

Health Matters

Degenerative Cervical Myelopathy



Royston Hospital

Area: Spine Surgery, Article written by:
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Degenerative Cervical Myelopathy (DCM) is a new term used to encompass all forms of cervical spinal cord impairment due to degenerative causes such as degenerative disc disease, ossification of the posterior longitudinal ligament or ligamentum flavum, and spondylitic cervical myelopathy¹.

This group of cervical disorders is the most common reason behind spinal cord dysfunction in the world². Certain genetic and developmental conditions such as collagen disorders, congenital spinal stenosis, Klippel-Feil syndrome and Down's syndrome can also predispose individuals to DCM. Compression of the cervical spinal cord due to degenerative conditions often leads to subtle dysfunction and requires a high index of suspicion in its early stages. Early diagnosis can be hugely beneficial in ensuring appropriate investigation and follow-up to prevent progression of the disease². Previously, treatment of DCM was mostly non-operative, with operative management believed to show little benefit in the late stages. This opinion now appears to be changing.

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“Very insightful”

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Message from Acurity Health

Chief Executive Officer
Jonathan Coleman, ph (04) 920 0131



It's been a really busy introduction since I started in my new role as CEO of Acurity at the start of May. My focus has been on getting around Acurity's network of hospitals and clinics, getting to know and understand the business, and of course meeting as many people as possible. A real highlight was the GP Conference at the end of May; it was an excellent series of presentations, and a very engaged audience.



Of course excellence in patient care is at the heart of what we do, and that's going to be supported by major new facilities and services, with the \$106 million rebuild of Wakefield, the \$16 million investment at Royston Hospital, the delivery of radiotherapy at Bowen Hospital to create a truly comprehensive cancer service, and the purchase of the Proactive chain of physio and rehab clinics.

It's very clear from all this that Acurity's owners Andrew Savage and Ben Thynne are making substantial investments in the company, and they are committed for the long term. There are some changes coming up though to the passive institutional investors who provide the capital for the ongoing growth and expansion of the business. The current range of investors have had money tied up in Acurity for nearly six years, and it's a normal part of the investment cycle for those

investors to rotate out after a certain period, just like individuals buy and sell stocks on the share market. That is what is happening over the coming weeks and months, and the process is being managed by investment bankers Cameron Partners and Rothschilds. Ben and Andrew will retain their current investment and involvement, and Acurity senior management remains in place; all the decisions about the Acurity business will continue to be made by this group and there will be no change in the day to day operations of Acurity facilities.

Anyway, there is obviously a huge amount happening at Acurity, and that reflects a dynamic, forward thinking healthcare company focussed on providing the best possible care for your patients.

Jonathan Coleman
Chief Executive Officer
Acurity Health Group Ltd

Degenerative Cervical Myelopathy

Continued from page 1

Neck pain is a common complaint in the early stages of the disease but is highly non-specific for DCM. Changes associated with compression of the spinal cord manifest as decreased motor activity and sensation in the extremities, decrease in coordinated movements, and altered bladder function. Patients can also present with complaints of radiculopathy in the arms, sometimes mimicking carpal tunnel syndrome quite closely³.

In my practice, I find great utility in quickly screening patients with the following questions:

- ① "Do you have difficulty doing up shirt buttons?" Or "Has your hand-writing changed despite your efforts to write neatly?"
- ② "Do your legs feel 'drunk' or not listen to you?"

Positive responses to both of these questions certainly merit further questioning and physical exam correlation.

Physical examination of DCM patients is highly variable depending on the severity of the disease as well as the specific location of cord compression. Motor and sensation testing of C5-T1 and L2-S1 is mandatory but can quite often be normal in early cases. Pathologic reflexes of the upper or lower extremities are worthy of referral for specialist consultation. Altered reflexes can be quite variable and may not be apparent until late in the disease. A positive Romberg's test, along with a positive history, may often be all that is present

on initial screening. In cases where the patient is unable to stand for a Romberg's test, proprioception of the great toes can be used as a surrogate⁴.

For patients with a worrying combination of history and physical exam findings, the next step should be specialist referral for assessment and appropriate imaging. If a diagnosis of cervical myelopathy is confirmed, then both operative and non-operative options can be discussed. Patients who have findings of cord compression but are non-myelopathic are at high risk for future myelopathic changes and should be counselled as such, but operative prophylaxis is not suggested. Current recommendations for mild DCM include close monitoring for deterioration and an appropriate rehabilitation programme. For patients with moderate to severe myelopathy, surgical intervention is now recommended based upon patient wishes and medical comorbidities².

Key Points

- DCM is common
- Early diagnosis is key
- Look for difficulty with buttons and subtle decreases in balance
- Surgical treatment may be an option.

Mr Austin Enright

Mr Austin Enright is a new Consultant at Royston Hospital. See p15 for details.

Surgical Treatment of Breast Cancer

Miss Ineke Meredith



Wakefield Hospital

Area: Breast Surgery, Article written by: Miss Ineke Meredith, Oncoplastic Breast and General Surgeon, ph (04) 381 8120

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Surgical Treatment of Breast Cancer in BRCA Mutation Carriers

Breast conserving surgery (wide local excision with clear margins, axillary management and radiotherapy) has been a standard in the management of breast cancer for over 20 years^{1,2,3,4}. In BRCA women without breast cancer, the benefit of prophylactic mastectomy is clear. However, after a diagnosis of breast cancer in these women, additional management options including bilateral mastectomy are frequently recommended, even though breast conservation is still appropriate.

The surgical treatment of women with BRCA who are diagnosed with a breast cancer is not standardised, and seems to be largely driven by the desire to prevent a second cancer. In a young woman who has a desire to give birth and breastfeed, the decision to undergo bilateral mastectomy is a difficult one. In addition to quality of life issues, prophylactic mastectomy can lead to negative effects on body image and sexuality^{5,6}. There is increasing agreement in the literature in regards to the acceptability of breast conservation in BRCA mutation carriers presenting with breast cancer. The option of breast conservation seems to be safe amongst BRCA women (with no significant difference compared with non-BRCA women) when examined by in-breast recurrence (longest follow-up 13 years)^{7,8,9} and survival¹⁰. Chemotherapy and prophylactic oophorectomy modify the risk of the former in these women.

The real question is over the contralateral breast with a 10-year risk of developing a second primary in the order of 20-40%⁸. Whether a contralateral prophylactic mastectomy confers a survival benefit in carriers with a breast cancer is open for debate and will likely be influenced by the prognosis of the initial cancer. Bilateral surgery is not a mandatory part of treating BRCA mutation carriers with a unilateral cancer. Any decision for more extensive or future surgery should be individualised and take into account the tumour prognosis, and the potential physical and psychological implications.

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HAL-RAR: Gain with Less Pain

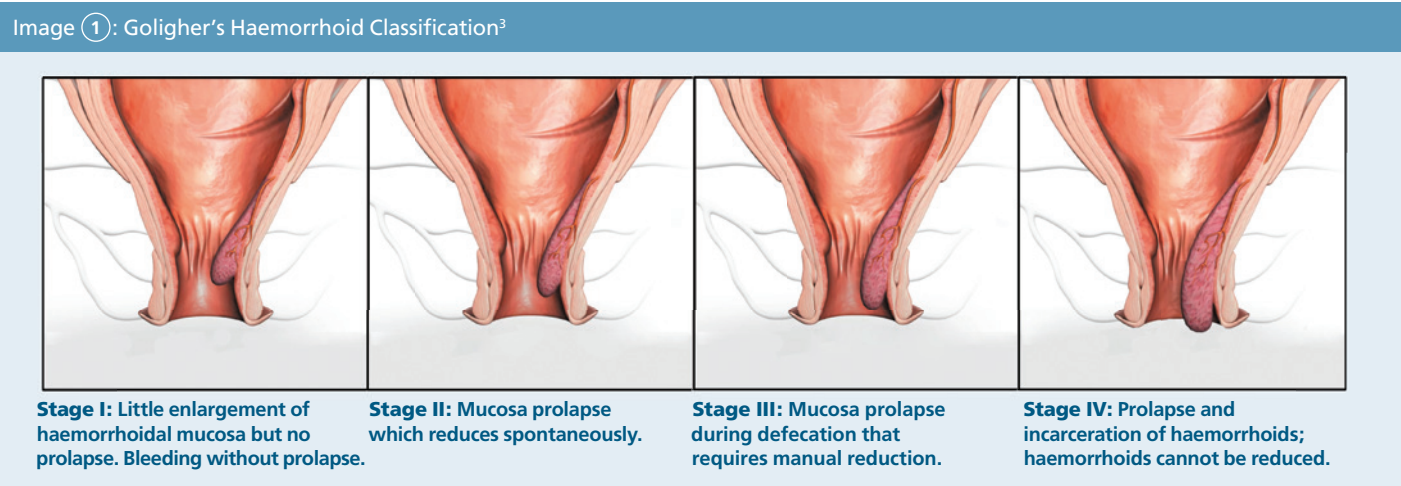
Haemorrhoidal Artery Ligation – Recto Anal Repair

Bowen Hospital

Area: General Surgery
Article written by: Mr Ali Shekouh, General Surgeon, ph (04) 479 8261

Haemorrhoids are common in the general population but the exact prevalence of the condition is difficult to quantify. This is because there are a number of patients who have haemorrhoids but do not present with any symptoms. The prevalence of haemorrhoids in a general population has been reported to vary from 4.4%¹ to 39%². When symptomatic, patients may present with painless rectal bleeding, itching, fecal leakage, mucus discharge and/or swellings outside the anus. They can also become painful when engorged and/or thrombosed.

Haemorrhoids can be classified as internal and external haemorrhoids in relation to the level of origin from the dentate line. Internal haemorrhoids are further sub classified – Stage I-IV depending on the level of protrusion (Image 1).



After other/sinister causes have been excluded for the symptoms, there are a variety of options for the treatment of haemorrhoids. There are number of conservative measures, non-surgical and surgical procedures for the treatment of haemorrhoids applicable to the relevant haemorrhoid stage (Table 1).

Table 1: Haemorrhoid Treatment Options	
Conservative	Operative haemorrhoidectomy procedures
<div><div>1</div>Avoid straining and constipation</div> <div><div>2</div>Increasing fibre and fluid intake</div> <div><div>3</div>Laxatives</div> <div><div>4</div>Banding of haemorrhoids</div> <div><div>5</div>Sclerotherapy injection of haemorrhoids.</div>	<div><div>1</div>'Open' Milligan-Morgan</div> <div><div>2</div>Ferguson 'Closed'</div> <div><div>3</div>Longo Stapled haemorrhoidectomy (PPH)</div> <div><div>4</div>Ligasure haemorrhoidectomy</div> <div><div>5</div>HAL-RAR (Haemorrhoidal Artery Ligation – Recto Anal Repair).</div>

What is HAL-RAR?

HAL (Haemorrhoidal Artery Ligation) was originally developed in 1995 by Dr Morinaga in Japan. The method was enhanced towards the end of 2005 by the addition of the RAR (Recto Anal Repair) component.

HAL-RAR is mainly targeted for the treatment of Stage III-IV haemorrhoids with/without mucosal prolapse. With the addition of the RAR component to the HAL procedure, mucosal prolapse can be dealt with alongside the haemorrhoids during the same operation.

The HAL procedure involves the accurate identification of the haemorrhoidal arteries within the rectum and ligation of the identified vessels (Image 2). "As a rule, between five and eight arteries will be found during the procedure. However, this number can vary from patient to patient, and will also depend on the severity of the haemorrhoids in each case. The ligations serve to reduce the arterial blood supply, causing the haemorrhoidal cushions to shrink back to normal size".

The RAR component is used to treat the prolapsing haemorrhoids that occur during more advanced stages of the disease. RAR involves one or more mucopexies of prolapsing mucosa, carried out after the haemorrhoidal arteries have been ligated (Image 3).



Does HAL-RAR work? YES

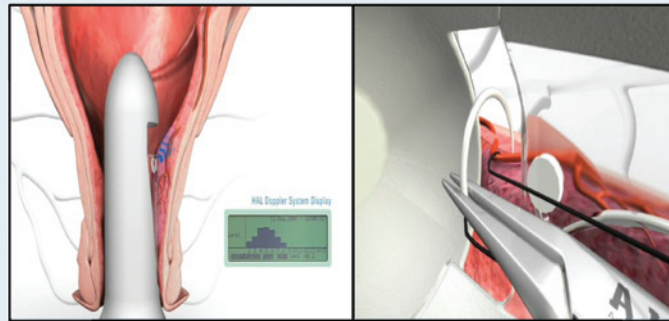
Since the introduction of HAL-RAR, there have been numerous prospective studies done which prove the safety and efficacy of the operation⁴. Patient satisfaction is high for overall improvement, improvement of bleeding and prolapse⁷. The post-operative pain incidence is low^{5,6}. HAL-RAR can be done as a day case⁵ and return to work and normal activities duration is much shorter when compared to traditional haemorrhoidectomy operations. In addition, the reported complication rates are low⁸.

In summary, HAL-RAR should be strongly considered as the treatment of choice in patients who have not responded favourably to conservative and non-operative measures for the treatment of their Stage III and IV haemorrhoids with/without mucosal prolapse. Each procedure can be modified and tailored to the requirements of each individual patient who could benefit from HAL-RAR's many positive attributes.

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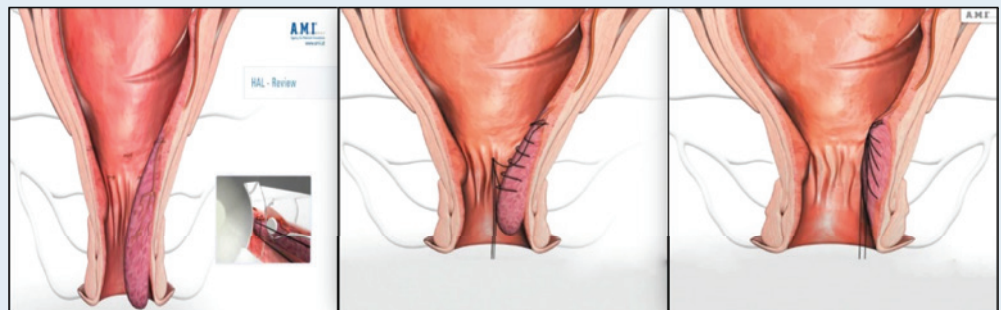
Image ②: Principles of HAL³



Doppler Sensor detects the hemorrhoidal arteries.

5 – 8 arteries are being ligated.

Image ③: Principles of RAR³

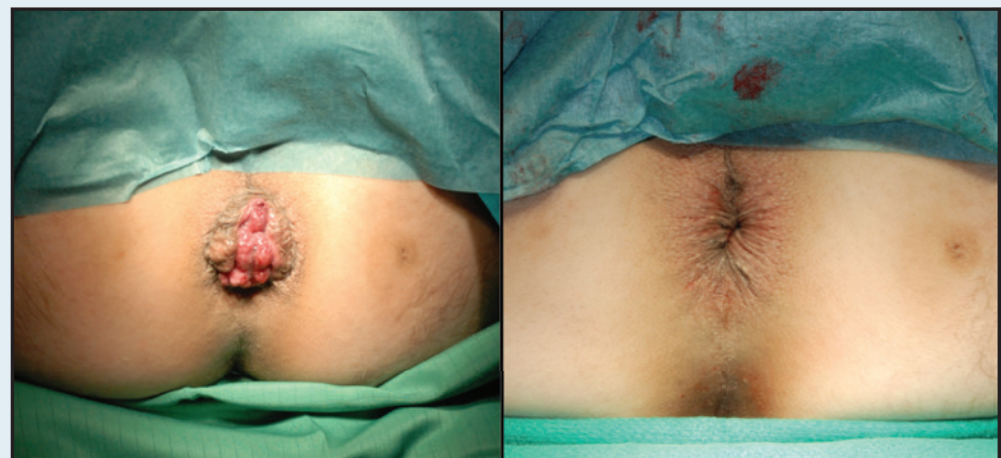


A running suture is started.

Then continued which ends proximal of the dentate line.

This causes the prolapsing tissue to be pulled up towards the initial stitch.

Image ④: Before and After HAL-RAR³



8 ligations and 3 mucopexies

Upcoming CME Meetings

Acurity Health Group hosts a variety of Continuing Medical Education (CME) sessions for GPs throughout the year.

Each session provides the opportunity to meet consultant physicians and surgeons, receive expert feedback and discuss topics.

To suggest a topic of interest or request information please contact Sarah Malone, Business Development Manager, P: (04) 920 0158, E: sarah.malone@acurity.co.nz

To register, please email marketing@acurity.co.nz

Upcoming CME Meetings					
Date	Speaker	Specialty	Topic/Details	Venue	CME endorsed
01 August Wednesday	Mr Ali Shekouh	General Surgery	1. New Treatment Options for Haemorrhoid and Fistula 2. The Colorectal Cancer Journey – From Diagnosis to Treatment	Bowen Hospital Seminar Room, Wellington	2 credits
08 August Wednesday	Mr Austin Enright	Spinal Surgery	Management of Back Pain: Are Opioids contraindicated?	East Pier Hotel, Napier	2 credits
15 August Wednesday	Mr Austin Enright	Spinal Surgery	Management of Back Pain: Are Opioids contraindicated?	Royston Centre Education Room, Hastings	2 credits
29 August Wednesday	Wakefield Heart Centre	Cardiology	Cardiology: An Update	Wakefield Education Centre, Wellington	2 credits
05 September Wednesday	Wakefield Heart Centre	Cardiology	Cardiology: An Update	Kapiti Lindale Conference Centre, Kapiti Coast	2 credits
25 October Thursday	Mr Stephen Toynton & Mr Paul Mason	ENT	Seminars in ENT Part 1 – Otology (Glue ear and beyond)	East Pier Hotel, Napier	2 credits
30 October Tuesday	Mr Stephen Toynton & Mr Paul Mason	ENT	Seminars in ENT Part 1 – Otology (Glue ear and beyond)	Royston Centre Education Room, Hastings	2 credits
07 November Wednesday	Bowen Icon Cancer Centre	Radiation Oncology	Topic to be confirmed	Bowen Hospital Seminar Room, Wellington	2 credits



Connect 2018

Acurity
Health Group Limited
GP CONFERENCE



Thanks to all of you who supported the Acurity GP Conference in its 20th year. Connect 2018 was certainly our best conference to date.

Presented by Acurity Health Group in partnership with the Department of Primary Health Care and General Practice, University of Otago, Wellington, on 25-26 May we welcomed GP's from all over New Zealand to Te Papa for this annual event.

MC Patrick Gower kept the audience entertained with the right amount of wit and humour as the key themes of Gut Health, Clinical Dilemmas, Pain Management and Musculoskeletal Conditions, Oncology, and End of Life Care were explored.

There was something for everyone, with plenty of new ideas, and solutions to some of

the everyday challenges faced in primary care. Forty presenters delivered a variety of short sharp sessions, in depth plenary talks and practical demonstrations. Boyd Swinburn's opening talk on 'Tackling the Obesity Epidemic' provided good tips and advice for general practice and Dr Colin Hutchison presented a thought provoking session on Chronic Kidney Disease, with a clear management guide to 'do the simple things well'. Other highlights included the panel discussion on 'Managing Severe Aortic Stenosis in the Elderly' and the practical lessons in balance for the older population proved extremely popular.

Friday nights networking function was a great end to a busy day and it was wonderful to see so many new and familiar faces.

As always we are grateful to our sponsors and exhibitors who make this conference possible every year, and provide valuable learning and updates in what's new in their individual fields.

Thank you to everyone who took time to provide feedback; planning for Connect 2019 is already underway and your ideas and suggestions will help us to deliver more of what you want. We look forward to welcoming you back next year, 29th – 30th March 2019 at Te Papa.

"Best
conference
I've been to"

Special thanks



Special thanks to the following speakers

Dr Malcolm Abernethy
Interventional Cardiologist

Mr Simon Bann
General Surgeon

Jamie Belesky
Physiotherapist

Karen Below
Occupational Therapist

Mr Marcus Bisson
Plastic Surgeon

Naomi Bondi
Speech Language Therapist

Mr Brendon Bowkett
Paediatrician

Dr Garry Brown
General Physician; Medical Advisor, ACC

Nicole Darkow
Occupational Therapist

Professor Elizabeth Dennett
General Surgeon

Dr Sinead Donnelly
General Physician

Dr Crawford Duncan
Psychiatrist

Mr Ilia Elkinson
Orthopaedic Surgeon

Dr Brian Ensor
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Mary Potter Hospice

Adj Professor Sean Galvin
Cardiothoracic Surgeon

Dr Colin Hutchison
Medical Director, Kidney Health
NZ; Nephrologist, Hawke's
Bay District Health Board;
Nephrologist and Spokesperson,
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Dr Giresh Kanji
Musculoskeletal Pain Specialist

Mr John Keating
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Connect 2018

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GP CONFERENCE



“Excellent
speakers
and topics”



Winner of the Practice Prize
Congratulations to Dr Nadine Kuiper of Porirua Union and Community Health Services, winner of the Acurity GP Conference Practice Prize, an Amtech MPVT 2 Section Surgical Bed.

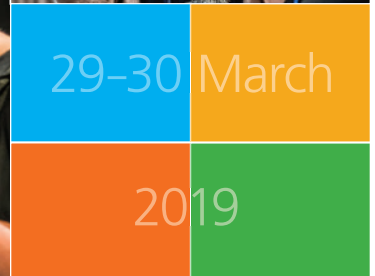
“Usual high
standard
– thank you”

“Great
conference, really
enjoyed it all”

“The best
year yet”

“The presenters
were consistently
very good”

Connect 2018



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- Wakefield Heart Centre
- Wakefield Hospital



Myopia: A Worldwide Epidemic?



Dr Anne-Marie Yardley

Bowen Hospital

Area: Ophthalmology

Article written by: Dr Anne-Marie Yardley, Ophthalmologist, ph (04) 472 1375

The prevalence of myopia (near-sightedness) worldwide has approximately doubled in the last 30 years¹. The highest levels of myopia are in East and South-East Asia where 80-90% of school leavers are myopic, and 20% are highly myopic^{2,3}. Although slower in other parts of the world, the rise in the prevalence of myopia has been universal. Twenty-five to fifty percent of North American, Australian and European children and young adults are now myopic².

Why is it a problem?

The basic structural change occurring in myopia is that the eye is longer than average. The eye grows throughout childhood, with myopia usually becoming evident in childhood or adolescence. Glasses, contact lenses and laser surgery can correct the associated defocus, but do not address the underlying changes which predispose highly myopic people (those with a prescription of -6.00D or stronger) to sight-threatening ocular problems^{4,5}. These problems include retinal detachment, glaucoma, and myopic macular degeneration⁵. Therefore, the increasing prevalence of myopia and high myopia is a major public health concern and interventions to prevent or slow myopic progression warrant attention^{4,6}.

What influences myopic progression?

Genetics and environment both influence myopic development. Although over a hundred regions of the genome have been linked to myopia, the timeframe for the increased myopic prevalence we are seeing cannot be explained genetically. There must be major environmental factors involved².

An association between levels of education and myopia has been appreciated for a long time².

Identifying the biological factors / drivers for this has been difficult⁵. The rise in myopia seems to match the trend for children to spend more time reading and using screens. However, studies looking specifically at near work as a factor in myopic progression have failed to prove this association².

A finding that has been reproduced worldwide is that time spent outdoors is protective against myopia^{2,7}. The Sydney Myopia Study showed children who spent more time outdoors did not become myopic, and they were not necessarily spending less time reading⁷. The mechanism for time spent outdoors being protective is not understood. While the brightness of the light seems to be important, it is not that simple^{7,8}. Also, the effect seems to be associated with total time outdoors rather than any particular activity (e.g. sports)⁷.

Can we intervene?

A recent Chinese prospective randomised control trial showed that prescriptive environmental interventions, such as increasing time spent outdoors by 40 minutes a day, do reduce myopic progression³. However, in our region of the world, spending time outside poses other risks related to UV exposure.

We need to establish exactly what is protective about time outdoors to ensure the benefits of any interventions related to this outweigh the risks.

Other more conventional strategies to slow myopic progression include pharmacological and optical interventions. A meta-analysis comparing the efficacy of sixteen interventions for myopia control in children concluded that the most effective interventions are pharmacologic, notably the muscarinic antagonist atropine. Specially designed contact lenses (orthokeratology and peripheral defocus modifying contact lenses) had moderate effects, and specially designed spectacle lenses had minimal effects⁹.



The effect of atropine drops on myopic progression has been known for years although, again, the mechanism is unclear. It had not been used routinely

as a treatment because of unacceptable side-effects with the 1% concentration used in early studies. These side-effects included photophobia, decreased visual acuity, near vision problems and rebound progression on treatment cessation. Recent trials using a low concentration (0.01%) of atropine show it effectively reduces myopic progression with minimal side-effects^{9,10}. Low-dose atropine drops are therefore being used around the world to try and slow myopic progression in some children. Studies to confirm safety, optimal treatment timing and applicability to all populations are ongoing.

In summary, the increasing prevalence of myopia will potentially increase the burden of sight-threatening problems. Prevention of myopic progression may provide substantial benefits. Low dose atropine drops currently seem the safest and most effective option for this. We are using these drops along with some contact lens methods in children whose myopia is progressing rapidly. Further research should improve our understanding about myopia, the factors that influence it and intervention options.

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Monoclonal Gammopathies in Primary Care

Bowen Hospital

Area: Haematology

Article written by: Dr Anup George, Haematologist, ph (04) 896 0200

With an aging population, a rise in malignant lymphoplasmacytic disorders is expected. In many patients with multiple myeloma the clinical disease is usually preceded by an asymptomatic phase with monoclonal gammopathy, where one clone of plasma cells produces an abnormal "monoclonal" protein (M-protein).

The abnormal protein in serum or urine confirmed, typed and quantified by electrophoresis (EP) as "M-spike" is usually an immunoglobulin (IgG, IgA, IgD or IgM) or a free light chain (Kappa or Lambda FLC), a sub-unit of immunoglobulin. Waldenstrom's macroglobulinemia (WM) is a paraproteinemic disorder where an IgM M-protein is present in conjunction with an indolent B-cell non-Hodgkin Lymphoma. Systemic amyloidosis can be a silent killer when the abnormal light chains infiltrate vital organs as "amyloid deposits" causing serious organ dysfunction like heart failure or nephrotic syndrome. Reactive non-specific hypergammaglobulinemia is seen in infections, chronic inflammation, liver disease and autoimmune disorders giving a polyclonal pattern in EP.

EP detects M-protein in 3-4% of those over age 50, 5% over age 70, and almost 10% of those 85 or older¹. Fortunately, only 20% of these individuals progress to myeloma and that too at a very low rate of 1% cumulative chance per year of progressing to multiple myeloma, WM or AL amyloidosis. The revised disease definitions were proposed by the International Myeloma Working Group in 2014² (Table 1).

MGUS and smoldering myeloma patients are asymptomatic and can be observed ("Watch and Wait") with regular clinical/laboratory monitoring whereas symptomatic myeloma patients need treatment. In addition to routine blood tests, other tests such as SEP, FLC, urine Bence Jones protein, bone marrow and imaging (skeletal lytic lesions) are required to complete the diagnostic work-up (Table 2).

Table 2: When should we do a SEP and/or sFLC assay?

If there is **unexplained** –

- ✓ – Bone pain
 - Pathological fracture
 - Age-inappropriate osteoporosis
 - Lytic lesions
- ✓ Heart failure (Probable Amyloidosis – Increased light chains with abnormal FLC ratio)
- ✓ Anaemia (usually normocytic) / raised ESR
- ✓ Renal insufficiency and/or significant proteinuria
- ✓ Hypercalcemia
- ✓ Hyper/Hypogammaglobulinemia (especially with recurrent infections)
- ✓ Peripheral neuropathy
- ✓ Hyperviscosity (headache, epistaxis).

How should we follow up these patients?

All patients should be assessed individually and younger patients and those with higher levels of M-proteins require closer follow up than the very elderly with very low levels of M-proteins.

MGUS prognostic score (based on three risk factors: M-protein >15g/L; non-IgG (IgA/IgD) MGUS or Abnormal sFLC ratio < 0.26 OR > 1.65) predicts risk of progression³. A **low-risk** MGUS patient (with no risk factors) has a 5% risk whereas a **high-risk** (all 3 risk factors) patient has a 58% risk of progression to myeloma in 20 years respectively. **Intermediate-risk** MGUS patients have a 21% (one risk factor) to 37% (two risk factors) risk.

After the initial work up a low-risk MGUS patient can be followed up in the primary care with annual clinical review along with CBC, calcium, creatinine, SPEP and sFLC assay. On the contrary, a high-risk patient is usually followed up in the specialist clinic with more frequent monitoring (3-6 monthly).

References

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- * Image: Histopathological image of multiple myeloma. Bone marrow aspirates. Hematoxylin & eosin stain. Source: [https://commons.wikimedia.org/wiki/File:Multiple_myeloma_\(2\)_HE_stain.jpg](https://commons.wikimedia.org/wiki/File:Multiple_myeloma_(2)_HE_stain.jpg)

Table 1: Revised Disease Definitions

	MGUS	Smoldering Myeloma	Myeloma
Symptoms	Asymptomatic	Asymptomatic	Symptomatic
M-protein	<30g/L	≥30g/L	Variable
BM clonal plasma cells	<10%	≥10-60%	Variable
"CRAB" criteria*	Absent	Absent	Present
MDE**	Absent	Absent	Present

* CRAB – HyperCalcemia, Renal impairment, Anaemia or Lytic Bone lesions

** MDE (Myeloma Defining Events) – ≥ 60% clonal BM plasma cells; sFLC ratio>100 or 2 or more focal lesions on MRI>=5mm.



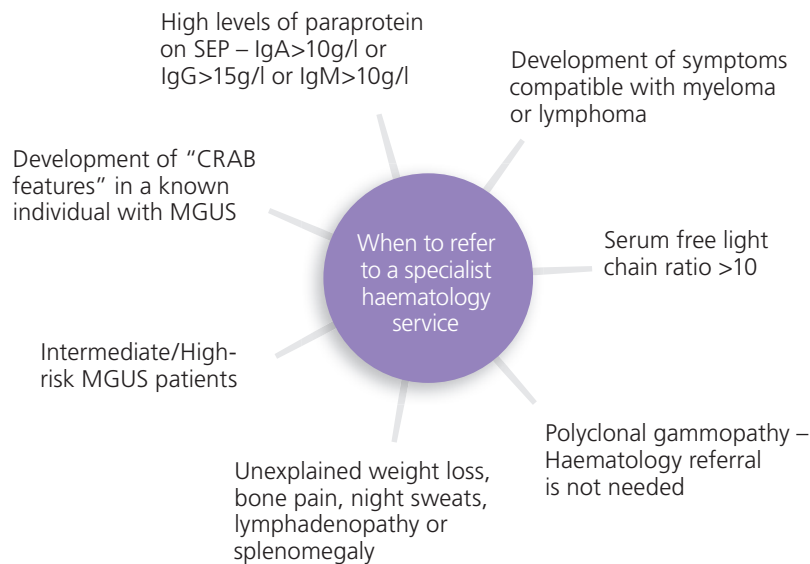
Dr Anup George is a new consultant at the Bowen Icon Cancer Centre. See p14 for details.

Bowen
icon cancer centre

Useful tips in the care of MGUS patients in the primary care

- Avoid NSAIDs (*risk of renal failure*)
- Maintain high fluid intake (*especially in those with high FLC and during summer*)
- Early/longer course of antibiotics (*esp. in hypogammaglobulinemic individuals*)
- Encourage Flu/Pneumococcal vaccinations
- Awareness regarding the increased risk of deep venous thrombosis
- Risk of osteopenia and osteoporosis (*Vit D/Calcium supplementation if needed*)
- Inter-laboratory variation of M-protein (*transfer of care from another GP practice*)
- Raised FLC or Abnormal FLC ratio can also result from renal failure.

When to refer a patient with MGUS to specialist haematology service



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Dr Anup George

MBBS, MD, FRACP, FRCPA

Haematologist

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Specialty

Haematology

Training

I completed my basic and post-graduate medical training in Internal Medicine from South India. After training in haematology through Christian Medical College, Vellore, India, I moved to Melbourne, Australia for my higher studies. I completed my fellowship in medicine and specialist training in haematology through St Vincent's Hospital and the Royal Melbourne Hospital. Before moving to Wellington, I was a clinical research fellow at the Peter MacCallum Cancer Centre, Melbourne with active participation in many clinical trials and publications in malignant haematology especially in the fields of lymphoproliferative disorders and myeloma.

Special interests

I am a haemato-oncologist with an active role in laboratory haematology. I care for patients with both malignant and non-malignant haematological conditions. But my special interests are with chronic leukaemias, lymphomas and monoclonal gammopathies/myeloma. I am actively involved in many clinical trials and am also a principal investigator for trials in myeloma and lymphoma at the Wellington Blood and Cancer Centre.



Dr Douglas Iupati

MBChB, FRANZCR

Radiation Oncologist

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EDl: acurityh

I consult at the Bowen Icon Cancer Centre in Bowen Hospital and can be contacted via their website. Radiation treatment (if required) will be delivered at the Wellington Blood and Cancer Centre until the Radiation Therapy is operational at Bowen (expected late 2018). Referrals can be sent to referrals.bowen@oncnz.team.

Specialty

Radiation Oncology

Training

- MBChB, Auckland University
- FRANZCR (Radiation Oncology)

Special interests

- Prostate cancer (including brachytherapy)
- Lung
- GU
- Upper and lower GI cancers.


Bowen
icon cancer centre

Dr Han Kim

MBChB, FRANZCR

Radiation Oncologist

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Specialty

Radiation Oncology

Training

- The University of Auckland (Medicine)
- Auckland City Hospital (Radiation Oncology Training)
- Princess Margaret Hospital, Toronto, Canada (Fellowship of Palliative Radiation Oncology (including oligometastases)).

Special interests

Han treats various tumour sites including prostate/ genitourinary, breast, head and neck, skin cancers as well as benign conditions such as Dupuytren's contracture and keloids. He is experienced in various radiation treatment techniques such as SABR/SBRT.

Background

Since moving to Wellington, Han has enjoyed teaching medical students as a senior clinical lecturer and working as a radiation oncologist. He continues his interest in research as part of the research committee at the Capital and Coast District Health Board.


Mr Austin Enright

BSc, MD, FRCSC

Spine and Orthopaedic Surgeon

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EDI: orthomed

Mr Enright is a fellowship trained orthopaedic surgeon with a specialty in operative and non-operative treatment of the spine. Having trained and worked in more than five countries, Mr Enright brings a fine-tuned set of skills and a unique perspective to Hawke's Bay.

Specialty

Spine and Orthopaedic Surgery

Training

- Fellowship, Spine Surgery, Middlemore Hospital, Auckland, NZ
- Residency, Orthopaedic Surgery, The University of Manitoba, Winnipeg, Canada
- Doctor of Medicine, St George's University
- BSc, Kinesiology and Exercise Science, The University of Victoria, Victoria, BC, Canada.

Special interests

Operative and non-operative treatment of:

- Cervical, thoracic and lumbar spine pathology
- Hip replacements
- Knee replacements
- General orthopaedics.

Mr Austin Enright is speaking at two CME meetings in August. See p6 for details.


Mr Andrew Ing

MBChB, FRACS

General Surgeon

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Specialty

General Surgery

Training

Graduated from University of Aberdeen in 2004; emigrated to New Zealand in 2005, completing specialist training before settling in Hawke's Bay.

Special interests

General, colorectal and laparoscopic surgery.

Background

Happily married father of two and a rugby fanatic.


Dr Sandhya Deo

MBChB, FRACS (plast)

Plastic and Reconstructive Surgeon

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Specialty

Plastic and Reconstructive Surgery

Training

Graduate of Auckland Medical School in 2003, with subsequent training in Plastic and Reconstructive Surgery in Christchurch, Auckland and Wellington. I became a Fellow of The Royal Australasian College of Surgeons in 2013.

Special interests

I have post-fellowship training in breast reconstruction, adult hand surgery and aesthetic surgery of the breast and trunk. Other areas of special interest include melanoma and skin cancer surgery and burn reconstruction.

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