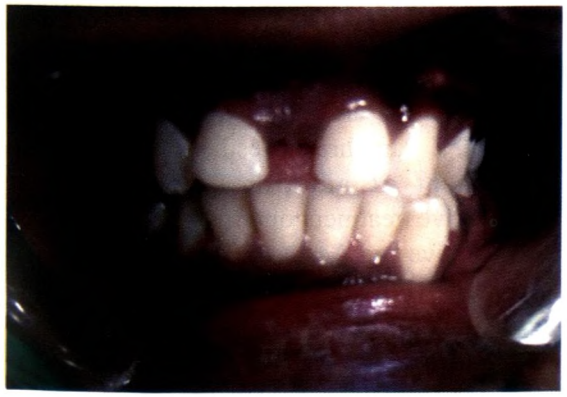

Figure 7. Pre operative appearance.

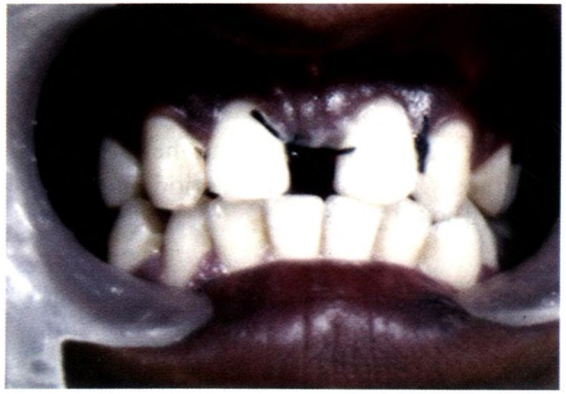

Figure 8. One week after the surgery (just before suture removal).

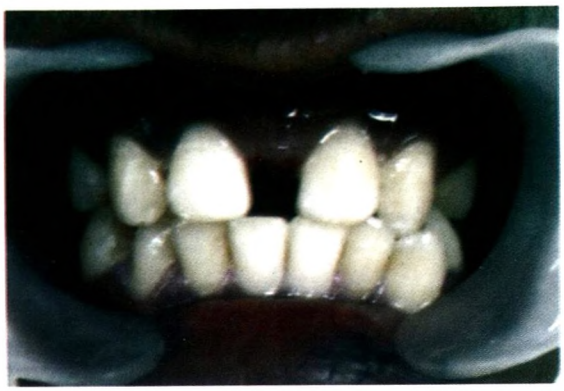

Figure 9. Three weeks after the surgery.

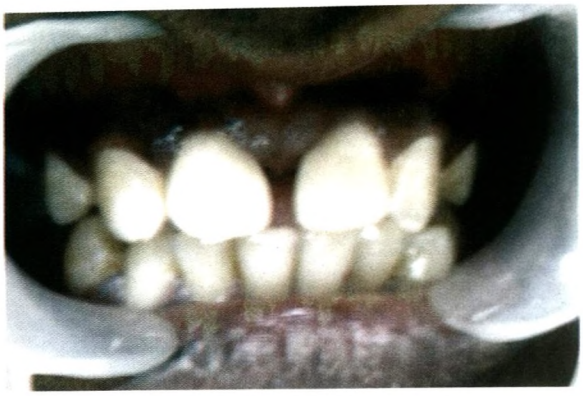

Figure 10. Final appearance following recontouring of 12 and 22.

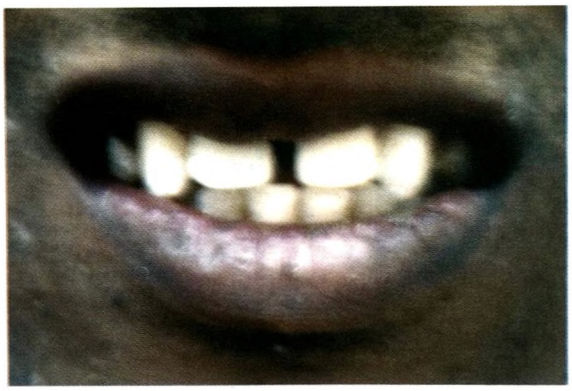

Figure 11. Final appearance following re-contouring of 12 and 22.

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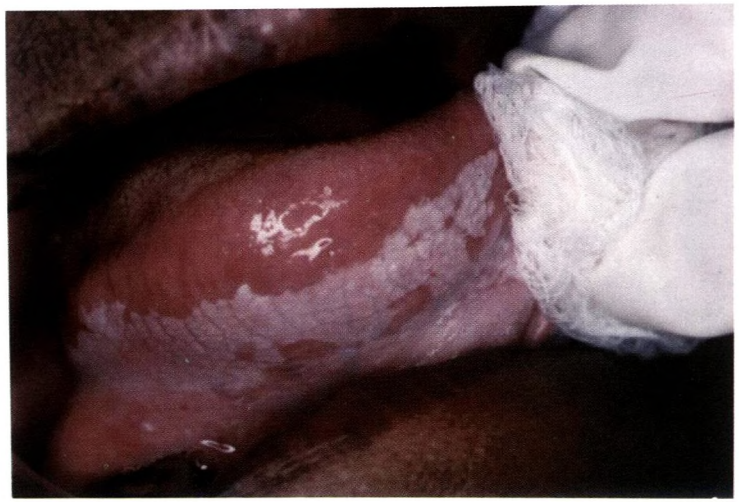
Self Assessment / Oral Diagnosis (SAOD)

A 65-year-old man complains of a white patch on the ventral surface of the tongue as shown in the picture.



1. Which of the following features in the history are important in arriving at the differential diagnosis
   a. Habit of smoking
   b. Family history
   c. Habit of betel chewing
   d. Drug abuse
   e. Dietary history

2. What are the moste probable differential diagnoses
   a. Oral submucous fibrosis
   b. Hairy leukoplakia
   c. Sublingual keratosis
   d. Pachyonychia congenita
   e. Lichen planus

3. What are the essential steps in the management of this lesion/condition
   a. incisional biopsy
   b. systemic antifungal therapy
   c. radiotherapy
   d. habit intervention
   e. application of topical corticosteroids

Answers

1. a,c
2. c,e
3. a,d

Prepared by Dr. Anura Ariyawardana BDS(SL), MS (Col),
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1. Leading articles - One article per issue. It may be solicited by the Editor. Authors are welcome to submit leading articles on current topics of interest. One's expertise or commentaries on general practice etc. They should be approximately 1500 words in length. References should be 20 or less.

2.Reviews - Reviews are detailed surveys of published research pertinent to dentistry and associated sciences. They should be critical in nature and should not normally exceed 3000 words and 30 references.

3. Research articles - Articles resulting from research work belong to this group. Results from routine clinical examinations or laboratory investigations will not be considered under this category. Subjects may vary from clinical trials to basic science research, historical analysis to dental economics. They should not exceed 3000 words and 30 references. A reasonable number of tables and illustrations will be accepted.

4. Short reports - These include reports on current topics, modified techniques. new materials, practice management etc. Interesting results from routine, clinical work or laboratory il1vestigations also may be accepted.

5. Case reports - Reports such as of rare diseases or conditions. Modifications to accepted treatment procedures, new management methods etc. may be included in this category.

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7. Miscellaneous topics - Subjects unlimited and the format are free. These may also include details of scientific meetings, conferences, annual sessions, examinations, news and views, visits and obituaries.

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I) Objective: An introductory sentence indicating the objective and purpose of the study.

2) Material and methods: A description of experimental procedure including applicable statistical evaluation.

3) Results: A summary of the new. Previous unpublished data and results.

4) Conclusion: A statement of the study's conclusion 3-5 key words according to Index Medicus should be provided.

Introduction - The introduction should carry sufficient background information on the subject of study.

Material and methods - Procedures should be described in such detail as to make it possible to repeat the work. Subheadings may be used to improve clearness. Correct unit abbreviations should be used (e.g.; "h", "min", "s" and "Fm" rather than "hr", "minutes", "sec" and "Fl". respectively).

The authors should consider the ethical aspects of their research and ensure that the work has been approved by an appropriate Ethical Committee. Where applicable, a copy of the ethical clearance certificate should be attached. In human experimentation. informed consent from individuals should be Obtained and this should preferably be stated.

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Results - The Results section should clearly and concisely present the findings of the research, as a rule in the past tense without subjective comments and reference to previous literature. The results should be supported by statistical or illustrative validation. For the sake of clarity this section may have subheadings.

Discussion - This section should present the interpretations of the findings and is the only proper section for subjective comments. Authors are strongly urged to avoid undue repetition of what has been reported in Results.

Tables - The tables should be numbered in the order" of appearance in Arabic numerals, Each table should have a brief explanatory title. Each table; should be typed on a separate sheet, with due regard to the proportion of the printed column/page.

Figures - All graphs, drawings, and photographs are considered figures and should be numbered in the order of appearance in Arabic numerals. Each figure should have a brief and specific legend, and all legends should be typed together on a separate sheet of paper. Photographs should be glossy prints and the reverse should give the figure number, title of paper principal author's name and have a mark indicating the top. Colour illustrations may be submitted in instances where their use may contribute significantly to the scientific value of the article. Colour illustrations may be printed free of charge at the Editor's discretion, whereas others may be printed at the author's expense.

References - References are listed double-spaced in a separate reference section immediately following the text. References are numbered consecutively in the order in which they appear in the text; do not, alphabetise. Identify references in texts, tables and legends by Arabic numerals (within parenthesis).

Congress abstracts should not be used as references nor may "unpublished observations" and "personal communications" be placed in the reference list. References cited as "in press" must have been accepted for publication and not merely in preparation or submitted for publication

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Journals

Standard journal article

Bartlett IG, O'Keefe P. The bacteriology of the perimandibular space infections. JOral Surg 1979; 37: 407409.

Corporate (collective) author

WHO COLLABORATING CENTRE FOR ORAL PRECANCEROUS LESIONS. Definition of leukoplakia and related lesions: an aid to studies on oral precancer. Oral Surg Oral Med Oral Pathol 1978; 46: 518-539.

Unpublished article

Barker DS. Lucas RB. Localised fibrous growth of the oral mucosa. J Dent Res 1965: in press.

Books and other monographs

Pindborg JJ Atlas of diseases of the oral mucosa. 5th edition.. Copenhagen: Munksgaard, 1992: 50-66.

Chapter in book

Boyde A. Amelogenesis and the structure of enamel. In: Cohen B. Kramer KH (eds). Scientific Foundations of Dentistry. William Heinemann Medical Books Ltd. London. 1976: 335-352.

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International statistical classification of diseases and related health problems, 10th revision, vol 1. Geneva: World Health Organisation, 1992; 550--564

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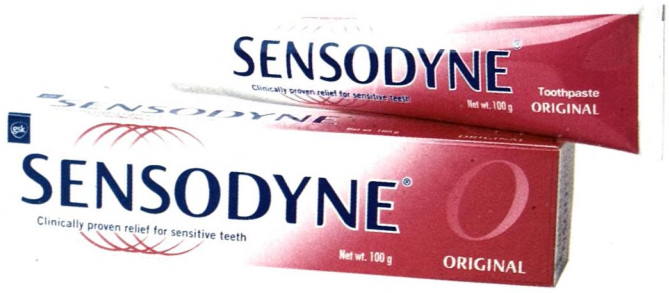
Dentine can be exposed by gum recession or enamel loss caused by:

* *Harsh tooth brushing*
* *Excessive flossing*
* *Intake of acidic food and drink*
* *Frequent vomiting*
* *Gum disease*
* *Previous dental work or*
* *The use of dental products with abrasive ingredients*

What usually triggers the pain?

* *Exposure to cold or hot food, liquids or air (including drying up of teeth)*
* *Exposure to sweet or sour food and drinks*
* *Tooth brushing*



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