Chronic Low Back Pain

A Practical Approach to Diagnosis and Management

Chronic LBP remains the most frequent and costly musculoskeletal complaint in Western Societies, and (to make matters worse) a precise anatomical cause can only be identified in a small proportion of cases. In simple terms, the prevalence of back pain is increasing, and we still don’t really know what is causing the problem!

The most recent advances in back pain diagnosis is to focus on developing classification subgroups for back pain that are based on the personal impact of the problem, as opposed to detecting a specific patho-anatomical source. This is very similar to the approach that we take in our Back Institute clinics here in New Zealand, and is also the direction that local district health boards are making to enhance and standardise the clinical pathway for patients seeking medical attention from primary health providers.

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Welcome to the latest edition of Health Matters. It’s always a pleasure to provide you with this educational and informative publication, and I am eternally grateful to our valued specialists for taking the time to prepare the information you have in front of you today.

Health Matters provides a great opportunity to welcome consultants new to private practice, and those who have moved their practice from elsewhere. Please take the time to read about the latest group of talented specialists working out of Acurity facilities. These can be found on pages 14 and 15.

New Websites
We have recently undertaken the daunting task of updating our websites, bringing a more modern feel to our online presence, and improving the navigability in the process. A number of GPs asked us to make it easier to find a specialist based on their subspecialty or specific area of interest – we hope you will find that the new search function makes this process a lot easier.

Wakefield Hospital Cath Lab
Together with the Heart Centre, Wakefield Hospital is very excited to have construction underway on our new state of the art Catheterisation Laboratory. We have selected Siemens as our partner in this development, and will be rolling out their Artis Q solution in the new lab.

Alongside the existing procedures undertaken within the Cath Lab, we are seeing rapid uptake of the Electrophysiology service offered by Drs Alejandro Jimenez Restrepo and Matt Webber.

Keep an eye on future issues of Health Matters for more details, including a pictorial tour, of our new lab.

‘Connect 2015’ Acurity Health GP Conference
Our 2015 GP Conference is rapidly gaining momentum, and will once again be held at Te Papa Tongarewa in Wellington – “the coolest little city in the world”. The conference will be held on Friday 8th and Saturday 9th May, and includes a fantastic programme along with some great new initiatives which I’m confident will be of interest to all.

CME Meetings
Our CME evenings continue to be well attended and well received. Sarah Malone and Persephone Georgiakakis, our Business Development team, put in a huge effort to make these events happen, and it’s great to see those efforts rewarded with strong attendance.

In delivering CME evenings, we place a strong emphasis on feedback from the GP community, and continually tailor our approach to ensure that our sessions are relevant, well structured, and in line with the expectations of the audience.

Our most recent event saw Ophthalmologists Steve Mackey, Reece Hall and Kenneth Chan present to a well-attended evening at Bowen Hospital. As this edition of Health Matters goes to print, we also see Gynaecologist Latha Vasan presenting on the topic of Abnormal Uterine Bleeding at Wakefield Hospital.

Early in 2015 we also have events scheduled for Cardiology and General Surgery in Wellington, and Vascular Surgery in the Hawke’s Bay.

Another New Year
It’s hard to believe that another new year is just around the corner, and I’m sure I’ll find myself writing up another message for the March edition of Health Matters before I know it.

In the meantime, I wish you and your families a happy and safe festive season.

Paul Quayle, Chief Operating Officer, Acurity Health Group Limited
The classification approach that we use was initially developed by the Canadian Back Institute (CBI). The system utilises a standard history and physical examination to subgroup patients into five distinct ‘pain patterns’ that are based on dominant site of pain and the response of symptoms to various spinal loads (postures and movement). A focused neurological screen and the elimination of systemic or organic disease, completes the exam.

Patterns 1 and 2
Two of the five patterns (pattern 1 and pattern 2) represent back-dominant pain (pain located in the low back, buttock, greater trochanter or groin). Pattern 1 defines back pain that is primarily aggravated with flexion based postures and spinal movement. A sub category for Pattern 1 (Pattern 1 – Prone Extension Positive (PEP)) exists to differentiate patients that get some relief during passive low back extension exercises or postures whilst lying prone. Those with back pain that is aggravated in both spinal flexion and extension positions make up a second sub category for Pattern 1 (Pattern 1 – Prone Extension Negative (PEN)).

Pattern 2 categorises back pain that is aggravated with spinal extension activities such as standing or walking, and is relieved during flexion activities.

Patterns 3 and 4
Pattern 3 and 4 classify leg pain of spinal origin. Pattern 3 represents true sciatica, with symptoms of leg dominant pain that is aggravated with spinal postures and/or movement. These patients must also have a positive straight leg raise (SLR) and/or a distinct neuropathy. Pattern 4 represents neurological claudicant pain, normally from spinal stenosis, with pain in the leg that is aggravated by upright activity such as walking or running, and is relieved with sitting.

The five patterns that represent mechanical spinal pain (Pattern 1 PEP, 1 PEN, 2, 3 and 4) are not permanent, and patients can present with altering patterns over the course of their recovery and many individuals experience a variety of pain patterns throughout their lifetime.

Pattern 5
A final pattern, pattern 5, identifies individuals with an abnormal pain focus that dominates the clinical presentation. This pattern is not mutually exclusive, and usually accompanies an underlying mechanical pain syndrome.

Once categorised into a ‘pattern’ the clinical decision making process to help determine treatment becomes easier. Pattern 1 and 2 patients have mechanical back pain that is usually self limited and responds favourably over time to active rehabilitation. Patients with long standing pattern 1 PEN type pain may require spinal fusion if rehabilitation has not been successful. Unresolved pattern 2 patients may occasionally require a steroid injection into the facet joint to break the pain cycle so the patient can resume rehabilitation. Pattern 3 (sciatica) that does not settle may require discectomy surgery and pattern 4 patients that fail to respond to a lengthy core strengthening programme can benefit from decompression spinal surgery. Finally, the most effective treatment currently available for Pattern 5 patients (individuals with CLBP and substantial chronic pain behaviours) is a multidisciplinary programme with a Cognitive Behaviour Therapy focus.

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Table 1. The Back Institute Patterns of Low Back Pain

<table>
<thead>
<tr>
<th>Pain Pattern</th>
<th>Subjective symptoms</th>
<th>Objective signs</th>
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<tbody>
<tr>
<td>Pattern 1 PEP</td>
<td>Back dominant pain</td>
<td>Pain is worse when bending forward and when bending backwards</td>
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<tr>
<td></td>
<td>Pain is usually intermittent (can be constant)</td>
<td>Pain is relieved when bending backwards</td>
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<td></td>
<td>Pain is worse with flexion based spinal postures and/or movement</td>
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<tr>
<td>Pattern 1 PEN</td>
<td>Back dominant pain</td>
<td>Pain is worse when bending forward, and when bending backwards</td>
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<tr>
<td></td>
<td>Pain is usually constant (can be intermittent)</td>
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<td></td>
<td>Pain is worse with spinal flexion and extension postures and/or movement</td>
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<tr>
<td>Pattern 2</td>
<td>Back dominant pain</td>
<td>Pain is worse when bending backwards and when bending forwards</td>
</tr>
<tr>
<td></td>
<td>Pain is always intermittent</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Pain is only worse with spinal extension postures and/or movement</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Pain is relieved with spinal flexion postures</td>
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<tr>
<td>Pattern 3</td>
<td>Leg dominant pain</td>
<td>Leg pain changes with spinal movement and/or distinct neuropathy</td>
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<tr>
<td></td>
<td>Pain is always constant</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Leg pain is aggravated and/or relieved with different spinal postures and/or movements</td>
<td></td>
</tr>
<tr>
<td>Pattern 4</td>
<td>Leg dominant pain</td>
<td>Normal or non contributory spinal examination</td>
</tr>
<tr>
<td></td>
<td>Pain is always intermittent and aggravated with upright activity (particularly walking) and relieved when sitting</td>
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References


When is it Iritis?

Rarely does the known anklyosing spondylitis patient walk into your clinic with four days of unilateral achey red eye, mildly reduced vision, photophobia, and perilimbal flush. While these patients do exist, you are likely to diagnose and refer without a second thought. It is the other ones we angst over.

Last week a patient presented to me with two months of intermittent unilateral redness, with a background story of similar symptoms on and off for 12 months. Only in the last few weeks had she noticed reduced vision and photophobia. Slit-lamp exam revealed anterior uveitis. Nothing typical about that presentation.

So what are you to do? Obviously if it walks and talks like an iritis, you will refer; however more commonly the unilateral red eye is simply the first manifestation of a viral conjunctivitis, and in a few days will be revealed by fellow eye involvement. If not, the harm in leaving a moderate iritis grumbling for a few days is not great. If there is an increasing inflammation, it will be manifest in the next 24 hours. Obviously diagnosing earlier means less inflammation, but practically the eye department can’t review every viral conjunctivitis – there has to be a compromise. As long as you aren’t missing acute closed angle glaucoma (corneal haze, pain, vomiting, fixed mid-dilated pupil, generalised redness, reduced vision and most likely different age group), a few days can help decide whether referral is warranted.

Given each Primary Care Specialist only sees ocular conditions intermittently, the best approach is to revert to your template: best-corrected and pinhole vision, lids (commonly conjunctival hyperaemia is blepharitis-related), conjunctival injection, cornea with fluorescein staining, pupil reaction (patient reaction to the light), and your best effort at fundus examination. If any of these signs or any history are suspicious, a slit-lamp examination is your best friend, via either your local Optometrist, Eye Registrar or Ophthalmologist.

Recently I came across this scenario: A first presentation to the Optometrist revealed Acute Anterior Uveitis (AAU). As the Optometrist was therapeutically trained, steroid eye drops were started, the patient monitored, and treatment tapered. When symptoms returned off drops (as can commonly occur), drops were restarted, and the patient referred to their GP with a request from the Optometrist to please investigate any underlying systemic condition. Previous to the advent of therapeutically trained Optometrists, patients would be seen by an Ophthalmologist and directed investigations ordered. As more therapeutically trained Optometrists graduate, this scenario of requesting the GP to order tests may occur more often.

In practice, a first presentation of typical AAU needs no tests, and a second needs ophthalmological referral anyway, where tests can be ordered guided by the clinical examination. In the above case, the ‘second’ presentation recurring on cessation of eye drops, was an exacerbation of the first, and no investigations were required (assuming review of systems was negative). Evidence of an associated systemic predilection may not occur for some years however, when they are no longer receiving Ophthalmic care, and tests will then be directed by systems involvement.

In summary

1. Diagnosing iritis without slit-lamp assessment is difficult and should be sought if the practitioner has concerns
2. If the suspicion of iritis is low, there is little harm in treating as conjunctivitis for a few days and reviewing
3. Investigations for any systemic associations should be titrated to the patient.
Paroxysmal supraventricular tachycardias (PSVT) are some of the most common cardiac arrhythmias affecting all age groups, but are especially prevalent in adolescents and adults without structural heart disease. Both men and women are affected equally but the demographics of the specific condition vary with age and gender. Although atrial fibrillation and atrial flutter are anatomically speaking supraventricular arrhythmias, they have a different pathophysiology from PSVT and are normally discussed as separate entities (not discussed in this article).

In terms of pathophysiology there are essentially two mechanisms responsible for supraventricular arrhythmias:

1. **Reentry**: By far the most common mechanism and especially prevalent in children, adolescent and young adults. The two most common reentrant arrhythmias are AV nodal reentrant tachycardia (AVNRT) and AV reentrant tachycardias (AVRT).

- **AVNRT** – Most common in women 20-50 years of age, but can present later in life. The mechanism involves an extra connection within the area of the AV junction. Normally the atrial impulses reach the ventricles via fast (beta) fibers. Some patients are born with dual pathways for AV conduction (Beta fast fibers and Alpha slow fibers). This is known as Dual AV nodal physiology and is a required condition for AV nodal reentry.
Continued on page 8

- **AVRT** – More prevalent in young adolescent men, but can manifest through adulthood (rarely above age 65). The mechanism involves an extra connection between the atrium and ventricle somewhere along the mitral or tricuspid annulus. Many patients have antegrade (downstream) conducting pathways, which leads to the classic ECG characteristic of “Delta Waves”. When patients present with delta waves on ECG and have symptoms consistent with palpitations or documentation of SVT they are classified as having pre-excitation syndrome, commonly referred to as Wolff-Parkinson-White (WPW) syndrome. Some patients with accessory pathways do not manifest delta waves on ECG (as the accessory pathway only conducts retrogradely or “upstream”) and they are referred to as having “concealed” pathways. (See figure 2)

*Figure 1 – AV Reentry (accessory pathway)*

- Fluoroscopy in LAO projection of ablation (Abl) catheter positioned in the lateral mitral annulus at the location of an accessory pathway
- Diagram showing the location of the patient’s accessory pathway (asterisk)
- ECG in sinus rhythm showing manifest pre-excitation (delta wave and short PR interval)
- ECG during ablation showing three beats with delta wave followed by loss of delta wave and normalization of the PR interval.

*Figure 2 – AV Nodal Reentry*

- Fluoroscopic image in RAO projection of catheters positioned in the His bundle (His), Coronary sinus (CS os) and right ventricle (RV). TV marks the location of the tricuspid valve. The ablation catheter (Abl) is positioned in the slow AV nodal pathway region (green), which is the target for ablation
- Anatomical diagram of the triangle of Koch and the AV node. The green coloured area represents the slow AV nodal pathway
- ECG during tachycardia showing AVNRT, followed by a few junctional beats and sinus rhythm.

Figure 1 – AV Reentry

![Figure 1 – AV Reentry](image)

Figure 2 – AV Nodal Reentry

![Figure 2 – AV Nodal Reentry](image)
2. Increased Automaticity

Less common than reentrant arrhythmias. Automatic or focal atrial tachycardias occur when atrial cells outside of the sinus node behave like automatic or “pacemaker” cells and develop automatism (ability to generate intrinsic electrical activity) by changes in the phase 4 of their action potential. This is usually a response to stretching of the atrial muscular fibers. They are most common in the setting of structural heart disease either by a disease process (hypertension, cardiomyopathy, pulmonary disease) or by endurance training (athletes’ heart). The classic ECG is that of a narrow SVT with a distinct P wave morphology (often times different to the sinus P wave) and a slight variation in the heart rate (known as the wobble). (See figure 3)

The most common presenting symptoms of PSVT are palpitations. They are characterised as sensations of chest “fluttering” or “thumping”, they are unpleasant in itself or can be associated with atypical chest pain, lightheadedness and dizziness (especially if the heart rate is very fast). Occasionally patients will present with syncope (loss of consciousness) and in those cases an urgent referral for a specialist assessment is recommended. In patients with underlying coronary disease, PSVTs can trigger chest pain and angina pectoris. Reentrant SVTs are commonly triggered by exercise and changes in body position, and are of sudden onset and termination (episodes can last minutes to hours). Valsalva maneuvers may terminate the arrhythmia. In contrary, focal tachycardias tend to have “flairs” of incessant arrhythmias throughout the day (bursts) and can be triggered by caffeine, alcohol, exercise or stress. They usually do not terminate with Valsalva, but the rate of the tachycardia may slow down.
The diagnosis of a PSVT is suspected on clinical grounds but is confirmed with ECG documentation of the arrhythmia, either by a 12 lead ECG, a Holter or event monitor or in some cases by interrogation of a cardiac implanted device (pacemaker, defibrillator or implantable loop recorder). The characteristics of the ECG help differentiate the different arrhythmia mechanisms.

Management of PSVT in the acute setting involves monitoring of vital signs (blood pressure, heart rate and oxygen saturation), forearm vein cannulation for IV access, attempts at termination with Valsalva maneuvers and administration of antiarrhythmic agents to terminate the arrhythmia.

The most commonly used agents are Adenosine, Verapamil, Diltiazem and Metoprolol IV. It is important to record an ECG during Valsalva and drug administration, as the response to the drug or vagal maneuver may provide clues of the underlying arrhythmia mechanism.

Investigations for patients with PSVT should include studies to rule out structural heart disease and/or coronary disease (echocardiogram, stress testing) when physical exam and/or symptoms/risk factors suggest an abnormality. Routine studies to rule out thyroid or adrenal disorders, anemia and electrolyte imbalances are also recommended.

Long-term treatment of patients with PSVT consists of either pharmacological or invasive treatment:

1. Medications

Commonly used chronic agents include beta blockers (cardioselective agents preferred), non DHP calcium channel blockers (Diltiazem, Verapamil), and sodium channel blockers (Flecainide, Propafenone). Patients can have chronic suppression of SVT episodes but the response rate is variable (between 60-80%) and side effects from antiarrhythmic drugs are common. For patients with occasional SVT episodes, which are sustained, a “pill in the pocket” approach is often times used. This involves the administration of short-acting antiarrhythmic agents if an attack occurs, with the expectation that the medication will stop the SVT within a short period of time. It is important to note that potent class III antiarrhythmics such as Amiodarone and Sotalol should not be routinely used for management of PSVT due to their toxicity profile and proarrhythmia potential.

2. Electrophysiologic Study (EPS) and Catheter Ablation

Is the preferred option for most patients with PSVT as it is curable, safe, ambulatory (day surgery) and spares the patient from chronic medication use. This procedure is a minimally invasive surgery where a vein is cannulated to advance catheters into the heart to localise the normal electrical pathways and identify potential abnormal circuits (reentrant or focal) and cauterise the abnormal tissue with a special ablation catheter that utilises radio-frequency (heating) or cryo (freezing) energy. Position of the catheters inside the heart is guided by fluoroscopy (figures 1 and 2) or using a specialised three dimensional mapping system (figure 3). EPS is particularly important for patients presenting with PSVT and syncope and patients with pre-excitation (delta waves) and atrial fibrillation. These patients have a small, but potential risk of sudden cardiac death and the EP study helps stratify that risk and provide a cure for the arrhythmia condition. Patients with asymptomatic delta waves also require risk stratification, which can be done with either an EPS or an exercise treadmill test (ETT). Recovery from an EPS only requires about four hours of bed rest. No sutures are needed. All catheters are removed and the access site heals completely within a few hours. Patients can return to a normal level of physical activity (including exercise) after a few days.

Key points

- Supraventricular arrhythmia related symptoms are a common reason for presenting to the general practice or emergency department
- SVTs are classified as reentrant or focal according to their mechanism
- ECG documentation of the arrhythmia is important to make an accurate diagnosis, as the specific arrhythmia type cannot be determined on the basis of symptoms alone
- Electrophysiologic study and catheter ablation offers a safe and highly effective treatment for most PSVT. Procedure is ambulatory for most patients (day case)
- Patients with syncope, angina or hemodynamic compromise during an episode of PSVT need urgent specialist referral
- Patients with preexcitation (delta waves), even in the absence of symptoms, need to be risk stratified for potential risk of sudden cardiac death. EPS and exercise treadmill testing are the preferred methods for risk stratifying these patients.
Radiofrequency Ablation (RFA) Treatment for Varicose Veins

Superficial venous insufficiency of the lower extremities is a common problem. There are several modalities available to treat varicose veins. This article specifically examines the technique of radiofrequency ablation (RFA).

RFA is a minimally invasive technique to obliterate the greater saphenous vein (GSV) in the thigh and/or the small saphenous vein (SSV) at the back of the calf. Both of these veins commonly feed into visible varicose veins. Duplex ultrasound is used to guide percutaneous needle and catheter access into the vein, placement of catheter position in relation to saphenofemoral junction and administration of tumescent anaesthesia.

The ClosureFAST (VNUS Medical Technologies Inc.) catheter is the most commonly used RFA catheter. This catheter allowed for segmental ablation of seven centimetre vein segment in one 20 second energy cycle.

The heat produced destroys the vein but the procedure relies on direct contact between the catheter and the vein wall. To facilitate this process, the vein needs to be as tightly wrapped around the catheter as possible so compression is applied to the vein using tumescent anaesthesia.

Tumescent anaesthesia (injecting fluid around the vein) is performed using ultrasound guidance. This will prevent pain being felt when the vein is being treated. Secondly, it provides a fluid bath insulation to prevent thermal injury of tissues surrounding the vein during RFA. The anaesthetic solution is dilute local anaesthetic mixed with large volume of saline solution. This is a simple and straightforward process.

The RFA method of venous closure primarily acts by inducing vein wall collagen contraction through heat-induced denaturation of the collagen matrix, followed by fibrotic sealing of the lumen due to inflammation of the vein wall.

RFA is an alternative to traditional surgical ligation of saphenofemoral and saphenopopliteal junction and stripping veins. Suitable patients will generally have greater saphenous vein (along thigh) and or small saphenous vein (back of calf) reflux.

They will need a vein that is reasonably straight in order to pass the RFA catheter up the vein. It is also important that fluid can be injected around the vein to separate it from the skin and surrounding structures so they do not get burned, but this is a very unlikely event.

Large visible tortuous varicose veins cannot be treated. Thread and reticular veins cannot be treated with RFA and are usually best treated with sclerotherapy. RFA is generally performed under local anaesthesia as a day stay procedure. It avoids general anaesthesia associated with traditional surgery. Patients can return to physical activities and work much earlier.

A randomised trial has compared results in a group of 500 patients from Denmark comparing surgery, RFA, EVLA (laser) and foam sclerotherapy. At one year all treatments were effective but the highest technical failure rate was in patients undergoing sclerotherapy (16%) with the lowest in the surgery and RFA groups (both at 4.8%). Interestingly the mean pain scores after intervention were highest in the EVLA group and lowest in the RFA group with surgery in between. The mean time off work was between three and four days. It is clear that surgery and RFA at least are comparable treatments.
In the UK, National Institute for Health and Care Excellence (NICE) has published the following guideline, 168 (2013).

### For people with confirmed varicose veins and truncal reflux:

- Offer endothermal ablation (RFA) and endovenous laser treatment of the long saphenous vein
- If endothermal ablation is unsuitable, offer ultrasound-guided foam sclerotherapy
- If ultrasound-guided foam sclerotherapy is unsuitable, offer surgery.

Globally, many clinicians are involved in treatment of varicose veins. These include sclerotherapy specialists, dermatologists, appearance medicine practitioners and vascular surgeons. Regardless of whom is the treating clinician, there is always a risk of recurrent varicose veins, up to five to 10 percent in five years.

There are two main reasons for this. Firstly the nature of the disease will always put patients at risk of recurrent veins. It is a life-long inherited tendency and over years new veins can appear whatever the treatment, no matter how carefully performed. Secondly, some techniques if not applied correctly may lead to increased risk of recurrence.

Other complications related to RFA treatment include brown staining of the skin overlying the treated vein, burning of the skin or surrounding tissues and deep vein thrombus (DVT). There is also a one percent failure rate for the procedure, i.e. the treated vein doesn’t completely obliterate following RFA. These complications are also potentially present at a greater extent with open vein surgery.

In my practice I offer RFA treatment to the GSV and/or SSV in appropriate patients. The varicosities can be treated with ultrasound guided sclerotherapy at the same time. Generally, RFA treatment takes about 30–45 minutes per leg. Grade II compression stocking was applied on completion of the procedure. Patients are encouraged to walk around after the procedure and discharged one hour later.

### References

Pelvic Floor Dysfunction – A Hidden Disorder

Wakefield Hospital | Area: Gynaecology
Article written by: Dr Latha Vasan, Gynaecologist, Wakefield Hospital, ph (04) 381 8120

Prolapse of the uterus or vagina is usually the result of loss of pelvic support caused by the stretching and tearing of the endopelvic fascia, the levator muscles and perineal body causing various non-specific symptoms like pelvic heaviness, genital bulge, and difficulties during sexual intercourse, such as pain or loss of vaginal sensation and urinary/defecation disorders.

It may affect over half of women aged 50 to 59 years.

Commonly associated risk factors are described as:

<table>
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<tr>
<th>Predisposing factors</th>
<th>Inciting factor</th>
<th>Promoting factors</th>
<th>Decompensating factor</th>
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<tr>
<td>Hereditary factors, collagen distribution</td>
<td>Advancing parity, increasing weight of the largest baby delivered vaginally, hysterectomy</td>
<td>Obesity, chronic pulmonary disease, smoking, constipation</td>
<td>Ageing-menopause.</td>
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Uterine vault prolapse is often associated with coexistent anterior, posterior vaginal wall prolapse and/or an enterocele.

The commonly associated symptoms of anterior vaginal wall prolapse are urinary frequency, urgency, incontinence, intermittent flow, straining to void, feeling of incomplete bladder emptying and poor stream.

Symptoms associated with posterior vaginal wall prolapse include difficulty in defecation with excessive straining to empty the bowels, feeling of incomplete bowel emptying, constipation and manual evacuation with digital assistance.

Deficiency of knowledge, decreased body image, feeling of shame and embarrassment delay accessing treatment.

Affected women try to isolate themselves (break down of marriage) from social activities when they have urinary or faecal incontinence. Many of them can live in silence due to embarrassment even after having been seen by health practitioners.

“There is now evidence that pelvic floor muscle training is effective in treatment of POP-up to 40% cure rate.”
Management of uterine prolapse has gone through dramatic and drastic twists from the time of Hippocrates (460-377 B.C.E.) until female genital anatomy was clearly understood in early 16th century by Andreus Vesalius, Professor of Anatomy at Padua (Italy).

There is now level one, grade A evidence that pelvic floor muscle training (PFMT) is effective in treatment of POP-up to 40% cure rate.

Dr Arnold Kegel, Gynaecologist, described PFMT as physiological ‘tightening up’ the pelvic floor. The theoretical rationale for intensive strength training of the pelvic floor muscle (PFM) to treat UI and POP is that strength training may build up the structural support for the internal pelvic organs by elevating the levator plate to a permanent higher location inside the pelvis and by enhancing hypertrophy and stiffness of the PFM and its connective tissue. This may increase the urethral resting pressure and the maximum urethral closure pressure, but also facilitate a build up the structural support of pelvic organs. This may increase the maximum urethral closure pressure and its connective tissue. This may increase the maximum urethral closure pressure and its connective tissue.

By the close of the 16th century pessaries evolved as treatment for uterine prolapse. French Royal surgeons used oval shaped pessaries made of brass and waxed corks. Pessaries offer a safe, non-surgical option for the treatment of pelvic organ prolapse and should be offered as an option for patients with bothersome symptoms of prolapse. Various types of pessaries are available with reasonable success rates and minimal complications. Major complications are extremely rare and tend to occur with neglected pessaries. But filling the vagina with such traps cannot be considered as treatment except when surgery is contraindicated or awaiting surgery.

Surgical management of uterine prolapse started as early as second century C.E. Various methods have evolved with time but failure rates are still high. There are currently no evidence-based guidelines to advise clinicians and women as to which surgical intervention is most appropriate.

Anterior colporrhaphy (AC) remains an excellent option for anterior compartment prolapse.

Recurrent prolapse is reduced with the addition of an apical suspending procedure to initial AC.

For apical posthysterectomy prolapse sacral colpopexy has superior outcomes to vaginal suspending surgery (sacrospinous and uterosacral colpopexy and transvaginal mesh) although uterosacral and sacropinous colpopexy are safe and equally effective.

Vaginal hysterectomy and vault suspending procedure is an appropriate and effective option for the management of uterine prolapse. In women needing to retain fertility sacro-hysterepexy is still a good option.

Obliterative vaginal surgery (colpocleisis) is a viable alternative to reconstructive prolapse surgery in those not wishing to retain option of being sexually active and is safe, well-tolerated and effective in elderly cohort.

Major milestones in the surgical management of uterine prolapse are advanced endoscopic surgery and vaginal mesh.

The concept that vaginal prolapse is akin to hernia attracted gynaecologists to adapt mesh to repair these defects since 2004.

Mesh erosion, bleeding, pain, infection, organ perforation and urinary problem can be common to any repair but mesh erosion and organ perforation are mainly attributed to mesh use and trocar placements.

Unlike abdominal hernia repair vaginal prolapse repair is anatomically very different with organ (bladder/rectum) and vaginal epithelium (atrophic and brittle) in close proximity with very little or absent connective tissue-muscular layer (stretched and torn). Placing a mesh in this space has to be precise and the mesh should be as inert as possible to avoid erosion and better integration.

Uncontrolled use of mesh (with inadequate quality control) by surgeons with varying level of experience has led to significant (known) complications and sequelae. Food and Drug Administration and other health authorities have intervened to regulate, audit and control appropriate use of mesh. In the interim most of the Health Boards have temporarily withdrawn the use of mesh for vaginal prolapse repair.

For recurrent prolapse, mesh may be the only option left in the absence of which these women will have to have no option but repeated failed surgeries.

References

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2. Uterine Prolapse: From Antiquity to Today, Keith T. Downing
   - Division of Female Pelvic Medicine and Reconstructive Surgery, Department of Obstetrics & Gynecology and Women’s Health, Albert Einstein College of Medicine/Montefiore Medical Center, Bronx, NY 10461, USA
   - Academic Editor: Hans Peter Dietz.

Dr Latha Vasan

Posterior colporrhaphy (PC) is the procedure of choice for posterior compartment prolapse

Fascial plication at PC is superior to site-specific repair and also is less painful at intercourse as compared to levator muscle plication

Neither biological or synthetic graft have demonstrated improved outcomes when compared to fascial plication

Facial plication is superior to transanal repair of rectocele

Recurrent prolapse is reduced with the addition of an apical suspending procedure to initial PC.
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Specialty
Oral and Maxillofacial Surgery

Background and training
Training in OMFS with specialist registration Medical Council NZ, Dental Council NZ.
Specialist registration 1995.
Gerard works part-time in private practice and has been a visiting consultant at Hutt Hospital since 1996.

Special interests
• Dento-alveolar and wisdom teeth surgery
• Dental implants
• Temporomandibular joint surgery
• Orthognathic (jaw) surgery.

Dr Anil Ranchord
MBChB (Otago), FRACP
Cardiology/Interventional Cardiologist
P: 04 381 8115
F: 04 381 8116
E: heart@whc.co.nz
Anil is a Cardiology/Interventional Cardiologist practicing at the Wakefield Heart Centre, Rintoul Street, Newtown, Wellington. He is also a Consultant Cardiologist at Wellington Hospital and a Clinical Senior Lecturer at Wellington School of Medicine.

Specialty
Cardiology/Interventional Cardiology

Background and training
Interventional Cardiology Fellowships in the USA at the Mid America Heart Institute in Missouri (2011) and Duke University Medical Center in North Carolina (2012), supported by a NZ National Heart Foundation training fellowship.
Advanced training and Research Fellow in Cardiology at Wellington Hospital.
Graduate of the Wellington School of Medicine (Otago University).

Special interests
• Interventional Cardiology:
  Coronary angiography, percutaneous coronary intervention and aortic valvuloplasty
• Heart failure
• Valvular heart disease
• Atrial fibrillation.

Dr Jeremy Lanford
MD
Neurologist
P: 04 381 8115
F: 04 381 8116
E: heart@whc.co.nz
Jeremy is commencing at the Wakefield Heart Centre as a consultant neurologist. Wakefield Heart Centre is located at Wakefield Hospital, from the Rintoul Street entrance, Newtown, Wellington.

Specialty
Neurology

Training
Medical degree at University of Texas Houston Health Science Centre.
Neurology training at University of Virginia.
Neurophysiology/Neuromuscular Fellowship at University of Virginia.

Background
Jeremy is commencing as part of the team at Wakefield Heart Centre as a consultant neurologist. Dr Lanford is originally from the US where he trained at the University of Virginia in neurology and neurophysiology/neuromuscular disorders. He is also working at Capital & Coast District Health Board as a consultant neurologist and clinical stroke leader. Dr Lanford’s main clinical interests include stroke and cerebrovascular diseases along with neuromuscular disorders and general neurological issues. He is now accepting referrals regarding any neurological condition requiring evaluation.

Special interests
• Stroke and Cerebrovascular Disorders
• Neuromuscular Disorders
• General Neurology.
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Save the date:
8 & 9 May 2015
Te Papa

Dr Richard Carroll
MB ChB, FRACP
Endocrinologist
P: 04 381 8120
F: 04 381 8121
E: specialists@wakefield.co.nz
Healthlink: wakespec

Richard is an Endocrinologist who consults at the Wakefield Specialist Medical Centre, 99 Rintoul Street, Newtown, Wellington. He will also commence consulting at the Kapiti Coast in 2015.

Specialty
Endocrinology

Background and training
Richard graduated from the University of Manchester, England before relocating to New Zealand over 10 years ago. He undertook basic physician training in hospitals throughout the greater Wellington region and Sydney, Australia. He completed his specialist training in Endocrinology in Wellington, and the Hammersmith and Charing Cross Hospitals in London, before being awarded FRACP in Endocrinology in 2012. Since then he has worked as a consultant Endocrinologist at Wellington Regional Hospital, and in addition, holds Endocrinology clinics in the Wairarapa and Kapiti Coast.

Special interests
Richard sees patients with the full range of endocrine conditions including thyroid, adrenal, calcium, and sodium disorders, all types of diabetes, and reproductive endocrine disorders (polycystic ovary syndrome, menstrual disorders and related hormonal conditions in women, and testosterone deficiency and replacement in men).

His special clinical and research interests include the management of patients with endocrine tumours including pituitary and gastrointestinal neuroendocrine tumours, genetic and inheritable endocrine disorders such as multiple endocrine neoplasia, the management of endocrine disorders during pregnancy (particularly thyroid disorders), and obesity. He is involved with national and Australasian wide specialist interest and guidelines groups for neuroendocrine, pituitary, and genetic diabetes disorders. In addition, he acts as a medical advisor for a number of groups supporting patients and their families with these conditions.

www.acurity.co.nz/connect
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