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VASCULAR UPDATE

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THE MANAGEMENT OF INTERMITTENT CLAUDICATION

Intermittent claudication is a common symptom of arterial occlusive disease and is experienced by approximately 4% of those aged over 55. Relatively few (2-8%) progress to limb loss and 75% remain symptomatically stable or improve. It is associated with high cardiovascular risk: 50 per cent of claudicants die within 10 years. Peripheral arterial disease is an independent risk factor for cardiovascular death.

Claudication has a major impact on quality of life, similar to that of class 3 or 4 heart failure. Treatment is primarily non-interventional and comprises risk factor control, exercise and pharmacological treatment. Selected patients with incapacitating symptoms may benefit from intervention whether endovascular therapy or surgery.

Non-interventional treatment

1. Risk factor control

Evidence of the benefit of correction of associated risk factors is strong. Smoking is the major risk factor and is associated with a 3x increase in cardiovascular mortality risk. Smoking cessation lowers the mortality rate and whilst there is no strong evidence that this will improve symptoms, the risk of developing critical limb ischaemia is reduced.

In patients with established atheromatous vascular disease, aspirin reduces cardiovascular events significantly (27% relative risk reduction). The addition of clopidogrel may confer an additional benefit (relative risk reduction of 9 % compared to aspirin).

Lipid lowering drugs such as statins benefit patients at risk of cardiovascular events. The benefit is greatest for patients with the highest cardiovascular risk regardless of baseline cholesterol level. Claudicants form one of the highest-risk groups and so statin therapy is justified. Cardiovascular mortality can be further reduced by the use of ACE inhibitors.

2. Exercise

Exercise programmes address not only the presenting symptom but may also reduce cardiovascular risk. A meta-analysis concluded that exercise can improve walking ability by approximately 150%. There is no clear evidence about the optimum form of exercise, but maximum benefit seems to be associated with supervised walking programmes, at least three times per week over 6 months.

3. Pharmacological treatment

Clinical trials have suggested improved walking ability from drugs such as pentoxifylline and the phosphodiesterase-III inhibitor, cilostazol. The clinical benefit is generally small and it is not clear if these agents are cost-effective.

Interventional treatment

1. Angioplasty

Angioplasty (PTA) is frequently performed for moderate to severe claudication. Results vary considerably, but 90-100 per cent primary success rates with 60-80 % 1-year patencies have been reported for short superficial femoral artery occlusions. Successful angioplasty improves the quality of life of claudicants. Percutaneous intervention for iliac lesions is associated with a better outcome than superficial femoral and popliteal lesions. Tibial angioplasty is seldom appropriate for claudication alone as the risks of intervention are increased. Only 2

randomized studies comparing PTA with exercise have been reported and both failed to show any long-term advantage to angioplasty. PTA, although less invasive than surgery, has a small complication rate. Patients who are offered intervention must understand this risk clearly. Peripheral angioplasty should only be undertaken by specialists with appropriate training and experience.

2. Surgery

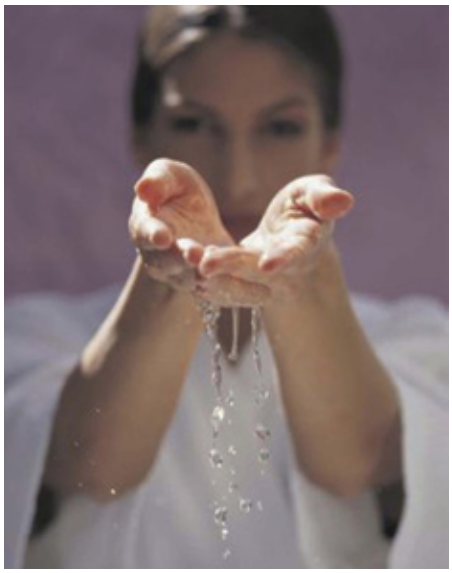
A large number of bypass procedures are undertaken for claudication in spite of concerns over associated mortality and morbidity (including limb loss if the graft fails) and cost. In the largest randomized study of intervention in claudicants, surgery improved walking distance and quality of life significantly more than supervised exercise or controls. No excess mortality or limb loss was found in the surgery group and ankle arterial pressure was significantly increased following intervention. As surgery is undertaken to improve quality of life for a condition that has a benign natural history it is essential that surgical intervention be performed at extremely low risk by surgeons with the necessary training and experience. **PM**

ENDOSCOPIC **SYMPATHECTOMY**

Sympathectomy of the upper limb is a well described technique. The act of dividing the sympathetic nerves was one of the earliest operations done for vascular insufficiency, and remained a standard open operative procedure until very recently.

Upper limb sympathectomy has been revolutionised by the application of minimally invasive techniques, and today offers a superb alternative to the open procedure in terms of cosmesis, effectiveness and reduction of morbidity.

Thoracoscopic sympathectomy is indicated for primary hyperhidrosis of the hands and axilla, digital ischaemia not treatable by revascularisation, some cases of Raynaud's syndrome, vasomotor disorders and causalgia. Its use has been described for the management of uncontrollable facial flushing, but this is not a generally recognised indication.



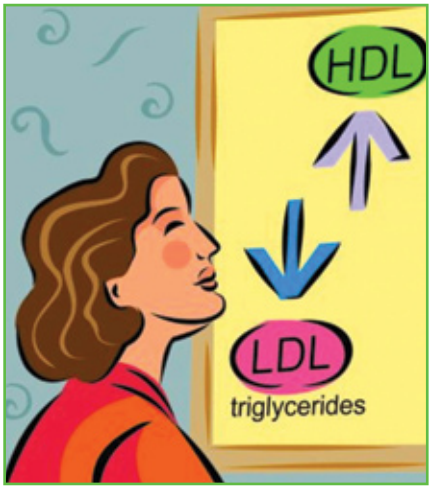
The procedure is performed via two tiny incisions in each axilla. A camera is inserted into the thoracic cavity, the lung is collapsed and the sympathetic ganglia identified on the posterior wall of the thorax. The second and third ganglia (for palmar hidrosis) and the fourth ganglion (axillary hidrosis) are then destroyed by diathermy. The lung is re-inflated, the instruments removed and the wounds sutured. The other side can then be dealt with if required.

Compensatory sweating (sweating from another area after the operation) is a potential complication that can be a nuisance and is unpredictable. It is not reversible. Other side effects include gustatory sweating (facial sweating on eating), neuralgic pain in the chest wall or arm, Horner's Syndrome (occurs in <1% of patients treated this way as opposed to >30% of patients undergoing open sympathectomy) and recurrent sweating..

Patients need VERY careful counselling prior to this procedure as it is irreversible, and some of the side effects are very unpleasant. However, in the right patient, it offers a superb treatment option of a very difficult medical problem with very low complication rates, and a very high degree of patient satisfaction. **JT**

THE ROLE OF STATINS IN THE MANAGEMENT OF VASCULAR DISEASE

The use of statins is now universally accepted as being vital to optimal care of the vascular patient. Their use improves outcomes and long term survival. Patients on statins have been shown to experience less recurrent stenosis following carotid endarterectomy, stenting and angioplasty, and reduced numbers of cardiac events following cardiac and non-cardiac vascular surgery. Aneurysmal change has also been reduced in patients receiving statins. It is therefore very surprising that statins are only used in 35-67% of vasculopaths.



Serum lipid concentration is composed of several fractions including HDL ("good") and LDL ("bad"). In general, a high HDL level is cardioprotective. In the Framingham Study, myocardial infarction rates increased by 25% for every 5mg/dl decrease in HDL below the median value. Total cholesterol may be normal in these patients. Lipoprotein (a) is another lipid fraction shown to be associated with increased risk. Triglycerides are predictive of increased event rates and mortality in patients when elevated.

Statins work by inhibiting HMG-CoA reductase and are the most effective lipid therapy at lowering LDL cholesterol. Not all the statins are created equal. The percentage reduction of LDL levels is more important than the absolute level measured. Rosuvastatin (Crestor) and Atorvastatin (Lipitor, Aspavor) are most effective, and are the agents of choice for those patients requiring a >30% reduction in LDL levels. Rosuvastatin is the most effective at increasing HDL levels while Rosuvastatin and atorvastatin are the most effective at reducing Triglyceride levels.

TARGETS FOR CHOLESTEROL	
Total	< 5 mmol/l
LDL	< 3 mmol/l
HDL	> 1 mmol/l
Fasting Triglyceride	< 2 mmol/l

Water soluble statins (Pravastatin, Fluvastatin) are less likely to have side effects than the lipid soluble agents, and may also have better plaque stabilisation characteristics. Pravastatin is the only statin metabolised in liver and kidney, making Pravastatin the agent of choice in patients with renal dysfunction.

Myalgia occurs in less than 2% of statin users, and myositis in less than 0.5%. Acute renal failure occurs in <0.1%. The mechanism of muscle injury with statins is unclear. The symptoms may take months to develop after commencement of therapy. Neuropathy has been estimated to occur in 1: 2200 patient years of statin use.

In summary, statins have a very positive effect on the conservative and operative management of vascular disease, and do so with very acceptable side effect and complication rates. **JT**

PRESCRIBED MINIMUM BENEFITS (**PMB's**)

What is a Prescribed Minimum Benefit (PMB)?

The council for medical schemes has identified 270 treatment pairs and 25 chronic diseases, which by law must be covered for by the member's medical scheme regardless of the scheme option the member has selected. This applies to both in and out of hospital treatment. They were introduced to make sure that patients who were on private medical schemes were not without care for certain medical conditions because they could not afford it or they had run out of benefits forcing them to be treated at an already over-burdened public sector. Medical Schemes must pay for these condition even if:

1. There are scheme exclusions
2. Waiting periods have been applied to the membership
3. Limits have been exhausted
4. and for outpatient treatment even for patients with hospital plans only.

Funding for Prescribed Minimum Benefits (PMB's)

If a patient is diagnosed with a PMB condition, the medical scheme is obliged by law to cover all costs of the treatment at the doctor's usual billing rate and not at the scheme's rate. The funds for these treatments must come from the scheme's risk pool and not from the members' medical savings.

Designated Service Providers (DSP's)

Some medical schemes have chosen Designated Service Providers, in the form of General Practitioners, Specialists, Hospital Networks and Pharmacies to be the first choice of service when its members need diagnosis, treatment and care for a PMB condition.

In the event of a patient voluntarily going to a non-DSP for a PMB condition, the scheme will charge them a co-payment. This penalty is only in the event of voluntary use of a non-DSP. However, the scheme may not penalize a member for voluntarily making use of a non-DSP if:

1. There is no DSP in your area or
2. The service is not available at the chosen DSP or
3. The service could not be provided without unreasonable delay

Should a patient suffer from an emergency condition, he or she may go to your nearest healthcare provider for treatment even if it is not the scheme's DSP.

Non-Designated Service Providers

Some medical schemes have opted not to elect DSP's for their members, allowing them to choose a health professional of their choice. The scheme will cover all cost for PMB treatment at an ethical rate.

Here at Matley and Partners we have introduced a dedicated PMB department for our patients. At the time of consultation or emergency admission into hospital, it will be determined whether or not they have a PMB condition. If they do, the patient will informed as such, we will see that they are correctly registered with their medical scheme for PMB cover and we will follow up on a daily basis with their medical scheme to make sure that they are refunded all their treatment costs in full. **KL**

We hope that the service we offer at Matley and Partners will be of great help to our referring doctors and to our patients, ensuring the best possible healthcare. **Kim Lambley** is the person in charge of PMB affairs. She can be contacted directly on **021-683 3893.**

GARRON CAINE TRAVELLING FELLOWSHIP

Bob Baigrie (Consultant, Colorectal firm, GSH), Claire Warden and Del Kahn (Head of Surgery, GSH)

The Garron Caine Travelling Fellowship was established by this practice 10 years ago. It is awarded annually to a Registrar or Junior Consultant in the Department of Surgery at the University of Cape Town. This year it was awarded to Dr Claire Warden who was able to visit two colorectal units in the UK and to attend the Association of Coloproctology Great Britain and Ireland Congress held in Bournemouth in June 2010.

Her itinerary was facilitated by Bob Baigrie, who arranged for her to visit close UK colleagues involved with cutting edge colorectal surgery. She spent time in theatre at Manchester Royal Infirmary and The Churchill Hospital, Oxford, observing laparoscopic rectopexies and other interesting surgery. She also visited the Intestinal Failure Unit at Salford Hospital and learnt from their experience of complex fistulae and small bowel transplants.

The highlight was presenting Groote Schuur's local data on colonic stents at the congress. A multicentre randomized control trial on colonic stents (CREST) is currently underway in the UK and the topic was thus of particular relevance to the audience. It was fascinating to compare local experience to that in the UK and realise that we are delivering a world class colorectal service at Groote Schuur.

DIABETES: **A VASCULAR DISEASE**

Diabetes and Coronary Artery disease

IHD and other heart disease accounts for 55% of deaths in diabetics. The rate of acute myocardial infarction is 2-3x that of non diabetics and the rate of congestive cardiac failure 2-5 x higher. Diabetes is a coronary heart disease equivalent which means that patients with diabetes and no previous coronary events are at as high risk of developing a coronary event as a non-diabetic with a previous heart attack. The coronary arteries are often more diffusely involved in diabetics and the infarcts more extensive. They present in cardiogenic pulmonary oedema more often which is a poor prognostic indicator. Diabetics have double the chance of death within the 1st 42 days post MI and an increased risk of developing CCF. Diabetics also have an increased risk of developing restenosis post angioplasty or stent.

Diabetes and Stroke

Diabetes is a major risk factor for stroke and the incidence of ischemic stroke is at least 2.5 x higher in patients with diabetes. Stroke is responsible for 10% of deaths in diabetics. The mortality and severity of stroke is also higher among patients with diabetes. Hyperglycaemia is toxic to infarcted brain tissue and impairs autoregulation and vasodilatation, resulting in decreased cerebral blood flow. Anaerobic metabolism of glucose leads to increased pH and cell death. This is proportional to the serum glucose level.

Diabetes and Peripheral Vascular Disease

The risk of a lower limb amputation in a diabetic patient is 40x that of a non diabetic patient.

The arteries in a diabetic are affected by 2 disease processes, namely atherosclerosis and (Monckenberg's) medial sclerosis. Atherosclerosis causes ischaemia by narrowing and blocking the arteries. Medial sclerosis is calcification of the media of the artery and causes rigid vessels without narrowing the lumen. It does not cause ischaemia, but does interfere with the measurement of doppler pressures as it results in falsely high readings.

20-40% of patients with diabetes have peripheral vascular disease and up to 50% of patients with a foot ulcer have signs of PVD. Ischaemia secondary to peripheral vascular disease is the most important factor related to the outcome of a diabetic foot ulcer. There are no atherosclerotic lesions specific to diabetes, although the pattern of atherosclerosis is slightly different.

The most noticeable differences compared to non-diabetics are:

- affects younger patients
- tends to affect smaller vessels below the knee with relative sparing of aorto-iliac segments
- sparing of foot vessels
- more aggressive
- faster progression
- no sex difference

The presence of peripheral vascular disease in diabetics is related to the traditional vascular risk factors including smoking, hypertension, older age and hyperlipidemia. The level of glycaemic control has also been shown to play a role with a 26% increase in the risk of PVD for every 1% increase in HbA1c. End stage renal disease is also a risk factor.

The majority of patients with clinically detectable PVD are asymptomatic. Less than 25% with significant disease report intermittent claudication. End-stage symptoms are ischaemic rest pain (especially at night) and ulceration/gangrene. Many of these patients, however, may not experience any pain despite extensive tissue loss because of sensory loss due to their peripheral neuropathy.

One of the most reliable signs of significant vascular disease in a diabetic patient is the absence of a foot pulse. Any patient with an absent pulse and tissue loss needs a formal vascular assessment. **MF**