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BUILDER

A QUARTERLY PUBLICATION OF HEALTH CARE TRUST FOR GENERAL DENTAL PRACTITIONERS

FROM THE EDITOR'S DESK

In June, Health Care Trust had two dental students from Sweden as visitors. They were directed to Belgaum by Dr. Douglas Bratthal, who is deeply involved in Cariology and is a WHO consultant.

After observing the dental teaching in India, these students from The Centre for Oral Health Sciences, Lund University, Malmo, exclaimed at the number of extractions done by dental students in our country.

The Centre for Oral Health Sciences in Malmo is unique in the world in training dentists exclusively by means of Problem- Based Learning (PBL). The method was developed for training doctors at Mc Master University in Canada and it is used today for medical training at many Universities including Harvard, the Health University in Linkoping, and Lund University.

PBL is a pedagogical approach that gives the students responsibility for their own learning. The central concepts are learning and problem solving. The principle of PBL is to give the students a task or challenge (practical cases from real situations, like an young boy with a fractured tooth) as a source for learning. These challenges from real situations are of the kind they will encounter in their professional future.

Normally six or seven students collaborate to solve each case, with a tutor to guide. To decide how to treat their patients, the students have to dig out facts about teeth, jaws, growth, methods of treatment and dental materials.

Reasons for adopting PBL in health education are the acquisition of such professional skills as the learning of decision making, clinical reasoning, the holistic approach, self-directed learning for life long learning and collaboration in teams.

In traditional teaching, we may have a false confidence that we have been taught dentistry, to practice for a lifetime. In PBL, each clinical situation will be a stimulus for continuous learning, and the individual is well aware that scientific information gets outdated every five years. **PBL inculcates an attitude to analyze and solve each confronting problem**.

Look at the havoc created by the abuse and misuse of antibiotic drugs! How many of us are aware of our role in this global threat of antibiotic- resistance problems? **It is imperative to put a restraint on antibiotic use-right now.**

Dr. Beena Rani Goel, M.D.S.

INSIDE

We plan to start an elecronic newsletter on the internet. If you have internet account please inform us your e- mail address.

*Please visit our website http://www.poboxes .com/goel

Removal of Amalgam Restorations- Ethical considerations

When a patient comes with a request to remove serviceable amalgam restoration from his teeth, **how** ethical is it to accede to his request?

According to ADA General Legal Counsel Peter Sfikas, "The ADA ethics policy clearly states that 'no dentist shall remove an otherwise serviceable amalgam filling for the sole purpose of curing a systemic disorder'". Removal of dental amalgam has not been shown to have any beneficial effect on the general health of the patient or their specific medical conditions.

There is **absolutely no scientific** proof that removing amalgams for the alleged purpose of eliminating toxic substances from the body is beneficial, unless the person is allergic to the mercury. The scientific community agrees that amalgam is a safe and durable tooth filling material.

If a patient asks the dentist to remove serviceable amalgam restorations, the dentist would do a right thing by adhering to the following **guidelines** :

Based on the overwhelming body of scientific data supporting the safety and efficacy of dental amalgam, there appears to be no justification for discontinuing the use of amalgam, where they are indicated.

-The dentist is free to suggest that the patient seek dental care elsewhere.

-If the dentist agrees to remove serviceable amalgam restorations from the non-allergic patient at the patient's request:

-The dentist should take special care to obtain the patient's informed consent to the procedure and thoroughly document that consent in the patient's records.

-The dentist should review with the patient the current scientific thinking on the safety of amalgam –that there is no evidence that amalgams pose a significant health risk to non-allergenic patients and that no known health benefits result from removal of dental amalgams.

-The patient should be informed of the risks involved in replacing amalgam restorations, including potential damage to healthy tooth structure and the loss of sound tissue in the process.

The patient should also be informed of the risks and benefits of the replacement materials and the cost.

Finally, the dentist should clearly state that he or she promises no health benefits to the patient by removing serviceable amalgam restorations.

Thus, serviceable amalgams can ethically be removed by a dentist but only under very limited circumstances and only if the patient provides informed consent to the procedure.

So, **is amalgam safe**? In relationship to mercury exposure from dental amalgam, available data have not identified any side effects. To date, there is no evidence to suggest that mercury from dental amalgams results in any adverse effects to health in the general population. However, several recent studies support earlier work suggesting that a very small percentage of people- less than 1 %- may have allergic reactions to mercury, as well as other metals from amalgam.

A recent study of Swedish twins came to the conclusion that there are **no negative side affects** from dental amalgam on physical or mental health or memory functions, even after the researchers controlled for age, sex, education and number of remaining teeth. Another Swedish study involving 1,462 women provided no evidence of a correlation between dental amalgam and cardiovascular disease, diabetes, cancer, death rate or various subjective symptoms such as irritability, depression, fatigue and readiness to cry.

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Based on the overwhelming body of scientific data supporting the safety and efficacy of dental amalgam, and the absence of any similar database attesting to the safety and efficacy of an alternative material, there appears to be no justification for discontinuing the use of amalgam, where they are indicated.

Ref:

-JADA 129:494-503, 1998.
-Community Dent Oral Epidemiol 24:260-267,1996.
-Community Dent Oral Epidemiol 21(1): 40: 44,1993
-Swedish Dent J 62(Supplement): 40-43,1989.
-Internet.

UNDERSTANDING DENTAL CARIES- (contd. from last issue)

With the present scientific knowledge,it is time we stopped using the term 'dental caries' synonymous with a carious cavity.

Dental caries is an infectious disease that manifests itself by the demineralisation of dental tissues. Coronal caries is seen first as an increased microporosity of the enamel, which can be detected only after carefully drying the tooth surface. Clinically, the opacity generated by bacteria appears white because of the alteration in the refraction of light through enamel. As the area of microporosity increases in size and volume, the covering enamel collapses, forming a cavity. The demineralisation process is slow and is reversible before the cavitation occurs.

With the present scientific knowledge on the etiology and pathogenesis of caries, **it is time we stopped using the term 'dental caries' synonymous with a carious cavity**. The signs of this disease should be recognised as being continuous changes of increasing severity leading to tooth destruction. They are not the disease itself. Carious process is dynamic demineralising and remineralising changes as a result of the microbial metabolism on the tooth surface which, over time, may lead (but **not always**) to cavitation.

Presently available evidence suggests that mineral loss and the subsequent cavity formation in teeth is a result of imbalance in the dynamic equilibrium between tooth mineral and surrounding plaque.

A carious lesion is the accumulation of numerous episodes of demineralisation and remineralisation. If at any stage of the lesion development, the physiologic balance between demineralisation and remineralisation is restored, progression of caries lesion will be arrested.

The **detection** of precavitated lesion is very **crucial** to prevent the progression of dental caries to cavitation.
The prevention of dental caries must be based on detection of early signs of demineralisation and disease activity. Though this concept is clear to the students of cariology, it is the need of the hour that this be understood and endorsed by dental practitioners. It is time we did a soul searching and see how we have managed dental caries and how we should manage it in future, armed with proper understanding of dental caries, its risk factors and prevention.

A **precavitated or non-cavitated lesion** has two important characteristics.
1. Lack of macroscopic loss of tooth structure.
2. Demineralisation of enamel, dentin, or both.

It is **important** to detect precavitated lesions due to several reasons.
- -Precavitated lesions are significantly more prevalent than cavitated lesions.
- -Dentists are often tempted to restore these lesions, which is not necessary.
- -Detection of precavitated carious lesions in infants and young children may **predict** high carious activity.
- -These lesions can remain unchanged for years with fluoride therapy.

The most valid currently available method for detection of precavitated carious lesion seems to be **Fiber Optic Trans Illumination** (FOTI), followed by radiographic examination and visual inspection.

Use of a sharp explorer in a precavitated carious lesion should be **avoided.** Such a lesion could be remineralised or managed by **tooth preservative technique** (sealants or micro restorations). The application of force on an explorer could damage a tooth surface and convert a white spot lesion into a cavity.

A **blunt periodontal probe** can be used when required, to clean a tooth surface of plaque and debris prior to examination and to check the surface texture of a lesion without penetrating it.

Precavitated lesion can be located in:
- ♦pits and fissures
- ♦buccal / lingual surface
- ♦approximal surfaces

Pits and Fissures: The usual detection of yellow or brown discolouration warrants the need for further inspection. The presence of white demineralisation lines around the sides of a pit or fissure provides further evidence that carious demineralisation has taken place. Softness should be assessed using a

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Antibiotics- Boon or Bane?

The discovery of penicillin by Alexander Fleming in 1928 marked the most important therapeutic advance in medical history. Penicillin symbolized man's triumph over the bacterial world. The antibiotic era was born in 1940, when the clinical use of penicillin began due to the pressures of World War II.

Unless we immediately stop abusing antibiotics, we may soon find these miracle drugs to be totally ineffective

Penicillin saved countless lives in World War II and led to the discovery of natural antibiotics and development of synthetic antibacterials. For 10 years after the end of World War II, penicillin and other new antibiotics were available over the counter (and still are, in our country) without prescription. The message perceived through massive advertisement was that antibiotics would cure all ills and even if they were not effective, would do harm.

It is a well-documented fact that **antibiotics have no effect on viruses and some other members of the antimicrobial world**. This fact is ignored as clinicians succumb to patient's demands for antibiotics for viral infections such as common cold and influenza.

Physicians and dentists began to prescribe antibiotics for almost any conceivable condition that may predispose to infection, often at the insistence of patients. Even the simplest of surgical procedures, with little chance of post surgical infection, were (and still are) frequently accompanied by routine prophylactic administration of antibiotics. Evidence that this practice often increased the incidence of post surgical infections was generally ignored.

In ancient times, infectious diseases were spread very slowly. During the last 50 years, air borne travelers incubating infectious diseases have been able to reach any point on the globe within 24 hours.

Some dramatic developments in infections of bacterial etiology are taking place now. Bacteria that once readily succumbed to one or more antibiotics have developed resistance to these medications. Fatalities from gonorrhea, pneumonia, tuberculosis, dysentery and septicemia are increasing with alarming regularity. This could happen because of two factors.

-the remarkable genetic plasticity of bacteria to develop resistance to antibiotics and

-the abuse and misuse of antibiotics

There are four major mechanisms that mediate bacterial resistance to antibiotics.

-Bacteria may produce enzymes that inactivate the drug.

-Bacteria can synthesize modified targets against which the drug has no effect.

-Bacteria can alter their permeability or

-effectively afflux the drug so that an effective intracellular concentration of the drug is not achieved.

The genetic changes in the organism which bring about antibiotic resistance are:

-Mutation (a chromosomal change or

-Genetic transfer (acquisition of extrachromosomal genes.)

Mutation.

Like humans, bacteria possess genetic information that is passed onto their progeny. Since bacteria reproduce by binary fission, under favourable conditions, this exponential growth may result in the production of very large numbers of bacteria in a very short time.

Mutations result from the change of one or more nucleotide base pairs in chromosomal DNA. Induced mutations which are caused by external factors such as chemical agents (e.g,antibiotics), heat or irradiation, occur at far greater frequency than spontaneous mutations. Once developed, a mutant trait is usually passed onto all succeeding progeny.

In the late 1940s, resistant strains first surfaced in big city hospitals. Soon they spread to small community hospitals, and subsequently to people living in these cities and communities, some of whom had never direct contact with hospital.

Genetic transfer

Genes, including antibiotic-resistance traits, can be transferred among bacteria of the same or different strains as well as other species and even other generaions. Each new progeny becomes resistant and is a potential donor of resistance traits to a myriad of recipient bacteria,

Genetic transfer is of tremendous concern because it occurs in most bacterial species, frequently mediates multidrug resistance, and has an extremely high rate of transfer from one cell to another.

The microbial transfer of genetic elements,

including the flux of antibiotic-resistance genes involves

-Vertical transfer (i.e. to progeny) and

-Horizontal transfer (i.e. to other existing genera, species and strains). Even bacteria in a moribund state can participate in donation of genetic material.

It is highly important to understand that the resistant genes are resistant not only to the challenging antibiotic, but also to other antibiotics. Bacterial strains that are resistant to three or more antibiotic agents with different mechanisms of bacterial inhibition are defined as multidrug resistant. Multidrug resistance of bacteria under selective pressure by a single antibiotic has become the rule rather than the exception.

Use of antibiotics creates a phenomenon known as **selective pressure**, in which the more susceptible bacteria are destroyed and the more resistant bacteria survive. Dosage regimens that do not destroy the target bacterial population create selective pressure, resulting in a population of resistant bacteria. This population may not cause disease in the initial human host but may be spread to others and cause serious consequences.

The antibiotic-resistance problem is global in scope and very serious. It is of utmost importance to be aware that

-More than 90% of the strains of staphylococcus aureus are resistant to penicillin.

-The incidence of Vancomycin- resistant enterococci in the United States increased 20 fold between 1989 and 1993.

-The prevalence of antibiotic resistant Streptococcus pneumoniae has dramatically increased in the past 5 years.

-Antibiotic-resistance genes are present in all major pathogens.

-Bacterial strains resistant to all available antibiotics have recently been identified.

An enormous natural reservoir of antibiotic- resistance genes has been created by the abuse of these miracle drugs. The two major routes of antibiotic abuse involve the human food chain and human therapeutic use.

Human food chain

Antibiotics are used as growth promotants in livestock feed for cattle, lambs and poultry. Pasteurized milk contains antibiotics which are used on dairy cows to prevent udder infections. Well-documented evidence shows a steadily increasing incidence of the spread of the spread of multidrug- resistant bacteria from animal to man.

Passage of resistant bacteria and small doses of antibiotics from the animal ecosystem into the human ecosystem occurs by two routes- direct and indirect,

Direct passage occurs by ingestion of animal meat contaminated with resistant bacteria.

Indirect passage occurs from the use of animal manure as fertilizer for food crops. Animal fecal matter containing resistant bacteria is tilled into the soil or sprayed on the field where the bacteria multiply, are retained by the crops and are subsequently ingested by the humans.

Fruit trees and plants are often sprayed with antibiotics to prevent or treat bacterial diseases. Antibiotics are also used extensively in Aquaculture, thus contaminating the fish.

Every human or animal taking an antibiotic, therapeutically or subtherapeutically becomes a **factory producing antibiotic-resistance genes**, adding to the growing environmental pool.

Human therapeutic use

After the introduction of each new antibiotic, there follows a pattern of discovery, excessive use and obsolescence. Humans have ignored clear warnings that excessive or improper use of antibiotics would lead to a world filled with human carriers of bacteria resistant to most known antibiotics. The ultimate responsibility for the excessive human therapeutic use of antibiotics lies with health care providers, primarily physicians and dentists.

Clinicians must educate themselves to understand the resistance problem, accept the importance of their role in both the problem and the solution, and resolve to use these drugs in a far more appropriate manner. They should not accept the philosophy of 'antibiotics on demand' by the patient.

Broad spectrum antibiotics should not be used when a narrow spectrum antibiotic is indicated. Unnecessarily long courses of antibiotic therapy should not be prescribed. Prophylactic antibiotics should be avoided for simple surgical procedures when there is little chance of post surgical infection.

Antibiotics should not be prescribed for patient withlimited infections that are being controlled by the body's immune and inflammatory systems. Same applies for patients with dental pain, but no signs of infection such as swelling, lymphadenopathy or

contd on page-6

gentle lateral pressure on a periodontal probe without penetrating the lesion.

Buccal and Lingual Surfaces: The most common feature is the presence of white demineralisation parallel to the gingival margin, either close to or in contact with the gingiva. The white opacity is usually covered with plaque. These lesions may get stained and appear yellow or brown.

A precavitated lesion that appears in the middle of the buccal or lingual surface may appear glossy and perhaps it is arrested.

Approximal surfaces: Characteristics are same as that of buccal and lingual surfaces, but it is harder to determine whether the lesion is arrested or not.

With the present knowledge, carious lesions can be **classified into three types**.

1. Lesions where 'No Care is advised.'
2. Lesions where 'Preventive Care is advised.'
3. Lesions where 'Operative Care is advised' and 'Preventive Care is advised.'

Dr. Graham Mount and Dr. Rory Hume outline a system of classification, which is primarily related to future restorations. Some lesions (those that are reversible by chemical means, or those are not active) do not need to be restored. The system of classification can be used to record that such lesions are present, using the "size zero" classification, which means 'do not restore'.

To describe the lesion two numbers are used.

Site 1,2, or 3

Size 1,2, 3, or 4

Site1 – Pit and fissure

Site 2 – Approximal surface

Site 3 – Cervical area

Size 0 – small and early enough to be mineralized, or the lesion has been remineralized and there is just residual stain. 'Zero' means no restoration is necessary.

Size 1 – minimal dentinal spread- just beyond what can be mineralized.

Size 2 – moderate involvement of dentin.

Size 3 – enlarged, with weakened cusps or incisal edges.

Size 4 – extensive loss of tooth structure.

When you put the two numbers together and you describe the lesion- say, a 1.3 means site1 (pit and fissure) size 3(enlarged). A 2.2 means site 2(approximal) size 2(moderate).

When we add the tooth number to that, we know exactly what we have- say, a 2.3 (approximal, enlarged) on tooth # 12.

We have to adopt a disease- based strategy for managing dental caries, which can bring about a 60%reduction in operative procedures. Armed with the knowledge to arrest and remineralise precavitated carious lesions, we can brace ourselves for dentistry in the next millennium.

List of New Life Members

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contd. from page-4 .....elevated body temperature.

Restraint in antibiotic use should be the immediate response by practicing physicians, dentists and veterinarians. We have to strongly resist the temptation that antibiotics be prescribed "just in case". Tuberculosis, which now infects 1/3rd population , is a sign of things to come, if changes are not made in our prescription behavior.

Ref: 1. Quint Int. 29:151-162, 1998
2. Clin. Infect. Dis. 15:77-83, 1992
3. N Engl J Med. 311:617-622, 1984
4. JAMA 243:546-547, 1980

IN THE NEXT ISSUE

* **How should you choose digital radiography equipment.**
* **What you should do to protect yourself from back pain and finger fatigue**

ENDODONTIC SERIES-3

Pulpal diagnosis.

	Symptoms	Radiographic	Pulp tests	Periapical Tests
Normal	None of significance	No periapical changes	Responds	Not sensitive
Reversible pulpitis	May or may not have symptoms to thermal stimuli	No periapical changes	Responds	Not sensitive
Irreversible Pulpitis	May have spontaneous or severe pain to thermal stimuli	No periapical radiolucency	Exteme pain to thermal stimulus or palpation.	May or may not have pain on percussion
Necrotic	None to thermal stimulus	No significant change	No response	Depends on periapical status

Periapical diagnosis.

Normal	Not significant	No significant change	Responds	Not sensitive
Acute apical Periodontitis	Significant pain on mastication or pressure	No significant change	Depends on pulp status	Pain on percussion or palpation.
Chronic apical Periodontitis and apical cyst	None to mild	Apical radiolucency	No response	None to mild pain on percussion or palpation.
Acute apical abcess	Swelling and/or significant pain	Usually a radiolucent lesion	No response	Pain on percussion or palpation.
Chronic apical abcess	Draining sinus	Usually a radiolucent lesion	No response	Not sensitive.
Condensing osteitis	Varies, depends on pulp or PA status	Increased trabecular bone	Depends on pulp status	May or may not have pain on percussion or palpation