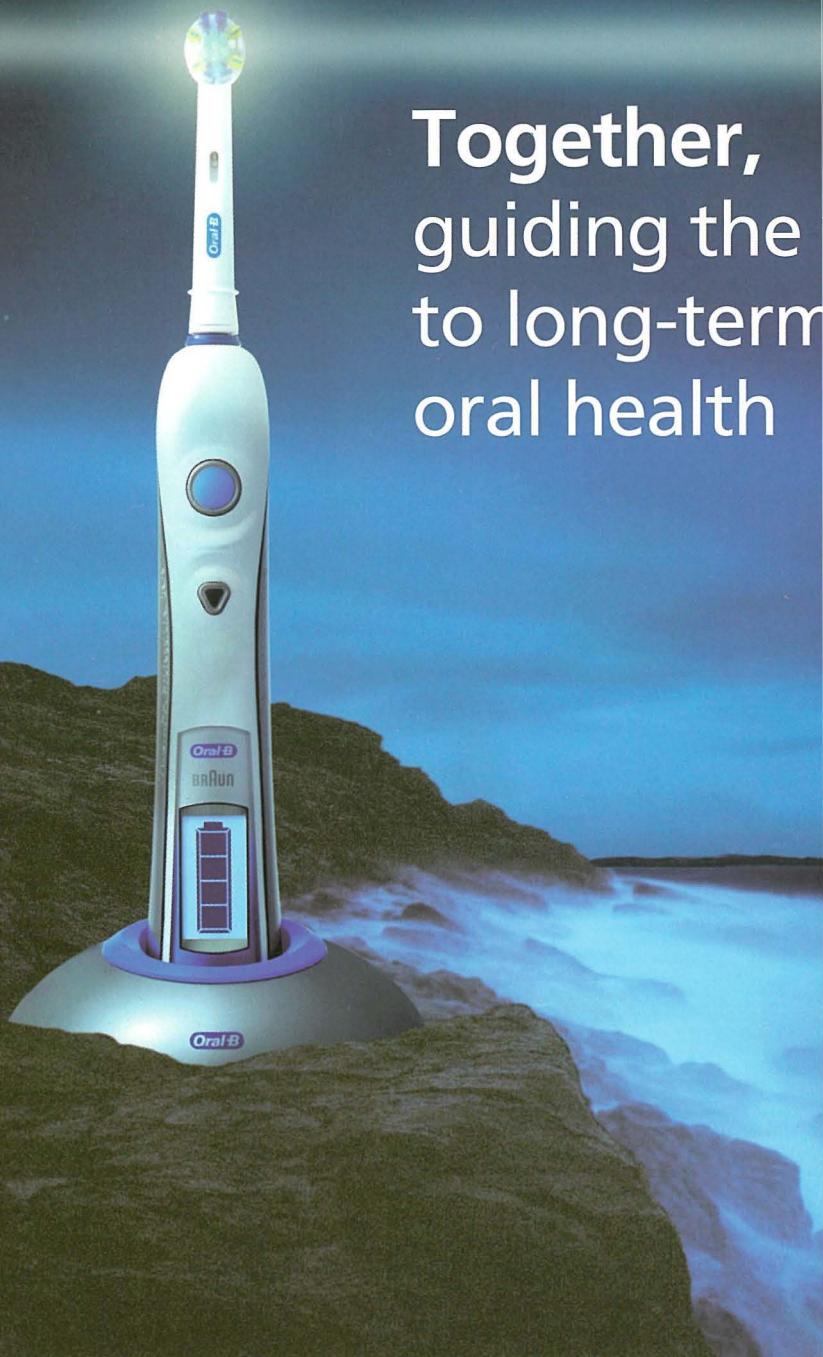


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The Maltese Dental Journal





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Editorial

By Dr David Muscat

Dear colleagues,

This issue features information about the latest AGM and also articles by Maltese professionals and about some of our colleagues, past and present. Drs Matthew Cachia, Ethel Vento Zahra and Robert Lautier have resigned from the DAM committee and have been replaced by Drs Noel Manche (treasurer), Darien Cini(CPE) and John Vella Bardon (federation Representative). We would like to thank the outgoing members for their hard work. They all resigned for personal reasons. The rest of the committee retained their posts. The next election will be held in January 2013.

This year promises to be another active one. So far our events have all been well attended with excellent organization and catering. This is the list of recent events, correct at time of writing this article.

Please note that the Medical Emergencies Courses are held every 3 months or so. Please book with Lino. The next one in April is already booked up so put your name down for the next. The course is open to all paid up DAM members at a reduced price of 50 euro and nurses can attend at the reduced price of 35 euro. The normal fee is 150 euro. In the absence of a sponsor the DAM is subsidizing the cost. Non DAM members will have to pay full fee so please ensure you pay your membership asap.

Front picture courtesy of Bart Enterprises on the occasion of Cerec 2nd anniversary of launch in malta

Best regards,

David

Dr David Muscat B.D.S. (LON)
Editor, Vice President and P.R.O. D.A.M.

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DENTAL ASSOCIATION OF MALTA

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RECENT/PLANNED EVENTS

4 FEBRUARY

Medical Emergencies course by Drs Adam Bartolo and Joanna Gonzi at Coastline Hotel sponsored by GSK.

8 FEBRUARY

8pm AGM with catering by Café Jubilee sponsored by DAM.

1 MARCH

Sanofi Aventis event .lecture by Dr Janet Mifsud."Effects Of Epileptic Drugs In Dentistry" At Yacht Club/ Guze restaurant.

10 MARCH

St Apollonia Mass at Madliena Chapel followed by lunch at Exiles restaurant.

19 MARCH

Lenten Mass St Joseph Convent Rabat 9.30am followed by lunch. Cost €20 each.

21 MARCH

8pm Agape Rabat lecture on 'The Doctors Case On The Corsairs Galleons' by Liam Gauci curator Maritime Museum sponsored by Abbott.

APRIL

Event by Galepharma – Protelos. Lecture on Bisphosphonates by a gynaecologist-to be announced.

MAY

Planned lecture/dinner at Civil Sports Club Valletta.

Planned Tour of Palazzo Parisio Naxxar followed by dinner.

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Above: The FEDCAR members of Europe in Paris 2011



Left: Dr David Muscat presenting the Dental Probe to Dr Diana Dabic from Slovenia in the presence of Dr Barney Murphy from Ireland at the Federation of Dental Competent Bodies and Regulators of Europe in Paris in 2011.

REPORT OF International Relations Officer

AGM FEBRUARY 2012

The role of international relations officer principally involves maintaining good contacts with FDI, ERO and CED (Council of European Dentists) and in providing constant update from the CED Brussels office. I will proceed to provide you with information on EU developments that are of interest to the dental profession and concerning health policy

MEETINGS ATTENDED

- a. CED meeting Budapest May 2011 (attended by Dr Audrey Camilleri)
- b. CED meeting Brussels November 2011 and Commemoration of the 50th Anniversary of CED (attended by Dr Paula Vassallo). CED President Dr Wolfgang Doneus recalled that the CED, established as the Dental Liaison Committee in 1961, evolved over the years into a fully autonomous political body, providing an authentic voice for practising European dentists in EU policies.

EU UPDATE ON TOPICS RELEVANT FOR DENTAL ASSOCIATION

1. DIRECTIVE ON THE RECOGNITION OF PROFESSIONAL QUALIFICATIONS (2005/36/EC)

The EC published the summary responses of the public consultation on the modernisation of the Professional Qualifications Directive (PQD) as well as the evaluation report of the PQD on 6 July 2011.

On 29 August 2011, the EC published a legislative Proposal for a Regulation

of the European Parliament and of the Council on administrative cooperation through the Internal Market Information System (the IMI Regulation). The IMI was developed by the EC to allow national, regional and local authorities to communicate quickly and easily with their counterparts abroad.

On 19 December the European Commission adopted a proposal for amending the Professional Qualifications Directive (2005/36/EC) - see link

http://ec.europa.eu/internal_market/qualifications/docs/policy_developments/modernising/COM2011_883_en.pdf

The Board Task Force Internal Market and the Working Group Education and Professional Qualifications Will prepare CED's position in this regard.

The CED Office will attend a briefing with the Commission on 2 February to discuss the details of the legislative proposal. The CED Office will also meet with the sectoral professions on 16 January to discuss possible joint strategies.

LANGUAGE REQUIREMENTS

The CED believes that particularly for healthcare professionals the knowledge of the host Member State's language(s) is necessary and justified for reasons of patient safety.

Healthcare professionals should be able to communicate with their patients in a proper way (to obtain informed consent, to

inform them about the procedure and the risks, to explain treatment options, etc.) and understand fully the information given by the patients. Misinterpretation in healthcare can lead to fatal errors.

As the vast majority of dental practitioners are self-employed, the control by employers of linguistic knowledge practically does not exist.

Hence, to access the profession, a control of language skills needs to be made by competent authorities or professional organisations before dental practitioners first come into direct contact with patients.

Therefore, the documents required under point 16 – "Linguistic knowledge" – as acceptable practice on the Code of Conduct approved by the Group of Coordinators for the Directive 2005/36/EC should become enforceable and incorporated in Article 53 of Directive 2005/36/EC.

CPD

The CED does not support harmonisation of continuing professional development (CPD) at EU level and therefore does not feel that there is a need to address this issue more extensively in the PQD.

Recital 39 and Article 22(b) of the PQD should be maintained as they are. CPD for dental practitioners is executed very differently in each Member State: it is defined according to each population's oral needs, in a given time, and it can

be obtained in different settings. In addition, each dental practitioner chooses his specific lifelong continuing education programme on the basis of his personal and professional interests, as well as his needs.

The diversity of continuing education activities on offer and the principle of free choice by the dental practitioners themselves should therefore be maintained in line with each Member State's specific rules for CPD.

These rules already meet dental practitioners' expectations and are adapted to national oral health needs. The principles of proportionality and subsidiarity should apply in this context.

MINIMUM TRAINING PERIODS FOR DENTAL PRACTITIONERS

The CED would support clarifying the minimum training requirements for doctors, nurses, midwives and dental practitioners.

The minimum duration of training for dentists should be expressed not only in years (5 years) but also in training hours (5000 hours), in order to safeguard against part-time training and the proliferation of "weekend diplomas" by private Universities

The new criterion of 5000 hours should be implemented in a flexible manner by Member States and universities.

In absence of the strongly supported introduction of cumulative criteria

for dental practitioners, the CED does not support any other amendments in relation to the minimum duration of training in the Directive.

2. TOOTH WHITENING

On 20 September 2011, the EU Council adopted the Council Directive amending Directive 76/768/EEC, concerning cosmetic products, for the purpose of adapting Annex III thereto to technical progress.

Following the Council's decision, tooth whitening products containing up to 0.1% of hydrogen peroxide will continue to be freely available to the consumers on the market.

For products containing between 0.1% and 6% of hydrogen peroxide, clinical examination and first treatment by a dentist will be required, to ensure the absence of risk factors or oral pathologies, after which the patient will be able to continue the treatment by him or herself.

The use of these products by persons younger than 18 years will not be allowed. Tooth whitening products containing more than 6% of hydrogen peroxide will continue to be prohibited.

In Malta the Directive is being transposed into national legislation at the moment and we have given them our feedback as to the appropriate wording to ensure proper transposition.

The CED had called repeatedly for appropriate regulation of tooth

whitening products at EU level in line with the advice from the European Scientific Committee on Consumer Safety which stressed that higher concentration products can be safe when used after a clinical examination and under the supervision of a dentist.

The CED considers the Council decision to be a very important step towards ensuring patient safety and removing any uncertainty as to how and by whom tooth whitening could be performed in the EU.

3. DENTAL AMALGAM

BIO Intelligence Service (BIOIS) has been contracted by the European Commission (EC) to carry out a study on the reduction of mercury pollution from dental amalgam, which will imply an assessment of the use of mercury in dental amalgam.

BIOIS has sent a questionnaire to key stakeholders and investigators across the EU to gather concrete information and sound data on the issue. BIOIS will present a final report in February 2012. A stakeholders' meeting is planned for March 2012.

Many thanks for the constant support in my work as International Relations Officer and if any member needs further clarification or advice on EU matters relevant to dentistry please feel free to contact me. 

*Audrey Camilleri
EU/International Relations officer*

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*Based on an *in situ* study.

Reference: 1. Maggio B *et al.* J Dent 2010; 38(53): 537–544.

THE DAM Administrative Report 2011

By Dr David Muscat PRO DAM

The Dental Association of Malta has in 2011 been involved either directly or indirectly with the organisation of 12 lectures and courses and 6 social events. We provide CPD certification for all our professional lectures.

The lecturers included Drs. Dan Keir, Michael Escudier, Charles Galea Bonavia, Kevin Mulligan, Harold Bergman and professor George Camilleri. The course was by Simplant(Bart Enterprises) and there were other lectures and social events organised by companies such as Voco, Kin,Aurobindo, Oral B, Denplan, Europharma, MT Pharma and Bial, Sanofi Aventis and Novartis.

We have had excellent social events and have been to new venues such as Lo Squero, Palazzo DePiro, Casino Di Venezia and the Red Tower in Landrijet. Cafe Jubilee has also proved very popular as an after lecture venue. This has been made possible by lino Dr. Lino Said our Social events organiser and I, the PRO have thoroughly enjoyed organising them and seeing them to fruition. Lino is one of the best genuine organisers I have ever worked with.

The DAM committee held 11 committee meetings.

The DAM has this year been involved in advising the Director General on Bisphosphonates and on antibiotic cover policy. We have also recently been involved with advising the government on its wording of policy regarding tooth whitening.

We have strongly opposed the introduction of tooth whitening technicians as we have also opposed denturists. We have worked on the language issue. From this year there

will be some checking of language before registration when it hopefully is passed at EU level.

We HAVE used 5000 euro to purchase a state of the art 'Resuscitation LAERDAL 'manikin and training defibrillator unit which we are now using for very beneficial medical emergencies courses for our members. We intend purchasing a paediatric manikin in the future to complement our work.

The DAM international relations officer has represented the DAM on a number of European and international meetings.

The Dental Probe - the indexed Maltese Dental Journal is going strong and is given to visiting lecturers to Malta and also to foreign professors when we go abroad. This way we raise the profile of the Dental Association of Malta. It remains the main source of remuneration through its adverts.

We have organized a Vodafone package-most of this work was done by Drs Adam Bartolo ably supported by Dr. Busuttil Dougall and Dr. Paula Vassallo.

The DAM has negotiated a professional indemnity package with MIB .We also met the Malta Standards authority and discussed custom made devices , CE markings ,laboratory work and statements of Conformity. All the details may be found in the March 2011 Dental Probe issue.

Sadly Father Jaccarini passed away a few days after St Apollonia in 2011 and we now have Father Mark Sultana, a professor of theology who will cater for our spiritual needs. This year we are trying a new venue-the chapel in

Madliena followed by lunch at the Exiles restaurant in Sliema. We thank Dr, Lino Said and Dr Charles Galea-Il General for helping organise this .One must not forget the great work done by Dr Roger Vella for 25 years in organising St. Apollonia.

In the DAM committee Ethel has had a baby girl, Leah. Audrey is due in March and also Robert Lautier's wife Elaine is also due in May this year- a very productive and also very reproductive committee indeed.

Matthew and Nick have both attained further degrees and they are to be congratulated. Dr Matthew Cachia has done an excellent job with our finances and Dr Dougall has been exemplary on his laptop .Dr Robert Lautier has given us certification and Dr Audrey Camilleri have steered us through the EU rapids. Drs Ethel Vento Zahra and Paula Vassallo, our secretary have been our ambassadors at the federation.

This year we hope to host events at Palazzo Parisio in Naxxar. Besides the medical emergencies course partially sponsored by GSK we are organising a lecture on drugs used in epilepsy in relation to dentistry at Guze restaurant and promote postgraduate education whenever we can. We hope to get funding to provide a BNF copy to all DAM members sponsored by Sanofi Aventis. Every member of the DAM committee gives up his or her free time to do voluntary work and this should be appreciated by all.

This year we raised 680 euro for the Equal Partners Foundation charity with our Christmas party raffle. ■

Dr David Muscat
Vice President PRO and
editor of *The Dental Probe*

THE DAM Secretary Report February 2012

During 2011 we had 9 committee meetings which often lasted well beyond midnight. We had our AGM on the 2nd February when the committee was re-elected.

In our First meeting following the elections the roles of each committee member were agreed.

Committee Roles were agreed

Adam Bartolo
President
Govt relations officer
SAC Representative

David Muscat
Vice president
Editor of the Dental Probe

Paula Vassallo
Secretary
MFPB representative
IRO substitute representative

Matthew Cachia
Treasurer
MFPB substitute representative

Nikki Busutil Dougall
IT officer
SAC substitute

Ethel Vento Zahra
Membership administrator
MFPB representative

Audrey Camilleri
International relations officer

Lino Said
Social events organizer

Robert Lautier
CPD officer

Kevin Mulligan
co-opted as EFOSA

This year we have focused on using some of our funds to buy resuscitation Annie in order to start providing basic life support courses to the members of the dental team at a highly reduced rate.

This can be seen by the success of last weekend at the Coastline hotel

We had numerous, over 20 social and scientific events thanks to the hard work by David and Lino but also by the rest of the committee who also work hard being the scenes.

We have also had several meetings with Vodafone to organize a good package for the members of the Dental Association.

As a committee we have also contributed to discussions within Europe as members of CED, where certain EU directives were passed as presented in the Probe Journal.

In fact the integration of Oral health into General Health, tooth whitening directive, lobbying in favour of amalgam, raising the importance of the language issue.

This year however some of the committee members will not be able to carry on in their role due to other commitments, namely Ethel, Rob and Matthew.

On behalf of the committee I would like to thank them all for their hard work and contribution and augur them well in their future.

Last and not least thank you to the members and the sponsors. ■

Paula Vassallo
Secretary

DR CHAR – AN APPRECIATION

Dr Charles Boffa was a consultant dental surgeon as well as an accomplished author and historian. He first qualified in pharmacy and then in dentistry. He went on to attain a PhD in Health Sciences as well as the FICD.

He was a demonstrator as well as an examiner in dental surgery and presented many papers at conferences in Malta, England and Austria. He was an elected member of the Medical Council, University Faculty Board as well as the National Archives Advisory Committee.

Charles was a caring individual and was a member of the Welfare Society for the Sick and Aged. The man cared about the welfare of the common man. In 1960 he received the Governor's commendation for giving dental attention to a crew member on a French tanker unable to enter Grand Harbour because of stormy seas.

In 1965 he was awarded a Testimonial by the Royal Humane Society (London) for rescuing a boy in imminent danger of falling off a bastion in Cottonera.

Dr Boffa was proud to be part of the dental profession and was respected by his colleagues and patients. He had a busy dental practice with clinics in Cospicua, Paola and, for some time, Zejtun and was a life member of the International College of Dental Surgeons.

Most importantly he was also a member of the Pierre Fauchard Academy of excellence. On a local level he was awarded "Gieh Santa Lucija" by the President of the Republic. He was keen on scouting and was awarded the Scouts Bronze Cross.

Dr. Boffa wrote about the ordinary citizens during the last world war. He researched on the life and characteristics of the Maltese people. Charles organized a natural history survey of Filfla and won the best documentary award for a film on Filfla.

LES BOFFA 26.9.30 - 4.12.11

ATION

By Dr David Muscat

He organised a natural history trip to Lipari, was in the first ever Maltese team to ascend Vulcano and also carried out humanitarian work in Calabria.

The main books by Dr. Boffa are as follows and the list is impressive: *The Second Great Siege, Malta 1940-1943* (1970); *It-Tlitt Ibliet Matul l-Ahhar Gwerra* 1940-1944 (1976); *The 'Illustrious' Blitz* (1995); *The Saga of the French Occupation of Malta 1798-1800* (1998); *Malta's Grand Harbour and its Environs in War and Peace* (2000); *The Islets of Comino, Filfla, Hagret il-General and the Volcanic One (Graham) that Was* (2003); *Santa Lucija and the Surrounding Area* (2007). As well as: *Maltese Cave Teeth – Pens of Prehistory; Significance of Teeth and Jaws in Forensic Medicine; The Islets of Comino and Filfla; Malta on the Verge of Starvation – 1942; April 1942 – Ix-Xahar Ta' Nar u Qirda fuq Malta; Over the Sea to Gozo; Occlusion, Mastication and the TMJ; Il-Friefet fil-Gzejjer Maltin; Hal-Tarxien u t-Tempji Neolitici; Effects of Certain Diseases on Dental-Oral tissues; Inharsu Sahlitna u Snienna, Dental Health Aspects.*



Charles was a family man. He was married to Melita née Schranz and had three children – Christine, Anne and Michael (who is a consultant dermatologist). It was fitting that at his funeral a sparrow flew into the church through an open window and



circled the altar for some time. Mother nature bid our friend farewell.

As past presidents and prime ministers mingled with the crowd, we laid to rest another of our country's greats – Charles, a scholar and a gentleman. ■

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MYOFASCIAL PAIN SYNDROME

By Dr Ryan Seguna MD BChd.

WHAT IS MYOFASCIAL PAIN SYNDROME?

Myofascial Pain Syndrome (MPS) is a painful musculoskeletal condition responsible for both acute and chronic pain. The main diagnostic criteria are the presence of trigger points (TrPs) and referred pain. Myofascial Trigger Points (MTrPs) are described as hyperirritable, taut/palpable muscular bands that are exquisitely tender to palpation and manipulation of which elicits referred pain with a characteristic pattern for each muscle.

Muscle weakness and reduced range of motion can also be part of the clinical presentation and these signs occur in the absence of muscle atrophy, thereby allowing the physician to differentiate it from other possible pathology like lower motor neuron lesions.

TrP manipulation should elicit a visible contraction of the involved muscle – called a Local Twitch Response. MPS may be missed on examination as it can manifest in a clinical conundrum masking its presence; like MPS complicating bursitis at a shoulder joint or rotator cuff syndrome.

Various factors are responsible for TrP formation; like muscular macrotrauma, repetitive microtrauma, reduced activity, nutritional deficiency, anxiety and psychogenic stress. The implication of muscle spindles in the pathogenesis of MPS explains why this condition is common in muscles with a relatively rich density of such receptor structures – like the cervical musculature and muscles of mastication.

Muscles subject to prolonged periods of activity will have an

increased neuronal output as is expected to maintain their state of tone. This can be a cause of repeated microtrauma that may ultimately manifest in chronic pain due to TrP formation. The following is just an example illustrating how complex and intricate MPS can present.

Individuals with a dolicocephalic profile tend to favour a degree of posterior repositioning of the mandible, thereby increasing tension within the muscles of mastication, namely the masseter m., pterygoids m. and temporalis m.

Such individuals also tend to bring their shoulders forward and this generates constant tension between opposing groups of muscles; the pectoralis major and pectoralis minor mm that protract the scapula are shortened, in contrast to trapezius m. and rhomboid mm. that retract the scapula which are stretched.

This also builds up tension in the suboccipital and posterior cervical musculature, even though they seem to keep their head in flexion.

SENSORY PATHWAYS FOR THE HEAD AND NECK

The modalities of sensation from the head and neck region are conveyed along fibres from the first three cervical segments and along sensory fibres distributed in the ophthalmic, maxillary and mandibular divisions of the trigeminal nerve, the fifth cranial nerve (VCN).

The branches from the cervical plexus that supply cranio-cervical territories are; the Greater occipital n. (C2-C3), Lesser occipital n. (C2), Transverse cervical n. (C2-C3) and supraclavicular nn. (C3-C4).

Like the other sensory pathways conveying sensation from regions of the body other than the head, namely the posterior column - gracile and cuneate fasciculi, and the spinothalamic pathways, the trigeminal system is a tri-neuronal pathway, with somas of first-order neurons housed in a ganglion (Gasserion) outside the CNS, the second order neurons originating from within the brainstem and whose fibres decussate en route to the contralateral medial division of the Ventral Posterior (VPm) nucleus of the thalamus, from where fibres project to the cortical regions reserved for the somatotopic representation of the head and neck – the inferior one-third of the primary somesthetic area.

As an exception to the general rule that primary/first order neurons lie outside the CNS, the cell bodies of afferent neurons mediating proprioceptive sensation in the head are located within the brainstem, more exactly within the mesencephalic nucleus. The central connections of these neurons are discussed below.

The sensory nucleus of the trigeminal nerve occupies the whole length of the brainstem and projects also into the upper three cervical segments of the spinal cord.

The spinal trigeminal nucleus and the dorsally related trigeminal tract are similar both functionally and histologically to the dorsolateral tract of Lissauer and the first four laminae of Rexed of the spinal cord. In the spinal cord, nociceptive fibres (C-fibres) come to lie more dorsal than fibres conveying the modalities of touch and this pattern is consistent also in the trigeminal nucleus.

The nucleus is divided into anatomically and functionally three distinct regions; the mesencephalic nucleus in the tegmentum of the midbrain, the pontine nucleus in the dorsolateral pontine tegmentum and the spinal nucleus extending the whole length of the medulla terminating in the cervical spinal cord as stated.

Central branches of first-order neurons enter the brainstem at the level of the pons to terminate in a specific region of the nucleus, depending on the modality of sensation they convey.

Large diameter fibres, for discriminative touch synapse with second-order neurons constituting the pontine nucleus. Fibres for light-touch divide on entering the pons, with one branch terminating in the pontine nucleus and another branch synapsing in the rostral two-thirds of the spinal nucleus – the Pars Oralis and Interpolaris.

Fine unmyelinated fibres responsible for afferent nociceptive input turn caudally on entering the pons and travel in the spinal tract to terminate in the spinal nucleus.

Like any other sensory or motor tract, there is a well delineated somatotopic organisation of fibres (homunculus) within the spinal trigeminal tract and also with respect to their target/junctional neurons in the nucleus.

As the fibres making up the sensory root enter the pons, they rotate and ophthalmic fibres that were dorsally related to the maxillary and mandibular fibres, (most ventral), now come to lie ventral in the spinal tract. As a result the mandibular fibres are shifted dorsally whereas

maxillary remain sandwiched in between the other two.

Like any other central nucleus, the trigeminal nucleus subserves a function, in this case general somatic sensation and so sensory fibres that anatomically constitute part of another cranial nerve but mediate this function also terminate in the spinal trigeminal nucleus.

This highlights the concept of nucleus homogeneity, in that it mediates a particular function, whereas a nerve is heterogeneous, consisting of fibres that may include branchial motor fibres, visceral motor, visceral sensory like the glossopharyngeal nerve.

As a consequence of this, central projections of first order neurons running in the facial and in the vagii nerves – supplying sensation in the ear and external auditory meatus, and fibres in the glossopharyngeal and vagii nerves conveying somatic sensation from the pharynx, larynx, middle ear, Eustachian tube, oesophageal mucosa, oral and lingual mucosa form the dorsalmost part of the spinal trigeminal tract.

Of the three subdivisions of the spinal nucleus, nociceptive fibres terminate in the pars caudalis. The pars oralis and pars interpolaris are mainly concerned with light touch. The somatotopic organisation of fibres terminating in the pars caudalis is such that pain afferents arising from the ipsilateral half of the peri-oral region come to lie in the most cranial part of the nucleus, with sequentially more dorsal facial regions following a crano-caudal distribution.

Of clinical relevance is the anatomical innervation of cerebral arteries by branches of the ophthalmic division (V1) of the trigeminal, in that they

mediate pathological mechanisms underlying certain headaches, like frontal headache, cluster headache and migraine, which will be discussed in the next chapter.

V1 is closely related to the internal carotid artery as they traverse the cavernous sinus. Branches of the ophthalmic division course with branches of the internal carotid artery forming a functional trigeminovascular unit.

The related phenomena of referred pain and poorly localised pain can be explained by the concept of 'convergence', whereby tract cells from the trigeminal nucleus may be excited and triggered to fire off action potentials by more than one afferent nociceptive projection, even though the latter show a relatively well delineated peripheral territory of which they are responsible – i.e. a small receptive field. This explains what may initially be confounding signs like headache of a temporal origin being referred to frontal and other regions that fall within the trigeminal territory.

'Convergence' and the fact that the caudal most aspect of the spinal trigeminal nucleus and the rostral-most aspect of the cervical cord are indistinguishable and intermingled provide an explanation. As a result, noxious stimuli conveyed along the greater occipital nerve (C2-C3), may for example, excite neurons that are usually responsive to stimuli elicited in the vertex or frontal region.

With regards to referred pain from deep somatic structures, some researchers do not accept the convergence-projection theory as the only explanation for pain referral.

Continues on page 12

MYOFASCIAL PAIN SYNDROME

Continues from page 11

Mense argues that with regards to deep somatic structures, there is relatively less convergence at the dorsal horn/spinal trigeminal nucleus, and that central sensitisation and activation of adjacent segments, (discussed in chapter 4), better illustrates the underlying events.

The mesencephalic nucleus of the trigeminal, located in the midbrain under the lateral aspect of the floor of the fourth ventricle, houses the somas of first-order neurons, a feature that, as already stated in the introduction to this chapter, is unique in that such cells normally lie outside the CNS.

Their axons divide into peripheral and central branches. The former convey proprioceptive sensation from the periodontal ligament supporting teeth and from muscle spindles, whereas the latter mainly target neurons in the motor nucleus of the trigeminal to establish the stretch reflex loop for the muscles of mastication and also to control jaw closure and the force of biting.

A note on the motor nucleus of the trigeminal: The motor nucleus of the VCN is located in the lateral aspect of the pontine tegmentum and houses the neuronal cell bodies of lower motor neurons, whose fibres innervate the muscles derived from the first pharyngeal arch, namely the muscles of mastication (masseter m, temporalis m, medial and lateral pterygoid mm), the tensor tympani m in the middle ear, tensor veli palatini in the soft palate, mylohyoid m in the floor of the mouth and anterior belly of digastric in the submental region.

This nucleus lies caudal to a region of the reticular formation called the supratrigeminal nucleus, with which it is intimately related via local neuronal circuitry.

This region, just like other regions of the reticular formation that

are involved with the pattern generation of movement; like the paramedian pontine reticular formation (PPRF) controlling aspects of gaze and other regions controlling locomotor systems, so is this nucleus responsible for generating the masticatory rhythm.

OVERVIEW OF ALTERNATIVE PATHOPHYSIOLOGICAL MECHANISMS CAUSING HEADACHE OTHER THAN DUE TO ACTIVE MTRPS

Headache is without doubt one of the most common symptoms that prompts patients to visit their doctor, and can be considered as a form of debilitating condition in that it does impair, although to different extents in different individuals, their normal daily life.

Migraine, for example and other forms of relatively severe painful episodic headache conditions, is one of the major causes of absenteeism from work. The literature reports estimated values for incidence of about 90% and for prevalence – 20%. This difference lies in the fact that incidence and prevalence are two distinct statistical entities.

Prevalence refers to the number of individuals suffering from the condition at any given point in time, it is a longitudinal analysis rather than a vertical one like incidence, which refers to the number of individuals diagnosed with headache per year. So these values show that ~90% of individuals suffer from an episode of headache a year and ~20% are managing headache at this point in time.

The headache chart provided gives the whole spectrum of conditions that can cause headache. In this chapter however, only the pathophysiology underlying the cause of migraine and cluster headaches will be mentioned. It is not intended in this chapter to fully describe in detail the pathophysiology of these conditions, but rather to mention the most

salient features and recent research advances about these conditions, and in doing so better highlighting the concept of how complex headache pathology is, even though signs and symptoms are sometimes almost identical among various conditions.

It is thus irresponsible and unethical to misuse medical terminology and tell patients "You have tension-headache" or "It's probably migraine or menstrual pain", without taking any interest to try and perform a proper diagnosis. Prescription of analgesics will only treat the pain and not the underlying cause of the pain, which is more important.

CLUSTER HEADACHES

In cluster headache (CH), as in migraine, the primary dysfunction that triggers the cascade of pathophysiological events is thought to reside in the CNS, more precisely in the hypothalamus (for CH). Before, it used to be believed that CH was mediated by vascular changes but is now known that changes in vascular calibre observed in cluster headache are a consequence of this abnormal discharge from the hypothalamus.

The main features that can serve as diagnostic criteria for CH are: episodes that tend to follow very faithfully a circadian pattern, autonomic phenomena that accompany such episodes and pain restricted to the area supplied by the ophthalmic division of the trigeminal nerve.

The suprachiasmatic nucleus (SCN), located in the hypothalamus just dorsal to the optic chiasm, acts as the 'biological clock' and is characterised by a fluctuation in its rate of firing during a 24hr period.

This periodicity is dictated by its input from the optic chiasm, consisting of retinal fibres that are sensitive to light, and is also serotonergically modulated.

Continues on page 16

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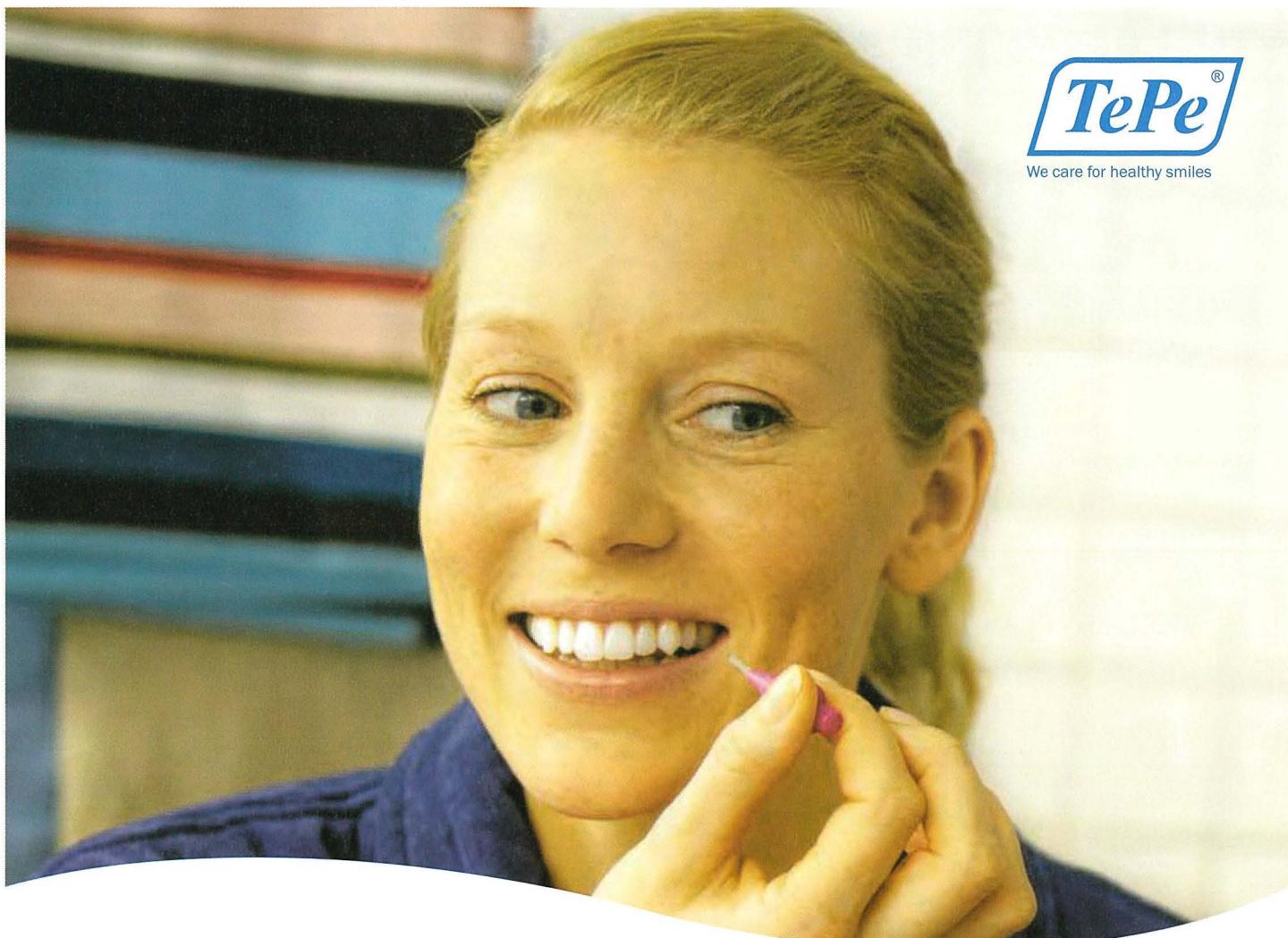
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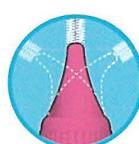
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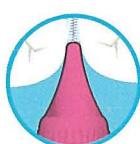
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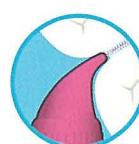
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Bradlaw and Dentistry in the British Commonwealth

By George. E. Camilleri

Loosely based on the Bradlaw Oration given at the Royal College of Surgeons of England in October 2011.

The Bradlaw Oration was instituted in 1974 by the Faculty of Dental Surgery of the Royal College of Surgeons of England to honour Professor Sir Robert Bradlaw, the first Dean of the Faculty.

Sir Robert Bradlaw was Dean at the Sutherland Dental School in Newcastle, Dean at the Eastman Dental Institute, founding dean of the Faculty of Dental Surgery, Royal College of Surgeons of England, and President of the General Dental Council and later of the British Dental Association. He guided, sometime cajoled, the dental profession in the United Kingdom forward. In this article I shall try to accentuate his role in Dentistry in the British Commonwealth with special reference to Malta.

BRADLAW AND BRITISH COMMONWEALTH

Bradlaw was an influential ambassador of British Dentistry and played a crucial role in the development of dentistry in many countries in the British Commonwealth.

His key position at this Royal College of Surgeons allowed him the possibility to spread its influence in promoting specialist and postgraduate training among dental graduates from the Commonwealth. A delegation from the College under Bradlaw and Dr. Senior visited India in 1949 conveying the Honorary FDS to leading Indian dental surgeons. In 1950, he flew to Singapore to confer the Fellowship in Dental Surgery of this college to C. F. Mummary Director of Dental Services in the Federation of the Strait Settlements. In 1958 he again went to India to conduct the first FDS examinations there.

Bradlaw actively participated in many educational missions to Commonwealth countries. He was a member of a UK Mission to New Zealand in 1951 and in 1971 traveled to Burma to help in

drawing up an WHO Assignment report on dental diseases, dental health services and dental education. This is by no means an exhaustive list of his role in Commonwealth dental affairs.

His tremendous work and influence on dentistry in the Commonwealth is attested by the myriad of honorary degrees, and honorary membership of Dental Associations bestowed on him. Doctorates from Melbourne, Australia and Montreal (Canada) together with honorary membership of the Dental Associations of the South African, Indian, Pakistani and Maltese Dental Associations were a small way of acknowledging his generous endeavours.

Apart from his many travels to Commonwealth countries on dental affairs he did not neglect the Commonwealth students on Postgraduate studies in the UK. Behind the apparent gruff and dominating appearance there was a heart of gold. Both as President of the GDC and especially as Dean at the Eastman Dental Institute he had the interest of Commonwealth students on postgraduate studies in the UK very much at heart. Besides fostering their experience as a group there are innumerable instances where he played a guiding and supporting role on an individual basis. Apart from students at the Eastman, Bradlaw must have kept a tab on all dental students at the Nuffield College of the Royal College. John Portelli, whilst at the Nuffield following an FDS course, got summoned to the General Dental Council offices. It was just a meeting of encouragement and a means to keep up to date with the events in Malta. Bradlaw's ability to oversee the wood as a whole and still look after the individual trees was one of strengths. I shall dwell mostly on Bradlaw's Malta connection. It can



serve as a model to highlight Bradlaw's commitment to the smaller colonies.

MALTA

The dental course in the University of Malta was started in 1933 with a new student intake once every three years. Uniquely, the General Medical Council of the United Kingdom recognised Malta's Diploma in Dental surgery as one entitling its holders to register without examination in 1936, a year before the first course had even graduated in 1937. Bradlaw was not a member of the Dental Board then so that we cannot attribute this signal event to him. An important duty of the Dental Board of the Medical Council was to assure the standards of the dental schools by regular official visitations. Once the Malta degree was registrable a visitation was in order. In 1950 the General Medical Council sent Bradlaw to Malta as an official visitor to report on the Dental School of the now Royal University of Malta. Malta was still recovering from the devastation of the Second World War and Bradlaw with his humanity and insight decided that the course was sufficient but added that "It is to be hoped that finance will be available to improve its equipment, including such necessary items as dental units or electric engines". At a dinner given by the Dental Association Bradlaw was presented a beautiful Maltese Silver Boat which he later presented to the Royal College (Fig 1). He was assured that a new dental clinic was being built although when I started my dental studies in 1953, I still had to drill my first cavities using a foot dental engine.

Continues on page 23

MYOFASCIAL PAIN SYNDROME

Continues from page 11

Retinal receptors detecting light enable the SCN to mediate the orderly execution of physiological events, such as release of hormones in a specific pattern.

Individual attacks of CH appear to follow circadian periodicity and this triggered research to try and determine if there was any possible relationship – abnormal serotonin release seems to provide a plausible explanation. One of the several connections of the SCN involves projection fibres to and from the periaqueductal gray matter – and hence it can be thought as being functionally linked with the central pain-modulating pathway.

Evidence shows that bouts of CH and migraine are linked with insufficient serotonergic neurotransmission. Also, serotonin is a vasoactive substance, mediating vasoconstriction.

Serotonin receptors have been shown to be present within the cranial and meningeal blood vessels; therefore a reduction of this mediator provides a possible explanation to the observed vasodilation that is then sustained by activation of the trigemino-vascular bundle, mentioned next. Vascular changes, which as said

are brought about secondarily to aberrant firing by central projections, activates trigeminal nociceptive fibres with subsequent antidromic release of the vasoactive neuropeptides; Calcitonin Gene Related Peptide and Substance P.

These induce an inflammatory response, (sterile inflammation as there is no infectious agent), thereby sensitising and recruiting other nociceptive fibres promoting further vascular changes and propagation of pain along trigeminal afferents. These fibres terminate in the spinal trigeminal nucleus, with projection to the thalamus as described in Chapter 2.

The autonomic phenomena that characterise cluster headache include ipsilateral, (with respect to pain localisation), lacrimation and salivation and are thought to be mediated by central connection in the brainstem, linking projections from the hypothalamus and spinal trigeminal nucleus with the superior salivatory and lacrimal nuclei.

Projections from the former constitute pre-ganglionic parasympathetic fibres that initially join the facial nerve and eventually leave the latter in the petrous temporal bone as the chorda tympani and join the lingual nerve (of

mandibular division of trigeminal) to synapse with short post ganglionic fibres within the submandibular salivary gland parenchyma providing secretomotor and vasodilator innervation within the gland.

MIGRAINE

Migraine is in some aspects, similar to CH; both involve dysregulated serotonin release that physiologically mediates vascular tone and influences the endogenous pain-modulating pathway within the PAG of the midbrain, and the exacerbation/propagation of pain via activation of the trigeminal-vascular bundle within the region innervated by the ophthalmic division of the trigeminal nerve.

Distinct to migraine though are the inherited hyperexcitability of neurons in the occipital cortex and the role of steroid hormones.

That role of the latter may explain the higher incidence of this condition in females. Apart from the yet still not clearly understood mechanisms that implicate steroid hormones in the development of the brain, oestrogen is thought to be an important determinant of the degree to which the condition is phenotypically expressed.

Continues on page 18



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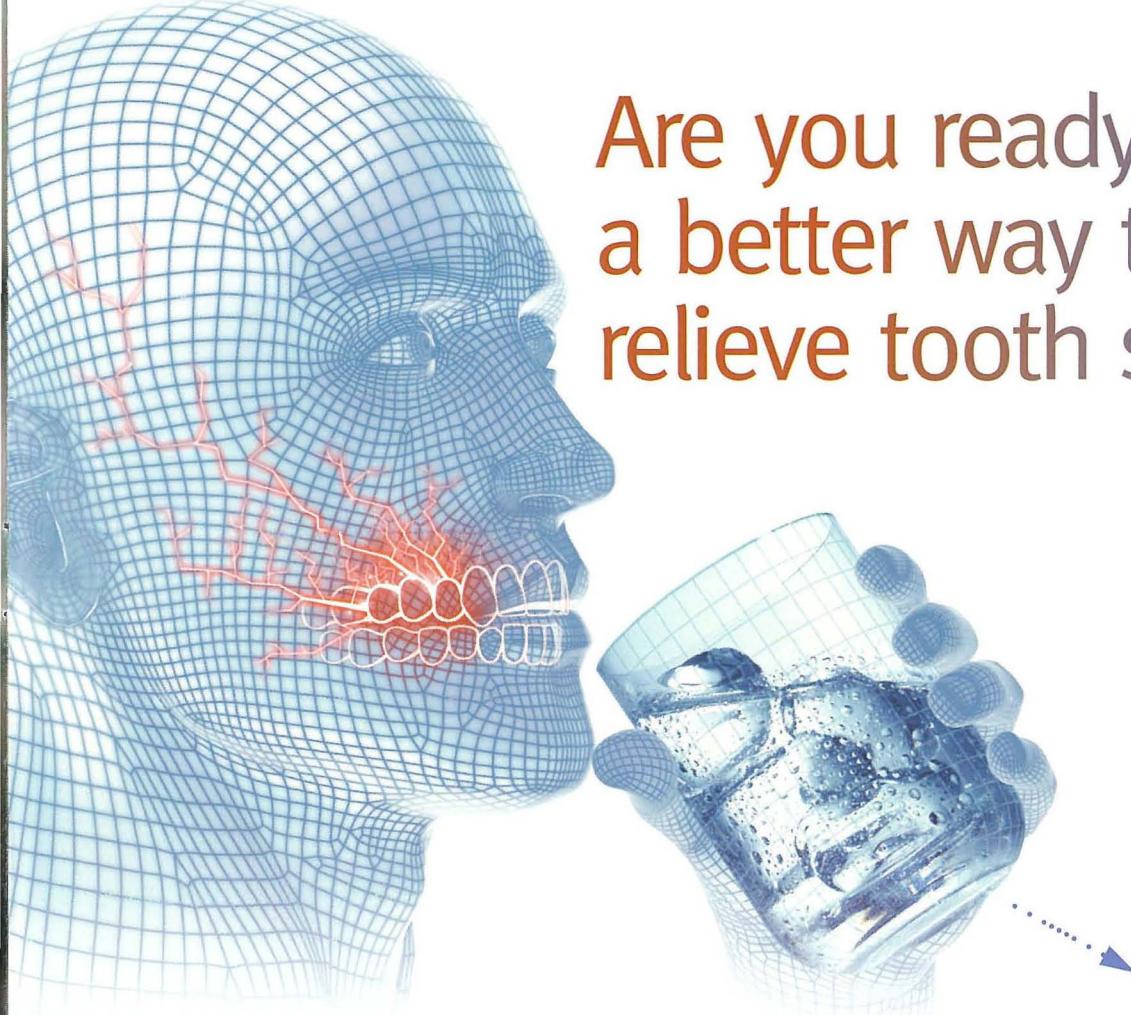
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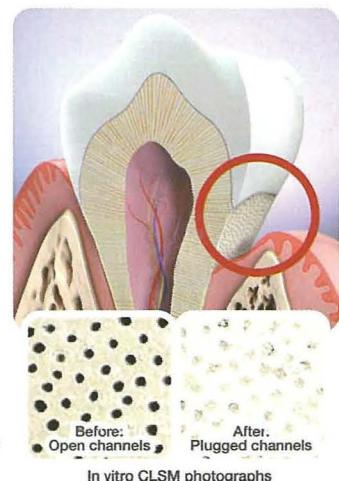
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MYOFASCIAL PAIN SYNDROME

Continues from page 16

Migraine episodes appear to coincide with the fluctuation of hormones in menstrual cycle.

Oestrogen is thought to play an influential role in mediating peptide neurotransmitter synthesis within the somas of first order neurons housed in the trigeminal 'Gasserion' ganglion. Reduced levels of oestrogen are correlated with the up-regulation of the gene coding for Neuropeptide Y (NPY). CGRP levels are not altered and hence it is this change in NPY which is thought to mediate the initial vascular phenomena.

The concept of Spreading Cortical Depression (SCD) describes the phenomena disclosed by ElectroCorticoGramms (ECOG), whereby a brief period of cortical neuron hyperexcitability is followed by a prolonged period of reduced excitation with a concomitant increase in the vascular supply.

Therefore an event triggered in the cerebral cortex, which like the pia and arachnoid, has no pain sensation, triggers a delayed inflammatory response in the dura mater, which is in turn supplied with nociceptive afferent fibres, supplied by branches of the trigeminal nerve and from the first two cervical nerves on the posterior aspect. Nitric oxide makes blood capillaries more leaky allowing for plasma protein extravasation.

Other chemical mediators involved are cytokines such as interleukins. As already stated, this initial event triggers activation of the trigemino-vascular bundle that generates further pain via a self-sustained cycle.

The transient visual disturbances that precede the actual onset of headache are believed to be brought about by localized temporary reductions in blood flow, although these reductions are not of a magnitude that can precipitate an ischaemic attack. These

prodromal features are referred to as aura, and can manifest in a variety of ways; ranging from the more common transient visual field defects, scotoma or anopsia, to the appearance of flashing lights, and less common events like ipsilateral numbness or weakness of the face.

THE PATHOPHYSIOLOGY OF MYOFASCIAL PAIN

The underlying mechanisms implicated in the pathogenesis of myofascial pain are discussed here. Some aspects are still not fully elucidated, nor there is general consensus.

- Local Twitch Response (LTR) and Palpable taut bands: - contraction of intrafusal muscle fibres "sympathetic spindle-spasm theory" and extrafusal muscle fibres "energy crisis and motor-end plate hypotheses; enriched Calcium Ca²⁺ ion intracellular storage and its subsequent release from the sarcoplasmic reticulum
- Pain: - mediated by deep somatic and sympathetic afferent nerve fibres
- Tenderness: - peripheral and central sensitisation mechanisms
- Referred pain: - via a centrally-mediated mechanism

Initially, development of trigger points was attributed to the events that followed trauma to muscle tissue. Basically, inflammation and healing by scar tissue were thought to be the underlying cause for the clinically palpable bands within muscles, but this was eventually negated because tissue biopsy from the affected muscle showed no histological evidence of fibrous connective tissue.

ENERGY CRISIS AND MOTOR END-PLATE DYSFUNCTION HYPOTHESES

Local Twitch Response (LTR) is one of the features that characterizes myofascial trigger points (MTrPs). Studies by Simon and Travell,

two of the greatest researchers on myofascial pain had proposed two possible theories that could explain the clinical manifestations of palpable taut bands and twitch response in patients suffering from MPS.

These two theories, as was later proposed by the authors themselves, should not be considered as distinct phenomena but rather should be envisaged as complementing one another.

These two theories are the Energy-crisis theory and the Motor end-plate dysfunction hypothesis, (described next), and also related to the way in which they could possibly give rise to MTrPs.

The motor end plate hypothesis proposes that the cascade of events following reduced oxygen supply and diminished ATP production are responsible for the sustained contraction of affected muscle fibres hence the clinical finding of taut bands, and also elevated levels of by-products of metabolism as a result of hypoperfusion are implicated in the stimulation and sensitisation of nociceptive fibres that give rise to pain and tenderness.

From a pathophysiological point of view, muscle pathology can be triggered by a number of plausible aetiological factors, like bad posture that increases neural input leading to repetitive microtrauma for example. Sustained neural input, a phenomenon referred to Spontaneous Electrical Activity (SEA), leads to a relative increase in the time spent by muscle fibres in a depolarized state with respect to the resting state.

Longer periods of sarcolemmal depolarization promote an increase in the calcium ion, Ca²⁺ influx, thereby enriching the intracellular stores of sequestered Ca²⁺, like the sarcoplasmic reticulum. Prolonged depolarisation times mean prolonged shortening of muscle fibres thereby leading to a reduction

in the regional blood supply by physically compressing the arteries.

This relative ischaemia due to hypoperfusion eventually leads to a decrease in the ATP production and accumulation of by-products of metabolism. ATP is required in two fundamental steps in sarcomere function; for the association of myosin heads with actin filaments and also for the dissociation of myosin from the active binding site on the actin. Therefore energy crisis hampers the physiological functioning of muscle fibres which enter a vicious cycle, further promoting and sustaining sarcomere shortening hence muscle contracture

The myoneuronal junction aspect of the theory postulates the aberrant release of acetylcholine transmitter at the synaptic cleft that generates post-synaptic currents ultimately giving rise to post-synaptic potentials (PSPs). Acetylcholine, activates voltage-sensitive cation selective channels, with a reversal potential close to 0mV (as determined by the integrated Nernst equilibrium potential values for sodium and potassium – Goldman's equation.

This means that Ach will bring sarcolemmal membrane closer to 0mV; hence PSPs generated at motor end plate by Ach are excitatory post-synaptic potentials EPSPs.

The latter are not of a magnitude that brings about contraction of the whole muscle, but rather action potentials are propagated along short distances along the sarcolemma of fibres supplied by that particular dysfunctional nerve, keeping in mind that extrafusal motor nerves give rise to a number of branches within the connective tissue of the perimysium with each branch responsible for the innervation of a number of muscle fibres.

The exact number of junctional units per branch varies from muscle to muscle depending on how precise

the movement generated by the action of the muscle in question is desired to be. As can be seen, these two hypotheses, do provide an explanation for the observed clinical signs and symptoms of MPS, although recent and current research is pointing to another hypothesis – the "sympathetic spindle spasm"

Studies conducted by Hubbard and his colleagues, hypothesize that the location of MTrPs lies within MSs, with preliminary EMG studies that contradict results of previous studies carried out by other researchers including those published by Simons et al, and which were believed to provide a definite answer to what was then regarded an unresolved mystery.

Dr. Hubbard at the University of San Diego California, is also another researcher who has published many papers on muscle pain and holds patents for the use of sympathetic blockers to treat trigger points. These latest studies report that SEA events do not occur at dysfunctional motor end plates but they are generated within the fibres of muscle spindle (MS). A clinical research conducted by this group of researchers was completed by the end of last year.

So the most important remarkable difference of pathophysiological and pharmacological importance, is the difference in what generates these SEA and contractions – with a distinct myoneuronal couple for each theory; consisting of adrenergic post-ganglionic sympathetic terminals–intrafusal muscle fibres in the latest hypothesis, rather than the alpha-motor neurons–extrafusal muscle fibres postulated by the other theories. This new hypothesis thus became known as – the "sympathetic spindle spasm theory".

NEUROPHYSIOLOGY OF THE MUSCLE SPINDLE

A number of authorities in neuropathophysiology believe that central to the clinical phenomena of chronicity that characterises MP,

is the disrupted function of the neuromuscular spindles (NMSs). These are structures that are physiologically designed to convey proprioceptive signals to the CNS.

Muscle spindles are small, (~1mm wide and up to 6mm in length) encapsulated structures, housing specialized muscle fibres – intrafusal muscle fibres – that receive both sensory (muscle spindle afferents – MSA) and motor innervation – fusimotor gamma motor neurons.

The fibrous connective tissue capsule housing NMSs is the same fascia that surrounds muscle, continuous with tendons and bone periosteum etc. Therefore fascia is a 3-Dimensional continuum surrounding and 'gelling' up together muscle, bone and nerves giving rise to definite neuromusculoskeletal functional units in discrete compartments.

Increase in muscle fibre length, as occurs secondarily to passive stretch during clinical examination of tendon reflexes, opens stretch-sensitive ion channels in the plasma membrane of the MSA, the annulospiral and flower spray endings (Type IA and II fibres respectively).

Opening of the channels allows conduction to ionic fluxes giving rise to an inward current that generates a receptor potential; if summation of such positive electronic waves brings the membrane potential to threshold potential, a nerve impulse will fire off.

Impulses are conveyed centrally via the dorsal horn of the spinal cord as previously described and synapses directly via a mononeuronal/monosynaptic pathway with alpha motor neurons supplying the extrafusal muscle fibres that will restore muscle length. This "unloads" MSA discharge as muscle length is restored and ion channels deactivated.

Continues on page 29

RADIOLOGY IN EGYPTOLOGY

Dr Charles Corney, MB BS, DMRD, FRCR
Diagnostic Radiologist

ABSTRACT

The value of radiology [illustrated from a personal collection of X-ray films of five mummies] and other techniques is discussed in the examination of an Egyptian mummy.

The ancient Egyptians, from about 3000 BC—300AD, mummified their affluent dead--particularly the pharaohs because they believed it helped the soul to find its body in the after life. They believed that preserving the body permitted a journey into another world to start a new life which lasted for ever. The word 'mummy' came from a Persian term for an embalming substance.

All the organs were removed, except for the heart which was believed to be the centre of emotion and intelligence necessary for the next world. Often it took two months for the body to dry out after the application of natural salt. The empty body cavities were filled with sawdust or linen sheets. Then other linen sheets or shrouds were wound around the body. To examine the body, these had to be unwound to reveal the details of the body—often causing damage.

The advent of X-rays opened up a new vista [mainly the bones and the teeth] of the body, without the necessity of this unwinding. Later, more sophisticated X-ray equipment, CT Scanning, was used to produce 3-D reconstruction images of various body parts—particularly the face.

Carbon Dating was useful to assess the age of the body, and DNA Determination helped in the quest for identifying

genetic damage, for example, suggesting malaria and sickle cell disease as the causes of the death of Tutankhamun in 1323BC. Occasionally the organs were not removed, permitting Endoscopy of the gastro intestinal tract and Arteriography of the arteries.

WHAT HAVE THESE INVESTIGATIONS SHOWN?

- **X-ray R1** shows osteoarthritis of both shoulder joints indicating old age. Also two metal bird-like structures are seen lying over the chest and neck. These emblems were attached to the mummy to speed the soul to the after life. The heart is present but the thorax is filled with linen/sawdust giving a mottled white appearance.
- **X-ray R2** shows a mismatch of the skull with the false external 'face'—suggesting that an adult appearance was desired, because the body was that of a child. The presence of multiple well preserved teeth confirms that the mummy was indeed a child. Another interesting feature is the presence of a false hand lying partly over the chest. This false hand is perfect in detail, including fingernails, but is not of bony construction. Most likely it is made of papier mache.
- **X-ray R3** shows edentulous jaws apart from the presence of impacted wisdom teeth with adjacent periodontitis—indicating an older mummy. Strangely however, loose well formed teeth [probably belonging to a person who was younger] have been flung around the outside of the skull before closure with the shrouds. Most

likely, these teeth enhanced the ability to eat in the next world!

- **X-ray R4** shows that both feet were cut off very neatly at the ankle joints and placed in another position. There was insufficient length available to accommodate the body plus the feet. Maybe there was only a child's size wooden coffin or sarcophagus available in which to insert the mummy, or there was some error of measurement seemed likely. Anyway, how was anybody going to discover such a mistake at that time?
- **X-ray R5** shows extensive arthritis of the joints of the foot. Once again a metal 'bird' has been placed on the diseased structure to prevent delay in arriving in the next world.

OTHER RADIOLOGICAL FINDINGS

1. Fractures, dislocations and osteoarthritis—often due to falling off a chariot, or the chariot turning upside down, equivalent to the present day road traffic accident.
2. Underdeveloped limb bones—suggesting poliomyelitis.
3. Missing hands/feet/digits—probably due to an accident or to a developmental defect.
4. Such loss, as in 3 had to be replaced by a realistic-looking prosthesis as the body's journey to the next world could otherwise be delayed.
5. Developmental bone disease, such as the 'fused' cervical spine of Tutankhamun.
6. Sclerotic areas in the pelvic bones—typical of bony spread from prostatic cancer.
7. Infected or absent teeth was common because the wheat used

in the bread was contaminated with sand which gradually ground down the teeth causing caries and eventual loss. If many teeth were present, then the subject was less than 20 years of age, but if all the teeth had disappeared then the subject was older.

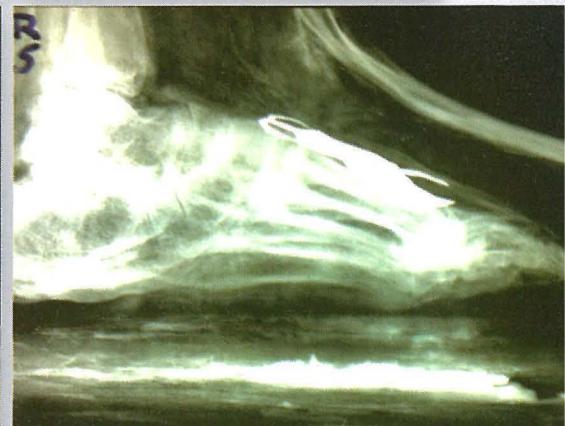
8. Age determination—by assessing the maturity [bone age] of the wrist bones
9. Gender determination—by measuring the sub pubic angle. If less than 90 degrees, subject was male. If more, subject was female.
10. Parity determination— separation of the pubic symphysis in a female suggested previous birth of a baby.
11. When the organs had not been removed, arteriograms were performed, often revealing, rather surprisingly, severe atherosclerosis at a young age.

CONCLUSION

The undoubted success of mummification in preserving the bodies for 3000 or more years has permitted radiology and other techniques to reveal the kind of life and health these subjects led.

Subjects married when they were about 12 years old, had children and died often before 30, due commonly to pestilence, premature cardiovascular disease and bone disease, trauma from chariot accidents.

Rather unexpectedly, many subjects suffered ill health from severe dental caries. Such short lives were also due to genetic defects enhanced by close consanguineous unions – a practice which still exists today in the Middle East. ■



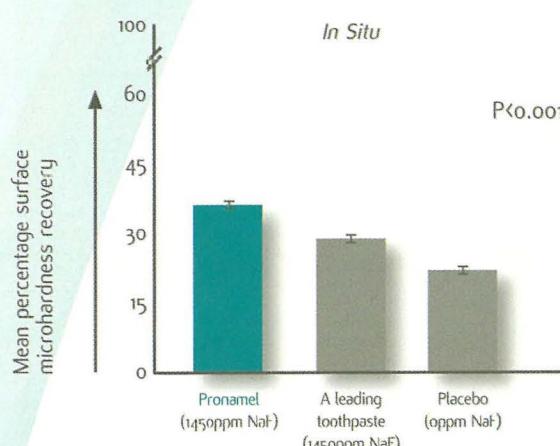
For extra protection against acid wear...

Modern eating and drinking habits increase the exposure of tooth enamel to dietary acid that can lead to acid wear (erosive tooth wear), the biggest contributor to tooth wear.¹⁻⁵

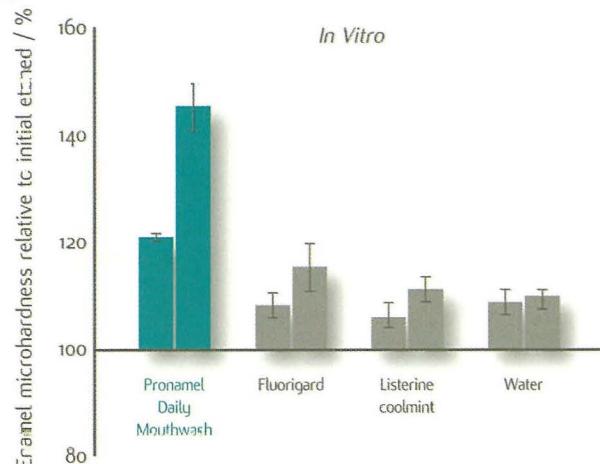
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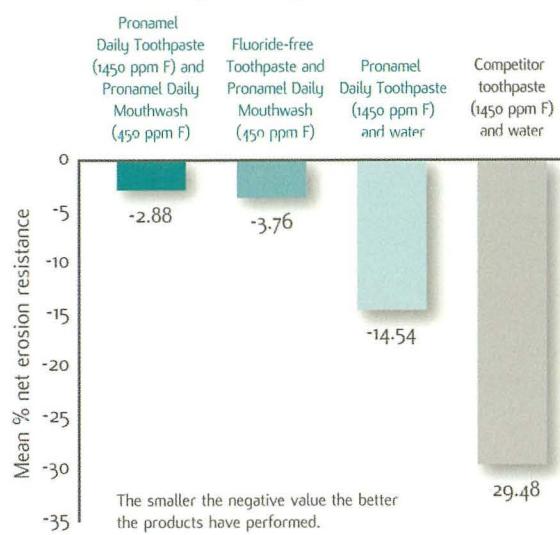


Adapted from Hara AT *et al.* Bovine enamel specimens were subjected to an erosive challenge. This was followed by fixation to palatal appliances and a 4-hour intra-oral phase in 58 human subjects.



Adapted from Young M and Willson R. 6 human enamel specimens were subjected to an erosive challenge *in vitro*. This was followed by a mean rehardening microindentation study after treatment with fluoride mouthwashes.

But used in combination, provide 80% more protection against acid wear than brushing with *Pronamel Daily Toothpaste* alone^{9*}



Adapted from Maggio *et al* 2010. Original study design contained 5 test cells; the one not included here is a fluoride-free dentifrice plus water.



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References: 1. Lussi A. Erosive Tooth Wear – a Multifactorial Condition. In: Lussi A, editor. *Dental Erosion – from Diagnosis to Therapy*. Karger, Basel, 2006. 2. Lussi A. *Eur J Oral Sci* 1996;104:191–198. 3. Bartlett DW *et al*. *Int Dent J* 2005;55:277–284. 4. Zero DT. *Int Dent J* 2005;55:285–290. 5. Zero DT *et al*. *J Clin Dent* 2006;17 (Spec Iss):112–116. 6. Deery C *et al*. *Pediatr Dent* 2000;22(6):505–510. 7. Hara AT *et al*. *Caries Research* 2009;43:57–63. 8. Young M and Willson R. GSK data on file. 2008. 9. Maggio B *et al*. *J Dent* 2010;38(5):537–544. Prepared: February 2011 Z-11-037

Bradlaw and Dentistry in the British Commonwealth

Continues from page 15

Fortunately we soon moved to a better equipped Dental Clinic. The automatic registration in the UK of the Malta degree, which Bradlaw helped so much to safeguard, was of inestimable value. It meant that we could come for postgraduate studies or work in UK dental hospitals and schools, without the need of further examinations.

The relationship of Britain and the Commonwealth countries has been evolving with undoubtedly loosening of the ties. In 1977 due to Maltese political problems the new Maltese degree was no longer recognised for automatic registration by the GDC which made it a bit more difficult for our graduates who wanted to work or study in Britain. Great Britain had already joined the European Community in 1973 and the registration problem was resolved when Malta became a member of the European Union in 2004. It was not our membership of the Commonwealth but that of the EU which gave us back our lifeline from which we still benefit greatly.

PERSONAL CONNECTION

My first contact with Bradlaw was in Newcastle in 1956. I attended the Congress of the International Association of Dental Students in the Newcastle Dental School where Bradlaw was still Dean and host to the Conference. I still treasure a signed photograph of him standing before his beloved Sutherland Dental School (Fig. 2).

The next meeting was not so relaxing when he was an examiner at my 1960 Edinburgh Fellowship examination. By that time he had already established close personal relationships with Lapira and Mangion of the Malta school and my viva consisted mostly of enquiries of his Maltese friends much to the dismay of the other examiners.

In 1965 the Royal University of Malta awarded the Honorary D.Sc. to the newly knighted Sir Robert (Fig 3).

I had then just returned to Malta and, very much in a junior capacity, participated in the ceremonies. The oration was given in Latin when we were told that "nulla unquam mater liberos sibi habuit curae ut candidatus noster



dentariam huius universitatis scholam fovit, iuvit, optulatus est", another way of saying "No mother ever cared for her child as did Professor Bradlaw for our Dental Faculty." He did visit Malta informally on short private visits and at one time was considering whether he should set up house in Malta. On a few occasions I was the official minder which, of course, was an opportunity for personal contact, although squeezing him into my Mini Minor was a problem. His sense of protocol, and politics, was of course paramount. I remember when direct from the airport he asked to be driven to the Governor's Palace in Valletta where he signed the visitor's book as President of the General Dental Council. Naturally he was later invited to dinner at the Palace.

He had a reputation for orchid growing and my inability to recognise, much less know the scientific name of the wild orchids we saw in country walks did not pass without comment. It was during a meal that as an aside he asked me my chief's birthday which I promptly supplied. With an incipient self-congratulatory smile he informed

me that he had never failed to get a prompt answer to that question from a contender. In 1969, when I was attending a World Health Organization Course at the London Hospital, run by another former dean of the Faculty, Geoffrey Slack. We were on an official visit to the General Dental Council when he took me aside inquiring of his Malta friends and sending his regards. My final encounter was in 1984 when he was a guest speaker at a Symposium given by the Faculty in honour of Professor Bert Cohen, held in the self same room where the Oration was held.

GUY'S AND RCS

Bradlaw was a Guy's graduate, had a close and influential relationship with this College and played a critical role in aiding the development of the Malta dental school. It is a remarkable coincidence that a century ago, three dental surgeons with the same common factors, Guy's, the Royal College of Surgeons of England and Malta figure prominently in the early history of Maltese Dentistry.

Continues on page 25



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to promote inter-profession relationships and to contribute towards the advancement of the interests of society.

The Association is heavily involved in the organisation of post-graduate lectures at University and also puts together events for its members.

President Dr Adam Bartolo expressed his satisfaction with the partnership, saying Vodafone's communications package will aid members in keeping in touch with patients and colleagues.

This collaboration will also mean members will have access to data and medical software applications

on their smartphones, and to the latest literature about patient care.

"We are very pleased to have reached this agreement with the Dental Association of Malta since it is in line with our current strategy to continuously build and improve our relationships with professional associations by offering tailor-made benefits to suit their needs," said Business Marketing Manager Daniel Grech.

Vodafone Malta's network of strategic alliances aims to support business and professional associations within the community. ■

Bradlaw and Dentistry in the British Commonwealth

Continues from page 23

A.B.Wills Rust. (1879-1959) studied at Guy's and became an LDS of this College in 1902.

In 1915 he was appointed Temporary Lieutenant for service abroad in the RAMC and was the first Army dental surgeon to serve in Malta. When, in 1918, the University of Malta was drawing up the examining board for the first examination to be held for the Diploma in Dental Surgery, he was appointed on it together with the Professors of Surgery and Medicine.

At that time, i.e. World War I, a number of young Maltese volunteers in the St. John's Ambulance Brigade, were helping out in the Military Medical Station. Rust was impressed by a young Maltese, Egidio Lapira, and suggested that he should article himself as an apprentice in a practice run by Bannar Martin. Thus Rust, besides being an examiner of Malta's first dental graduate was also the instigator for Lapira to take up dentistry.

Harry Bannar Martin (b 1880) also studied at Guy's Hospital and passed the LDS examination in 1905. He was appointed Demonstrator in the Conservation Room at Guy's in 1903 and Dental House Surgeon a year later.

In 1905 he served as Civilian Dental Surgeon at HMS Haslar, Portsmouth and in 1909 he acted as Instructor in Dental Surgery to the Junior Medical Officers.

He came to Malta as a private practitioner in 1910 and in 1915 was nominated civilian dentist to the Malta Military Command. Lapira, on Rust's recommendation, worked in his practice up to 1922. The University accepted Bannar Martin as a recognized teacher on the strength of his earlier stint at HMS Haslar and Lapira was allowed to sit for the examination and obtain the Diploma in Dental Surgery in 1920, the first Maltese dental graduate.

Lapira, a man in the Bradlaw mould, started the first dental course in 1933 and became Dean of the Faculty of Dental Surgery at the University of Malta in 1954. He is rightly considered the father of Maltese Dentistry. In his unpublished biography Lapira recounts his visit to Cambridge for the BDA Meeting in 1937 when he records "the pleasant memory of having met for the first time, Mr. Robert Bradlaw, then a young man, who eventually became, and notwithstanding that he scaled all the ladder, still is, one of my best ever friends, and who by his experience and sound advice, and unending help, has been my guiding

light in promoting the evolution of the Dental School of the Royal University of Malta". Lapira was awarded the Honorary FDS of the College in 1949, no doubt we can see Bradlaw's guiding hand. On that occasion Lapira presented to this Faculty a Silver Salver (Fig 4), which had been given to him by the students of the First Course. The third gentleman with these three common attributes, Guy's, the College and Malta, is John Eskdale Fishburn.

He was a conjoint student at Guy's up to 1917 when he joined the Royal Navy in the rank of Acting Probationary Junior Sub-Lieutenant as a ship's surgeon serving in the Mediterranean. Whilst at Guy's he was secretary of the Guy's Hospital Dental Society. He somehow got to know of the possibility of sitting for the Diploma in Dental Surgery of Malta and successfully applied to sit for it. He had the final examination with Rust as one of the examiners on November 11th 1918, Armistice Day. He is the first dental graduate of the University of Malta. He never practised dentistry, became a very successful businessman and used to commute between his New York and London offices weekly on the Queen Mary.

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Prof. George Camilleri appointed to The National Order of Merit

On Republic Day each year, the President, on behalf of the Government and people of Malta, pays public tribute to a number of Maltese citizens who distinguish themselves in different fields of endeavour. This year Prof George Camilleri was appointed to The National Order of Merit in view of his dedication and work in the field of Dentistry.

This year's Investiture Ceremony again took place today at The Palace, Valletta immediately following the customary Ceremonial Parade held at the Palace Square. During the Ceremony, one companion, one officer, one

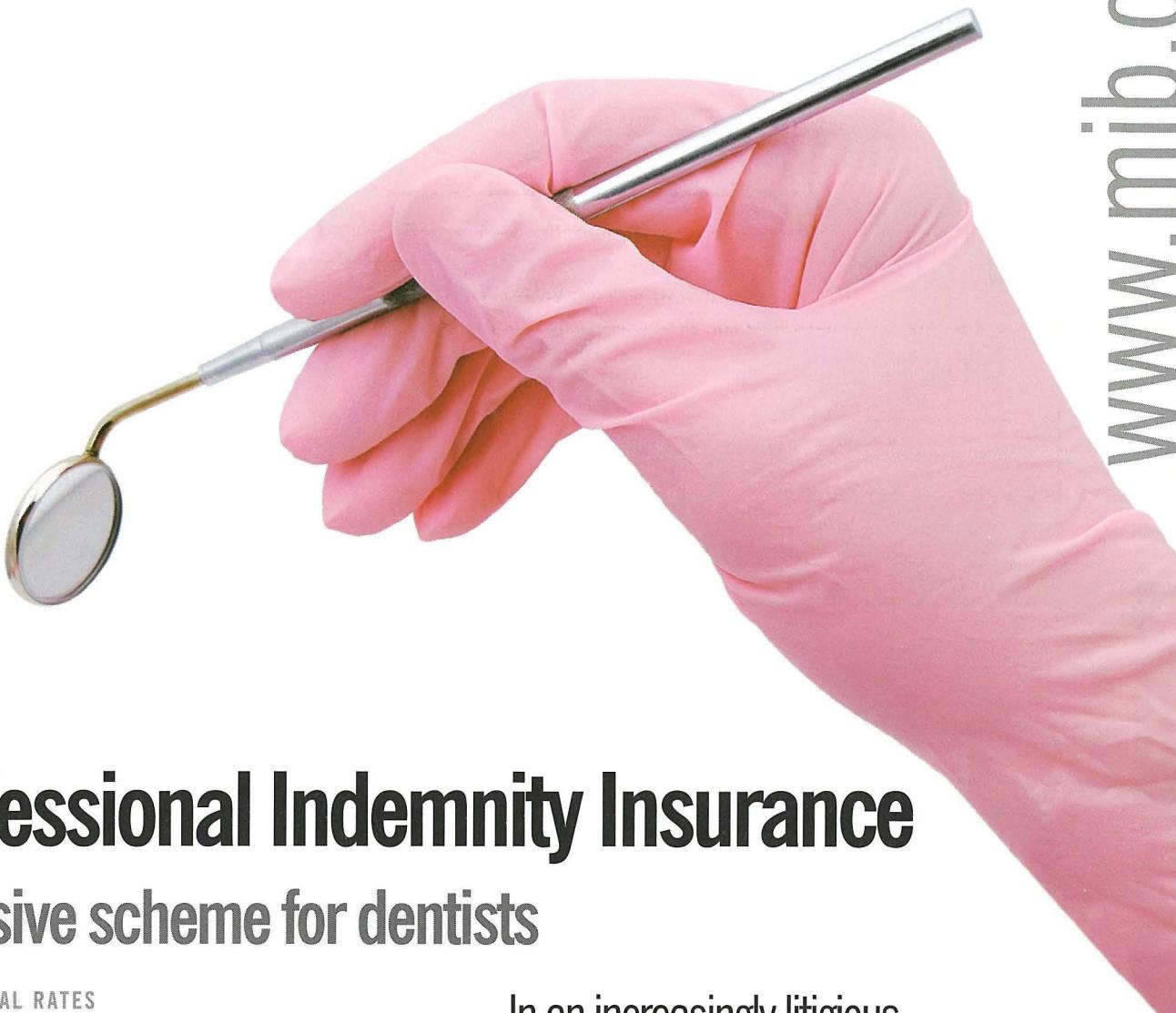


honorary member and nine members were appointed to The National Order of Merit and seven individuals were

awarded the Midalja ghall-Qadi tar-Repubblika. Dr Mercieca and Professor George Zarb have also received this award in the past. ■



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MYOFASCIAL PAIN SYNDROME

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However, apart from this sensorimotor innervation, muscle spindles also receive branches from the sympathetic nervous system.

This discovery triggered new research to determine in the most accurate manner possible, the role of autonomic nervous system (ANS) influence on muscle spindle function, and the literature reports the following consequences:

- it couples the ANS and somatosensory motor system at a receptor level, this is exemplified by the fight-or-flight response; a physiological response involving a number of systems including the musculoskeletal system, that work in synergy to enable an individual face a danger situation.

Sympathetic stimulation will contribute here by temporarily inhibiting and depressing the firing rate from MSAs thereby momentarily 'loosing' the tight control over the stretch reflex. This will minimise unwanted oscillation and hence promote greater stability on performing rapid movements (such as running) as in such a situation stability should prevail over the control of highly elaborate and precise actions - like the lumbrical muscles of the digits and the precise determination of the force required for jaw closure during mastication are not required and can be temporarily omitted in favour of greater stability and reliability of muscles involved in running for example.

- ANS can modulate such functions as motor reflexes, proprioception and coordination, previously attributed solely to afferent and fusimotor neurons of MSs.
- also, given that sympathetic afferents are pain sensitive,

the presence of nociceptors in MS gives a new perspective to the mechanisms underlying abnormal sympathetic discharge, providing an explanation or at least helping to give an insight to describe phenomena of NMS dysfunction associated with conditions of increased sympathetic activity such as stress.

The post-ganglionic sympathetic terminals are noradrenergic and relatively recent studies (using antibodies and immunofluorescence to disclose the presence of a target molecule) also showed the presence of Neuropeptide Y (NPY) being liberated by these terminals as a co-transmitter.

There is no dispute amongst neuroscientists with respect to the presence of sympathetics at the MS and also with respect to the chemical substances liberated at the synaptic transmission.

However, when it came to determine the actual neuronal connections involved in ANS modulation of MS function, there was no general consensus amongst the various researchers; an indirect control mechanism brought about by changes in vascular tone, namely vasoconstriction, was initially implicated.

At present, this initially postulated mechanism of MSA modulation secondary to vasomotor changes is not only regarded as erratic but it is now known that actually changes in blood flow are independent of MSA effects and a number of studies were designed to clarify this matter. In one study it was concluded that, induced muscle hypoxia did not alter the magnitude or time course of the depressant action of MSA modulation by ANS.

Even though intense research and studies were carried out worldwide in the past two decades, there is still some debate with respect to what

constitutes the synaptic targets of the sympathetic fibres, although results of studies indicate that the most likely candidates for this post-synaptic role are the sensory endings of the Ia and II afferent neurons.

Sympathetic excitation results in reduced afferent discharge by all Ia fibres and by ~50% of II fibres and increased afferent discharge in the rest of the II fibres. Intrafusal muscle fibres are also thought to be directly innervated by sympathetic fibres, at sites other than those regions where gamma-motor fibres form junctional complexes with spindle fibres.

Also, Sennano et al, correlated the clinical phenomena observed in MPS patients with results of studies carried out on animal models and concluded that although mechanisms are unclear, the pathophysiological process underlying MPS seems to involve, at least in part, disinhibition and/or dysfunction of the gamma-motor neuron circuitry, that would explain the spontaneous muscle spasms and also the more pronounced tendon reflexes.

MSA input is involved in spinal and supraspinal reflexes, and proprioceptive input, (sometimes referred to only kinaesthetic – which basically refers to purely proprioceptive input from the musculoskeletal system excluding the sense of equilibrium to emphasize that here one is dealing with muscular input), integrated to execute complex functions, such as execution of skilled motor movements involving not only perception of movement but with auditory and visual input.

This is a clear example of the concept of hierarchy within the (mammalian) human nervous system, where higher brain centres can control and exert influence on more than one system at the same time, and this integration at a central level is expressed as integration of such systems involved.

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MYOFASCIAL PAIN SYNDROME

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Sympathetic outflow attenuates/dampens the feedback control of muscle length and tone. In the previous paragraphs it was stated that muscles employed in motor actions requiring a particularly high degree of precise action, such as muscles of the fingers and muscle of mastication, have a relatively higher density of such spindles.

Unlike the biceps muscle of the upper limb for example, which performs a rather 'crude' action resulting in flexion at the elbow, execution of tasks requiring a certain degree of precision like endodontic filing of the second mesiobuccal canal (MB2) of an upper first molar or eating a meal characterized by hard and soft foodstuff, whereby the force of the bite is adjusted accordingly, demands a system providing continuous input (MSA) that modulates output. This physiological harmony between sensory (proprioceptive) and motor systems is particularly evidenced in edentulous patients wearing complete F/F dentures, by difficulty experienced during mastication.

Apart from the postganglionic fibres, sympathetic nerves supplying MSs also contain afferent fibres that convey nociceptive sensation centrally. The presence of such pain receptors in muscle spindles, together with other nociceptive fibres located in muscle tissue, facia and tendons, and the process of central sensitisation, account for the exquisite tenderness elicited by MTrPs palpation. To make the situation more complex, pain may elicit a reflex spasmotic contraction of muscle via excitation of alpha-motor neurons, thereby further exacerbating the effects of contracture – the 'Pain-Spasm-Pain' cycle phenomenon.

ROLE OF THE CENTRAL NERVOUS SYSTEM IN MYOFASCIAL PAIN

POSSIBLE MECHANISMS MEDIATING CENTRAL SENSITISATION

Allodynia and hyperalgesia are two diagnostic symptoms characteristic of MTrPs. The former refers to when

a patient perceives certain normally innocuous stimuli as painful and the latter refers to the exaggerated painful response to normally noxious stimuli. The central nervous system is believed to play a role in the manifestation of these symptoms, via mechanisms similar to those underlying other chronic neuropathic pains. This gives MPS a new dimension in addition to the peripheral events already described mediated by receptors and sympathetic part of the ANS.

Just like the adventitia and tunica media of arteries and large veins get their oxygen supply and nutrients from a distinct arterial-capillary-venous supply and not by diffusion from the blood conveyed through their lumen, the vasa vasorum, so is the nerve sheath supplied by small nerves that convey nociceptive signals when nerve is injured, compressed or inflamed. This is in fact a common cause of chronic pain in cancer patients where tumour compression of nerves can give rise to neuropathic pain similar to non-malignant pathology.

Some authors do in fact postulate the hypothesis that some of the pain generated by TrPs could be due to nerve entrapment in taut bands. These nerves supplying nerves are called 'nervi nervorum', and as stated previously, impulses conveyed centrally can promote a reflex spasmotic contraction of the muscle, via a circuit involving interneurons integrating the sensory and motor nuclei of the trigeminal nerve, further exacerbating the contraction.

The spinal trigeminal nucleus as mentioned in chapter two is the equivalent of the dorsolateral tract of Lissauer and the first four laminae of Rexed of the dorsal horn of the spinal cord. The central connections of nociceptor fibres that convey high-intensity stimuli, are neurons in laminae more dorsal to those targeted by fibres conveying low-intensity stimuli for light touch and temperature. The latter two modalities of sensation together with pain reach the spinal trigeminal nucleus.

Following increased excitation of these nociceptor fibres as a result of trauma and subsequent peripheral sensitisation,

axons that normally convey impulses for the other modalities of sensation to this region of the spinal nucleus of the Vth, sprout out new branches that terminate in those areas transmitting high-intensity (nociceptive) signals. As a consequence of this neuronal plasticity, light touch and temperature that are usually not perceived as painful, will now be interpreted as such. This central mechanism would explain the phenomenon of allodynia, although studies carried out by Huighes et al in 2003 showed that this sprouting does not occur.

Loss of inhibition following nerve injury/inflammation is another mechanism that may mediate both allodynia and hyperalgesia, and studies on animal models in which GABAergic and glycinergic antagonists were administered, the animals hyper-reacted to noxious stimuli of an intensity identical to those applied before the administration of the antagonist and experienced pain to previously innocuous stimuli as evidenced by their behaviour.

It is as yet still undetermined whether the loss of GABAergic inhibition is the result of apoptosis selectively triggered in inhibitory internuncial neurons or whether it simply involves reduced efficiency in synaptic transmission at inhibitory synapse. Also, some studies report a reduced number of opioid binding sites, and this down-regulation of opioid receptors is in turn thought to be mediated by a mechanism triggered by peripheral nerve damage. This would imply that the physiological pain modulatory system is rendered less effective, hence the hyperalgesia.

Protein kinase C is thought to be an important mediator of chronic neuropathic pain. Some of the reported effects of this second messenger are next outlined. The voltage-gating of the NMDA (N-methyl D-Aspartate) glutamate receptors is removed, thereby enhancing Ca^{2+} influx. This on its own is a very important intracellular second messenger. Other effects promoted by protein kinase C are up-regulation of Nitric oxide synthase which via the nitric oxide (NO) generated, promotes release

of excitatory neurotransmitter from the pre-synaptic terminal. Protein kinase C is also implicated in the stimulation of sodium-ion channels, thereby favouring action potential generation.

The above mentioned events ultimately result in a shift in favour of enhanced impulse projection from dorsal horn/trigeminal nucleus tract cells to higher brain regions – called “wind-up” which by itself can only last for few minutes. Therefore this cannot explain the chronicity of pain. Chronicity is better explained by the concept of Long-Term-Potentiation (LTP). Broadly, LTP can be defined as a brief high-frequency burst of synaptic activity that leads to a sustained enhancement in synaptic efficiency. LTP is thought to be mediated by cells in the superficial/dorsal nuclear layers of the spinal trigeminal nucleus and the dorsal spinal horn expressing a type of receptor, NK1, which binds Substance P.

Such neurons constitute tract cells, whose axons project to the thalamus. Ikeda et al in 2003 reported the observed effect following co activation of NMDA receptors (as previously discussed), and of NK1 receptor, showing both enhanced synaptic strength between afferent fibres and projection neurons and enhanced firing of the latter. Of clinical and pharmacological relevance in this respect is the fact that better understanding of the opioid system, especially the μ -Opiate receptor agonists (fentanyl), which are thought to prevent LTP from setting in in-vivo, may not only alleviate chronic pain, but also possibly preventing the acute pain from evolving and progressing into a chronic one with all its debilitating effects.

TRIGGER POINT REFERRAL PATTERNS FROM OROFACIAL AND CERVICAL MUSCULATURE

MUSCLES OF MASTICATION:

Masseter:

Superficial belly of the muscle: MTrPs located in the upper third of the superficial belly refer pain mainly to the maxillary posterior teeth (pre-molars and molars). Pain may also radiate to the anterior maxillary surface and adjacent

malar process/ zygoma region. From the mid-section of the superficial belly, pain is referred to the mandibular posterior teeth and body of the mandible. Of interest is the pain referral pattern of active TrPs in the lower third region. Primary pain referral zones are in the mandibular body and the superciliary arch region and in fact, patients do complain of “pain over the eyebrow”. Less intense pain is referred also to the region adjoining the primary trigger centre with the superciliary region.

Deep belly of masseter: has a distinct pain referral pattern with respect to its superficial counterpart. The area of referral is restricted to the ear and temporomandibular region. Associated symptoms may include a degree of restricted jaw opening and tinnitus

Temporalis muscle

This muscle has a wide area of origin, occupying the whole floor of the temporal fossa and inserts into the anterior margin of the mandibular ramus, down to the retromolar region. It acts on the TMJ via its anterior fibres that elevate the mandible and the posterior fibres retract it. Accurate execution of such actions demands a relatively rich density of MSs to provide the required sensory input and is reflected in MPS by the wide area of pain distribution.

Hypersensitivity in the maxillary dentition can be a presenting sign as a result of pain referred from TrPs within this muscle. Also, headache in the temporoparietal region is a common effect of referred pain. As with the masseter, TrPs in specific regions refer pain to particular sites, namely fibres from the anterior third refer pain to the orbital margin con frontal bone and incisor teeth, TrPs in the mid-portion refer pain vertically up to the vertex and down, anteromedially over the whole of the upper portion of the face to the canine, premolar and molar regions, whereas from the posterior third pain is referred to the temporoparietal region with no referral to teeth.

Medial Pterygoid muscle

Sites of pain referral from the medial pterygoid muscle include pharyngeal (throat) region and infra-auricular

region, with possible involvement also of the Eustachian tube

Lateral Pterygoid

This muscle bears a direct relation to the TMJ. Referred pain arising from TrPs within the lateral pterygoid is referred to the TMJ and teeth, and so it can be very easily misdiagnosed for primary TMJ pain, or occlusal disharmony with secondary TMJ involvement. Superior part of lateral pterygoid muscle: - excessive shortening brought about by active trigger points may result in internal TMJ disk derangements as a consequence of excessive traction on the inter-articular disk. This will prevent the disk from attaining a proper anatomical position at the end of the jaw opening-closure cycle.

Inferior part of lateral pterygoid: - this part pulls the head of the condyle forward, allowing translation of the latter down over the articular eminence of the temporal bone. This action protracts the mandible and is important in achieving full opening. This can give rise to acute occlusal disharmony when excessively shortened.

Anterior digastric

Pain is referred to the mandibular incisor teeth.

Sternocleidomastoid

The pain referral pattern of this muscle is one of the most complex possibly found in the whole body not just in the oro-cervical region. This could well be a reflection of the wide array of functions and actions performed by this muscle, ranging from stabilisation of the head whilst allowing the TMJ to operate during chewing, to its recruitment in forced inspiration.

Also it is believed that TrPs in the sternocleidomastoid may give rise to TrP generation in the masseter and temporalis muscles, further augmenting to the complexity of the pain pattern. Such trigger point induced trigger point generation is called ‘satellite trigger point’ and is thought to be induced in nerves included in pain referral pathways.

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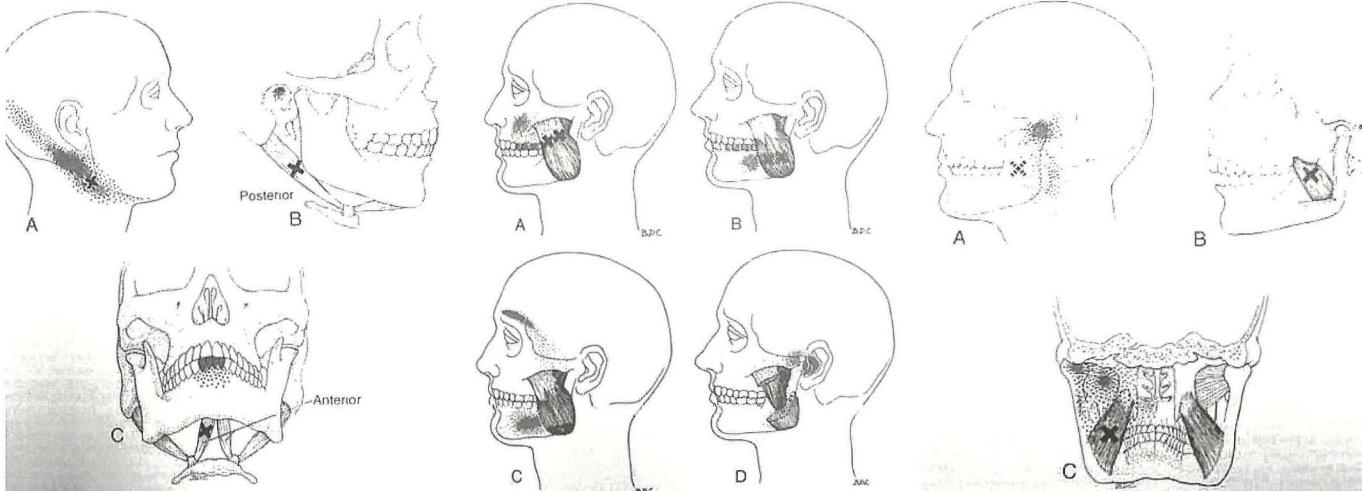
MYOFASCIAL PAIN SYNDROME

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Superficial sternal head: pain is referred to occipital, vertex, superciliary, temporal and maxillary regions.

Deep clavicular head: - frontal and retro-auricular pain. Apart from referred pain, concomitant autonomic phenomena secondary to trigger point activation are also reported. These include tear

discharge, conjunctival reddening and postural dizziness. Tinnitus can also be a presenting symptom, but this is thought to be brought about indirectly via activation of satellite TrPs in the masseter muscle. ■



Diagrams, clockwise from top left:

Fig.1: Myo Digastric

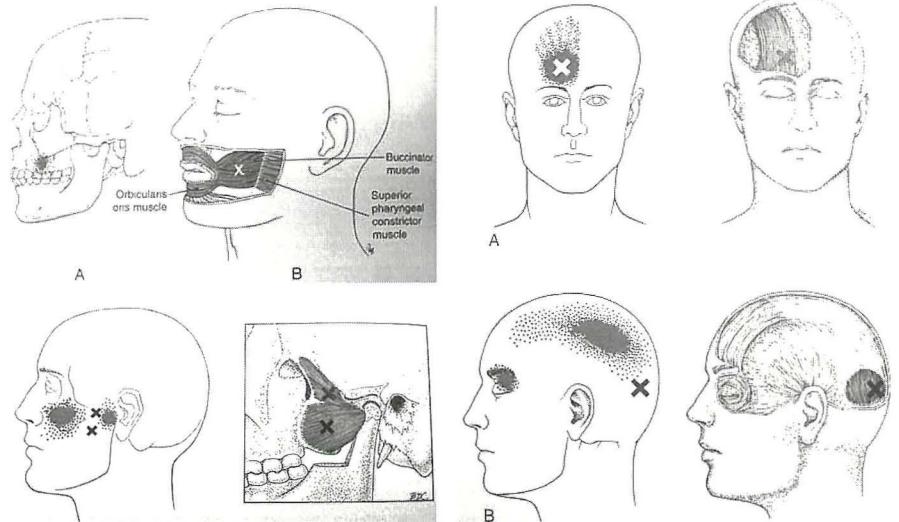
Fig.2: Myo Masseter trigger points diagram

Fig.3: Myo Medial Pterygoid

Fig.4: Myo Occipitalis and Frontalis

Fig.5: Myo Lat Pterygoid

Fig.6: Myo Buccinator



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By Dr Lino Said

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The effects of ANTIEPILEPTIC DRUGS in relation to Dentistry

Janet Mifsud, BPharm (Hons); PhD(QUB)

Vice President (Europe), International Bureau of Epilepsy

Advisor, Caritas Malta Epilepsy Association

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INTRODUCTION:

'THERE IS MORE TO EPILEPSY THAN JUST SEIZURES'

Epilepsy is the most chronic neurological disorder with an incidence of approximately 45/100,000 per year and point prevalence of 0.5-1%. It is important to distinguish between seizures and epilepsy (see Figure 1) and epilepsy is currently defined as a tendency to have recurrent seizures (sometimes called fits). A seizure is caused by a sudden burst of excess electrical activity in the brain, causing a temporary disruption in the normal message passing between brain cells. This disruption results in the brain's messages becoming halted or mixed up¹.

It is more accurate to refer to epilepsies. The International Bureau for Epilepsy classifies epilepsy in over forty different types and conditions. Epilepsy is classified "generalized" when the discharge affects the entire brain cortex. The main seizures types in this group are tonic-clonic (grand mal) and absence (petit mal). An epilepsy is called "partial" when the electrical discharge causing it occurs in a specific area of the brain.

Simple partial seizures result in no loss of consciousness, while complex partial seizures are characterized by automatisms. Based on the cause, epilepsy can be symptomatic (caused by a developmental mal-formation), idiopathic (when a genetic condition is responsible) or cryptogenic (when the cause is unknown). There are various

possible etiologies of seizures and epilepsy across various ages: prenatal or birth injury, inborn error of metabolism, or congenital malformation, idiopathic/genetic syndrome, CNS infection, or trauma, head trauma, drug intoxication and withdrawal or stroke, brain tumor, and neurodegenerative conditions².

Despite huge medical advances which have resulted in 70% of persons with epilepsy being seizure free due to the availability of pharmacological therapy, epilepsy is surrounded by social stigma, superstition, and rarely discussed in media. In Malta it is colloquially known as "tal-qamar", a term associated with a huge stigma and large efforts are being made to replace it with the term epilepsija. It is a condition which has a significant negative impact on the quality of a patient's life and the treatment goal of epilepsy is to make the patient be completely seizure-free without significant side effects.

WHY IS EPILEPSY OF INTEREST TO DENTISTS?

Understanding epilepsy and seizures is important in order to raise awareness of the this condition's impact on a patient's general medical and psychological health. In almost all aspects of oral health and dental status, the condition of patients with epilepsy is significantly worse than age-matched groups in the general (nonepileptic) population. Patients with poorly controlled epilepsy and who experience frequent generalized tonic-clonic seizures have worse oral health in comparison with

patients who are better controlled or who only have seizures that do not involve the masticatory apparatus.

The number of decayed and missing teeth, the degree of abrasion and periodontal indexes are also significantly worse in patients with epilepsy. Those with epilepsy also have significantly fewer restored and replaced teeth than the general population³.

ANTIEPILEPTIC DRUGS (AEDS)

Appropriate treatment is often lifelong and usually consists of pharmacological medications with antiepileptic drugs (AEDs). In Malta, persons with epilepsy are entitled to free medication under Schedule V (yellow cards). On first therapeutic choice, 60% of patients become seizure free which increases to 80% with some dose/therapy adjustments. Yet many of these drugs may have severe adverse effects which may impair the quality of life of persons with epilepsy, including dental care. These side effects can be direct ie affecting dental care, or indirect influencing the patient's ability to ensure their dental care.

ADVERSE EFFECTS WITH AEDS

Phenobarbital, which despite being an older drug is still widely used, can cause CNS depression, resulting in sedation, lethargy, and cognitive impairment. It is associated with an increase in falls in the elderly, porphyria, folate deficiency, osteomalacia, and megaloblastic anemia.

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The effects of ANTI-EPILEPTIC DRUGS in relation to Dentistry

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Phenytoin can result in fatigue, lethargy, blurred vision, drowsiness, incoordination, and nystagmus, hirsutism, acne, osteomalacia, folic acid deficiency, coarsening of facial features, Stevens-Johnson syndrome, bone marrow suppression, and hepatitis. Yet the main problem is the high incidence of gingival hyperplasia which is characterized by unusual growth of the gingival subepithelial connective tissue and epithelium, which is reversed once the drug is discontinued. About 50% of patients will develop it within 12–24 months of initiation of treatment.

The use of chlorhexidine, folic acid rinses or both, and good oral hygiene will significantly decrease the severity of the condition. In severe cases, surgical reduction is needed. Carbamazepine will cause diplopia, dizziness, headache, nausea, vomiting, sedation, and lethargy, skin rash, hyponatremia, and, very rarely, bone marrow suppression.

Valproate may result in nausea, vomiting, and dyspepsia, tremor, weight gain, thinning or loss of hair, and menstrual irregularities including amenorrhea, liver toxicity, thrombocytopenia, pancreatitis, polycystic ovary disease and has a high significant risk for teratogenicity⁴.

The newer AEDs, despite various promises, also have adverse effects that may affect dental care. Lamotrigine can lead to vomiting, coordination abnormality, dyspepsia, nausea, dizziness, and rhinitis; ataxia, somnolence, headache, diplopia, and blurred vision. More serious is a hypersensitivity reaction primarily presenting as rash which may progress to Stevens-Johnson syndrome and may result in fatalities.

Topiramate causes ataxia, poor concentration, confusion, dysphagia, dizziness, fatigue, paresthesia, word-finding difficulties, and cognitive slowing. Levetiracetam users have reported psychiatric and behavioral events such as emotional lability, nervousness and hostility. (see Table 1)⁵

DRUG INTERACTIONS WITH AEDS

In addition, AEDs interact with several drugs which the dentist may prescribe in certain conditions. Antifungal agents and antibiotics may interfere with the metabolism of certain AEDs. The coadministration of fluconazole and phenytoin is associated with a clinically significant increase in phenytoin's plasma concentration, and the dose of the latter may require adjustment to maintain safe therapeutic concentrations. Clarithromycin increases the plasma concentration of carbamazepine, and coadministration of these drugs should be monitored very carefully to avoid carbamazepine toxicity. Valproate may be displaced from plasma proteins and metabolic pathways may be inhibited by high doses of aspirin; this interaction will free serum valproate concentrations resulting in subsequent toxicity¹.

PROBLEMS THAT A DENTIST MAY ENCOUNTER

It is important for dentists to ensure prevention of peri-operative seizures. Patients must take their anticonvulsant medication and if necessary dentists should consult with patient's neurologist or family physician. Most persons with epilepsy who are well-controlled on medication, and can undergo surgery without difficulty or complication

Generalized tonic-clonic seizures often cause minor oral injuries, such as tongue biting, but also frequently lead to tooth injuries and in some cases to maxillofacial trauma. Patients with epilepsy can be at increased risk of fracture because enzyme-inducing antiepileptic drugs (e.g., phenytoin, phenobarbital, carbamazepine) alter the metabolism and clearance of vitamin D and have been associated with osteopenia and osteomalacia. It is important to ensure adequate calcium and vitamin D supplementation especially in patients taking phenobarbital, phenytoin or primidone³.

A recent analysis of the prosthodontic status of patients with epilepsy, found that compared with age-matched controls, PWE have a tendency to become edentulous earlier.

Prosthodontic treatment is suboptimal, as significantly fewer teeth are replaced, despite the fact that epileptic patients tend to have more missing teeth. Specific guidelines are recommended such as the discouragement of incisal restorations, use of fixed rather than removable prostheses and inclusion of additional abutments if fixed partial dentures are to be used. The use of metal base for complete dentures and telescopic retention with denture bases made of metal or reinforced with metal for nearly edentulous patients was recommended for those having seizures involving the masticatory apparatus, and seizures associated with falls⁶.

WHAT THE PATIENT SHOULD KNOW ABOUT AEDS AND DENTAL CARE

Children with epilepsy who are prescribed AEDs in syrup form may have problems with their teeth due to the syrup content in the medication, particularly if the dose is taken at night. It is important to use sugar-free liquid preparations. Some drugs e.g. carbamazepine, primidone and phenobarbital, may cause dryness of the mouth, due to changes in the composition or reduction in the production of saliva which is necessary for digestion of food. Other possible oral side effects of AEDs include glossitis, a smooth and painful tongue and oral ulceration (carbamazepine) and taste disturbances (phenytoin)⁴.

Patients should be advised that in the event of a fall due to seizures tooth fragments, crowns, fillings or even loose dentures may become dislodged or swallowed and if teeth are damaged, appointment with the dentist should be arranged as soon as possible. Sometimes, during a seizure, the inside of the mouth is bitten making it painful to brush the teeth and the use Corsodyl® mouthwash instead of brushing until any soreness or ulcers subside. Swelling can be reduced by holding a cold compress to the cheek or sucking an ice cube. Hot salt water mouthwash (half a teaspoon of salt to one tumbler of water) can help soothe and heal¹.

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The effects of ANTI-EPILEPTIC DRUGS in relation to Dentistry

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RECOMMENDATIONS

It is important to dentists to ensure that persons with epilepsy who are medication which may affect dental care are educated about the importance of seeking appropriate dental treatment due to the adverse effects of pharmacological therapy or damage to their teeth due to physical injury subsequent to seizures. Dental surgeons in turn should keep abreast of their clients medical and drug history and if possible liaise with the patient's neurologist if it is necessary to alter the patients treatment plan due to unacceptable adverse effects due to the AEDs (see Table 2). Treatment plan and design restorations should be undertaken in order to minimise the risk of damaging or displacing dental prosthesis due to an epilepsy seizure.³

Patients should also be made aware of local and national resources for information and support relative to their condition. The Caritas Malta Epilepsy Association (CMEA) (www.caritasmalta.org/epilepsy) was set up by voluntary workers in 1996 and now lists almost 300 members. In May 2001, the Association was accepted as a Full Member of the International Bureau for Epilepsy (IBE). The Association is for persons with epilepsy and their families. It aims to promote education and local awareness about epilepsy, especially because of the stigma suffered by people with this condition in society particularly in employment and improve health care services, treatment and social acceptance of epilepsy, as a serious yet treatable brain disorder. It has often invited dentists to the meetings to explain the importance of dental care to members. CMEA has also liaised with the Association of Dental Students of Malta in the production of an information leaflets about epilepsy. ■■■

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Figure 1: Seizure vs Epilepsy

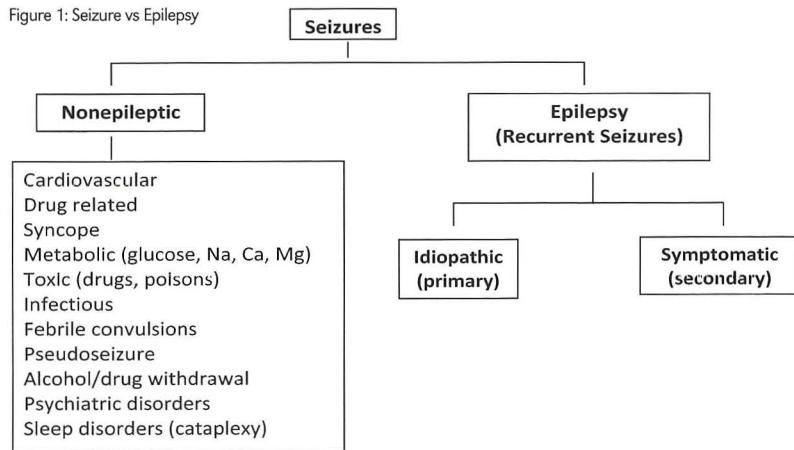


Table 1: Summary of most common oral side effects and dental considerations with AEDs

Medication	Indications (seizure type)	Most common oral side effects and dental considerations
Phenobarbital	Partial and generalized	secondarily Drowsiness/sedation, osteopenia/osteomalacia
Carbamazepine	Partial and generalized	secondarily Xerostomia, stomatitis, gingival bleeding, rash, osteopenia/osteomalacia
Phenytoin	Partial and generalized	secondarily Gingival hyperplasia, gingival bleeding, osteopenia/osteomalacia
Valproate	Partial and generalized	Gingival bleeding, petechiae, decreased platelet aggregation
Primidone	Partial and generalized	Drowsiness/sedation
Lamotrigine	Partial and generalized	Rash
Topiramate	Partial and generalized	Mild cognitive side effects
Clobazam	Partial and generalized	Drowsiness/sedation
Ethosuximide	Generalized	Drowsiness/sedation
Vigabatrin	Partial	Unknown
Diazepam	Generalized	Drowsiness/sedation
Gabapentin	Partial	Drowsiness/sedation
Levetiracetam	Partial and generalized	Unknown

Table 2: Acute, Dose-Related Adverse Effects of AEDs

Neurologic/psychiatric: most common	Mild to moderate laboratory changes
<ul style="list-style-type: none"> Sedation, fatigue <ul style="list-style-type: none"> More pronounced with traditional AEDs Unsteadiness, uncoordination, dizziness <ul style="list-style-type: none"> Mainly traditional AEDs May be sign of toxicity with many AEDs Tremor <ul style="list-style-type: none"> VPA Diplopia, blurred vision, visual distortion <ul style="list-style-type: none"> CBZ LTG Mental/motor slowing or impairment <ul style="list-style-type: none"> TPM 	<ul style="list-style-type: none"> Hyponatremia <ul style="list-style-type: none"> CBZ Increases in ALT or AST <ul style="list-style-type: none"> VPA Leukopenia and thrombocytopenia
	Endocrine/Metabolic Effects <ul style="list-style-type: none"> Osteomalacia, osteoporosis <ul style="list-style-type: none"> CBZ PHB VPA PHT Folate (anemia, teratogenesis) <ul style="list-style-type: none"> CDZ PIB VPA PHT Altered connective tissue metabolism or growth (facial neoplasia)
Tetratogenicity <ul style="list-style-type: none"> increased risk of cleft lip and/or palate in the new born <ul style="list-style-type: none"> PHT CBZ VPA 	Weight gain/appetite changes VPA GPB PGB Weight loss TPM ZMS FBM

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8 actions against the frequent problems identified by dentists.

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Bradlaw and Dentistry in the British Commonwealth

Continues from page 23

DENTAL SERVICES

There is no doubt that dental officers of the Royal Naval Dental Services, Royal Army Dental Corps and the Royal Air Force Dental Branch had an influence on the development of the dental services in many of the Commonwealth countries where they served. I have not managed to unearth much information on Bradlaw's relationship with the services. He was a Civilian Consultant to the Royal Navy and in 1971 as President of the GDC he addressed the 50th Anniversary commemoration of the Army Dental Corps giving a typically well researched history of the Corps which was published in the British Dental Journal.

The Eastman Postgraduate Institute played a major role in the further training and specialisation of service dental officers, many of them from the Commonwealth. Maltese dentistry definitely benefited greatly from the large presence of the British dental officers in Malta. I have already referred to the then Lieutenant Wills Rust's participation in the first University dental examination in 1919 and his identification of Lapira as a good prospect. Victor Vella Grech, a Maltese graduate from the first course joined the Army Dental Corps soon after qualifying in 1937 and served in the Corps for the rest of his career. In 1943 he followed DV Taylor as Deputy Assistant Dental Director Services Malta Command and eventually retired as a Brigadier in 1972. In the early post war period before the rundown of the British Services there were in Malta more Service Dentists than Maltese civilian ones. I remember, as a student going to the Royal Naval Hospital at Bighi to participate in the much appreciated clinical meetings. Later Naval dental specialists, such as Cdrs. Roy Travis, Geoff Keeble and George Rudge very kindly actively participated in our dental school teaching programme. I also had the honour to be appointed Civilian Consultant to the Royal Navy in 1972.

DENTAL ORGANISATIONS INCLUDING THE SURGICAL COLLEGES.

Towards the end of the Second World War when the building of the Royal

College was severely damaged, the College had a farsighted and courageous President in Lord Webb Johnson, who expanded the functions and scope of the College. Bradlaw was a close collaborator who fully grasped the opportunity to enhance further the role of dental surgery in the College. The earlier plans to establish a Faculty of Dental Surgery which had been shelved because of the war came to fruition in 1947 when the Faculty was established with Robert Bradlaw as the first Dean.

The College enlarged considerably its scope, establishing various departments including the Department of Dental Science, and added to its then primary role of an examining body, the functions of postgraduate teaching courses and research. Concomitantly the building expansion and the presence of the residential Nuffield College gave a big impetus to College life.

The training courses were a mecca for Commonwealth Dental students, many of whom would return to their own countries and play a vital role in the development of their dental schools and of Oral Surgery. A cursory glance at the staff list of Commonwealth schools shows that nearly all the staff had British postgraduate degrees. The second dean at the Malta Dental School, Joseph Mangion obtained his Fellowship, by examination in 1948, the first non-British dental surgeon to do so. Even now most members of the teaching staff at the Malta Dental School have a College postgraduate degree and many of the junior hospital staff are following College courses. It has not been all one-sided as many Commonwealth dental surgeons were to stay in the UK to practise in general dentistry, or become consultants and teachers in British Dental Schools.

This Faculty of Dental Surgery of the RCS has been evolving over the years, responding to the then current needs. The teaching Courses, initially geared to the Primary and Final Fellowship examinations, were for a long time models in postgraduate education. There has since been a big expansion of the range of courses offered in many instances jointly with other Colleges.

The original FDS examination has now been superceded by a number of qualifications at various levels and specialities, this is still very much work in progress. I know that from our school a high proportion of our graduates participate in the teaching programmes leading to the various degrees offered.

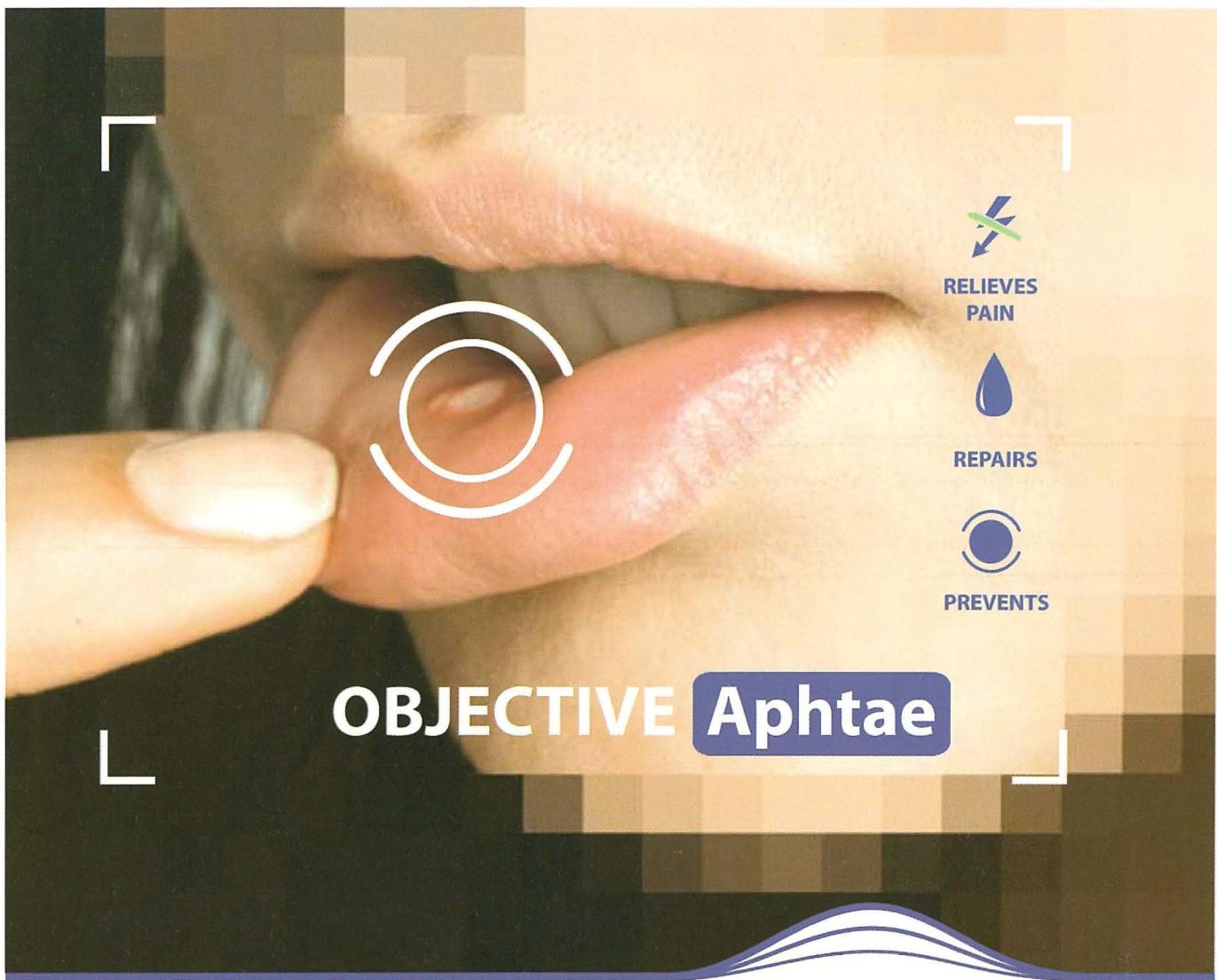
The clinical guidelines, excellent teaching and practical aids are keenly followed, and the new Journal of the Faculty is already making its mark. The College is considering another route for Fellowship in Dental Surgery, that is by submission of a CV and satisfying certain criteria.

It will be interesting to see, if adopted, whether it will be availed of by dental surgeons from Commonwealth Countries. Teaching unfortunately often leads to examinations, also a major arm of the College. The Faculty's active participation in the ORE and the revamping of the LDS, which was the first national dental examination in Britain, are very much topical items.

The importance in encouraging research is apparent in the establishment of the FDS Research Awards. It is obvious that the Faculty is a vibrant body closely responsive to the development of dental surgery in the UK and the needs of Commonwealth and foreign dentists wishing to continue the age old tradition of coming to Britain for work, study or research purposes. The overseas role Bradlaw was pursuing in the early days of the Faculty, has now developed into the International Committee.

The Malta Dental School external examiners have been or still are members of the Faculty Board of Dental Surgery of the College. In this way the ethos of the College continues to be transmitted to staff and students of our school.

We are proud of and congratulate Ms Kathryn Harley our new dean, the first female dean of this Faculty but I am sure Madam Dean will forgive me if I say that it is not her gender but her abilities and dedication to the Faculty and the College which will allow her to lead the Faculty to continued success and build on Bradlaw's founding dream. ■



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