VOLUME 13 #2 OCTOBER 2009 www.surgcare.co.za DR MATLEY & PARTNERS

VASCULAR UPDATE

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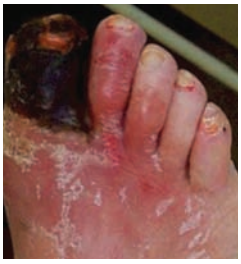
FIVE THINGS YOU SHOULD NEVER DO TO A VASCULAR PATIENT

Errors in the primary care of patients with peripheral vascular problems frequently result in serious consequences. Five of the most common errors we see are the following:

NEVER TAKE A TOENAIL OFF WITHOUT FIRST CHECKING THAT THERE ARE FOOT PULSES



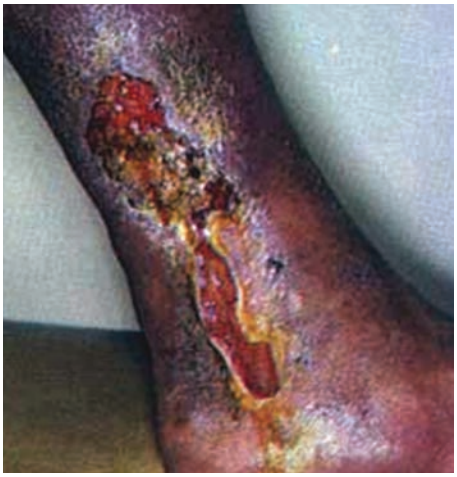
Ingrown toenails and chronic sub-ungual fungal infections are common. Nail avulsion is seldom appropriate management for these conditions. Toe pain is invariable when feet are critically ischaemic but usually the toenails are not the cause. Avulsing a toenail in a critically ischaemic foot invariably leads to disaster, with the frequent development of gangrene and a high risk of major limb amputation if the limb is not urgently revascularised. One should never perform a toenail avulsion without first determining that the foot pulses are palpable. If they are not, the patient should be referred for a full vascular assessment including ankle Doppler pressures before any procedure is performed on the toenail itself.

NEVER MERELY GIVE DIURETICS WHEN LOWER LIMB OEDEMA IS UNILATERAL

Unilateral lower limb oedema usually indicates a local problem within that leg. Most cases are due to venous or lymphatic dysfunction. Venous causes include deep vein thrombosis, iliac venous compression by tumours or nodes, the May-Thurner syndrome where the left iliac vein is compressed by the adjacent artery and all forms of deep and superficial valve incompetence. Rarer causes include AV fistulae, soft tissue tumours and bone tumours. Primary lymphoedema is common and secondary cases usually result from attacks of cellulitis or lymphatic obstruction in the groin following surgery and radiotherapy. It is therefore never appropriate to merely administer diuretics. These patients all require investigation.

NEVER FAIL TO PROVIDE COMPRESSION FOR A VENOUS ULCER

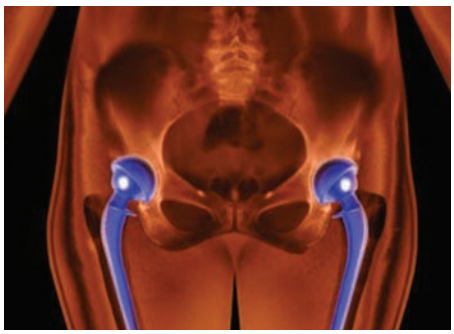
Venous ulcers are extremely common and easily recognized as they tend to involve the medial aspect of the calf or ankle and are associated with other features of venous insufficiency such as oedema, varicose veins, hyper-pigmentation and lipo-sclerosis. The most important aspect to the management of these patients is the provision of adequate graduated compression. This reverses the altered haemodynamics that are the basis of chronic venous insufficiency. Compression improves venous return and eliminates oedema. With adequate compression, virtually all venous ulcers will heal. The choice of topical treatment or dressing is far less important. There is evidence that a four-layer bandage technique is the most successful. We use a zinc bandage directly on the ulcer followed by velband followed by a compression bandage aiming for an ankle pressure of 40mmHg. Finally a cohesive bandage is applied to keep it all together and provide support. Beyond compression it is important to recognize that the large ESCHAR trial has proven that superficial venous surgery is indicated in any venous ulcer patient



where superficial (long or short saphenous) incompetence exists irrespective of the state of the deep veins. This may accelerate ulcer healing and will definitely reduce the risk of recurrence. It follows that all leg ulcer patients require a non-invasive venous assessment to determine whether superficial venous incompetence is a factor..

NEVER FORGET TO CHECK THE FEMORAL PULSE IN A PATIENT WITH HIP PAIN

Common Iliac arterial disease is very common and frequently results in buttock or hip pain. This tends to be experienced after walking a certain distance and is immediately relieved by rest. Patients



(FIVE THINGS YOU SHOULD NEVER DO TO A VASCULAR PATIENT continued from front page...)

have frequently already concluded that they have a hip problem. The femoral pulse will usually be weak or absent in these patients and an abdominal or femoral bruit is common. In males, erectile dysfunction is often present as well. It is important not to miss the patient whose apparent hip or buttock pain is due to arterial insufficiency as this is usually easily treated by standard vascular surgical techniques including iliac angioplasty or stenting. A hip replacement is not a good treatment for iliac arterial occlusion! Always feel the femoral pulse carefully in a patient presenting with hip or buttock pain.

NEVER SIMPLY REASSURE A PATIENT WITH ACUTE CALF PAIN WITHOUT INVESTIGATION.

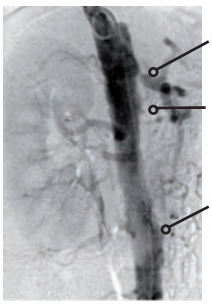
Distinguishing deep vein thrombosis from all the other causes of acute calf pain is usually impossible without a duplex scan. On occasions a d-dimer estimation may be helpful. Other causes of acute calf pain include calf muscle tears (which can result from episodes of nocturnal myoclonus which the patient may not recall), cellulitis, ruptured baker's cysts, superficial thrombo-phlebitis, spider bites, tibial stress fractures and acute arterial occlusion. The usual clinical signs of a DVT are unreliable and "Homans sign" should never be performed as it is both painful and un-informative. The consequences of missing a DVT are serious. All patients with acute calf pain need a duplex scan or d-dimer estimation before they can be reassured. ***PM***

MESENTERIC ISCHAEMIA

Mesenteric ischaemia is defined as primary failure of blood supply to the intestine (as opposed to strangulation of a segment of gut by mechanical means). It is uncommon, but must be recognised and treated promptly to avoid major complications or even death.

Acute mesenteric ischaemia can be extremely difficult to diagnose, and the resulting delay in diagnosis and treatment often leads to poor outcomes. Chronic mesenteric ischaemia is notoriously under-diagnosed, and is often only diagnosed very late in its course. Patients are thus often severely malnourished at the time of diagnosis and this, combined with their multiple co-morbidities, means that surgery carries high risks of morbidity and mortality.

Aortic Angiogram showing Visceral Disease

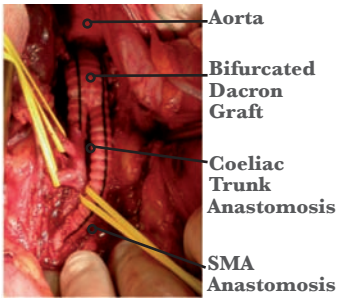


Acute mesenteric ischaemia is caused by acute occlusion of the superior mesenteric artery (SMA). This may be a new occlusion (embolus, dissection), or an acute-on-chronic thrombosis of a stenosed SMA. This presents as **severe, unremitting, central** abdominal pain. Early on, there is a remarkable **lack of abdominal signs**. (This is virtually pathognomonic of acute SMA occlusion). Diagnosis at this stage allows intervention before bowel necrosis is established, and is associated with a much better prognosis. Diagnosis once peritonism is present implies that bowel necrosis is established, and mortality rates rise dramatically from this point.

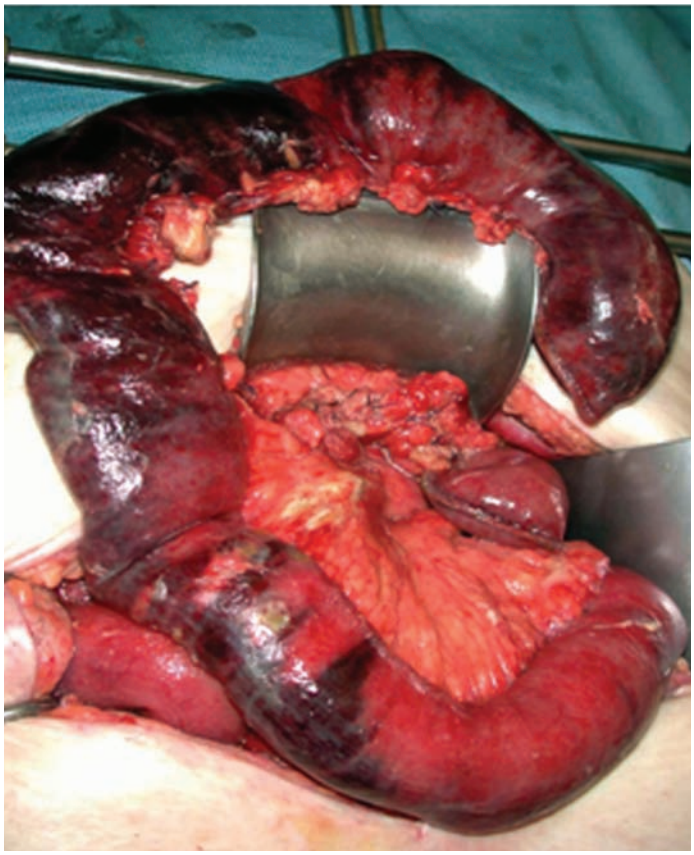
The diagnosis is established most reliably and rapidly by means of CT angiography.

Management includes rapid reperfusion of the ischaemic gut, resection of non-viable bowel with anastomosis to maintain continuity, anticoagulation, and supportive measures. (Antibiotics, nutritional support etc.)

Surgical Visceral Bypass



Chronic Mesenteric Ischaemia occurs when there is a gradual stenosis of the feeding vessels of the gut. Symptoms do not occur unless at least 2 of the 3 named feeding vessels are occluded and the third stenosed. The presenting symptoms are **mesenteric angina, food fear** (anorexia), and **weight loss**. The differential diagnosis includes GIT malignancy, pancreatic dysfunction, and functional bowel symptoms. Investigations for these problems can be very time consuming, and accounts for the frequently delayed diagnosis. There needs to be a high index of suspicion, especially in elderly patients with multiple vascular risk factors.



If suspected, the diagnosis can be confirmed with Duplex Doppler, or – more reliably – with CT angiography.

Management requires reperfusion of the ischaemic gut. This is achieved with endovascular procedures (stenting or angioplasty of SMA and/or Coeliac trunk) or open surgery. The surgical reconstruction usually requires a bifurcated graft from Aorta to SMA and Coeliac trunk and carries a 10% mortality rate. Endovascular procedures have dramatically decreased the mortality and morbidity rates, and are now the preferred method of treatment. ***JT***

DVT PROPHYLAXIS: WHO, WHY AND HOW ?

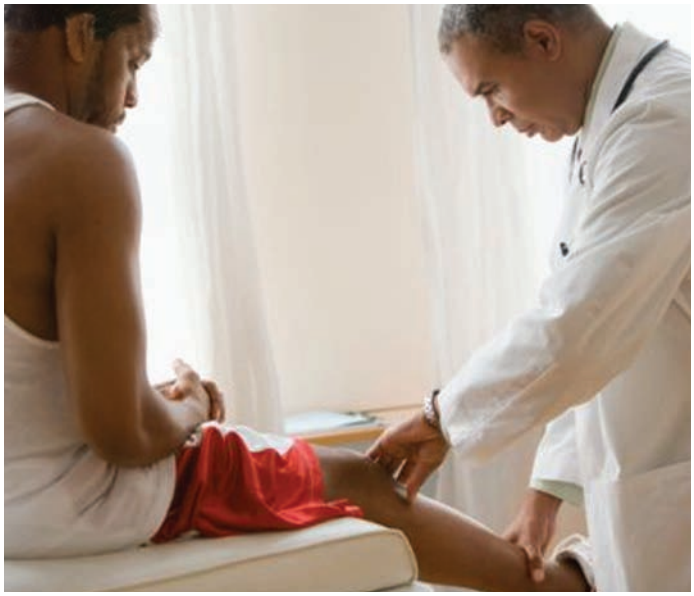
The rationale for DVT prophylaxis is well established. Pulmonary Embolism is estimated to cause at least 10% of all hospital deaths. This number can be reduced dramatically by appropriate measures.

WHO ?

The risk factors for DVT can be grouped according to degree of risk. Some factors are patient related, others are disease related, and others are "environmental". All these need to be considered in deciding on **who** to give prophylaxis to, for **how long**, and **what** to use. The risk factors are cumulative, and the necessity for prompt commencement of prophylaxis increases exponentially with increasing risk.

Table 1: Risk Factors for Deep Vein Thrombosis

surgery	acute medical illness
trauma	heart or respiratory failure
immobility, paresis	nephrotic syndrome
malignancy	myeloproliferative disorders
cancer therapy	obesity
previous VTE	smoking
increasing age	thrombophilia
pregnancy/post partum	central venous catheterization
estrogen containing oral contraceptive	selective estrogen receptor modulators



WHY ?

Table 2 gives a good guide to the risk stratification for DVT in In-Patients. This applies **regardless** of the presumed length of hospital stay. It thus applies to **day case** patients as well as longer stay patients. This is particularly relevant to patients coming in to hospital for **endoscopic** gynaecologic al procedures, as well as **arthroscopic** procedures (particularly if these are to be performed with tourniquet haemostasis.)

HOW ?

Table 2: Risk Stratification For DVT

	DVT, %		PE, %	
Level of Risk	Calf	Proximal	Clinical	Fatal
Low Risk Minor surgery pts < 40 y.o no additional risk factors	2	0.4	0.2	<0.01
Moderate Risk Minor surgery in pts with additional risks Surgery in pts 40-60 y.o no additional risks	10-20	2-4	1-2	0.1-0.4
High Risk Surgery in pts > 60 y.o Age 40-60 y.o with additional risks (prior VTE, cancer, thrombophilia)	20-40	4-8	2-4	0.4-1
Highest Risk Surgery in pts with multiple risk factors Hip or knee surgery Major trauma Spinal cord injury	40-80	10-20	4-10	0.2-5

The options for prophylaxis include **mechanical** (TED stockings, calf compressors, sequential compression devices, foot compressors), or **pharmacological** (unfractionated Heparin, LMWH, Rheomacrodex, Fondaparinux, Warfarin.) The prophylactic regime can thus be tailored very carefully to suit both Patient and Doctor, and reduce significantly the risks of DVT and PE.

With this degree of adaptability, there really should be **no excuse** for failing to utilise effective prophylaxis before, during, and after hospitalisation. It is perhaps wise to discuss this aspect of care with patients prior to their admission, in order that suitable options can be considered and implemented. It must be noted that prophylaxis may be extended for up to 6 weeks after discharge following Total Hip Replacement or Hip Fracture surgery. ***JT***

WHAT'S HAPPENING AT MATLEY & PARTNERS?

PHIL MATLEY has been elected as President of the Vascular Society of Southern Africa (VASSA) and has been appointed as Course Director of the Crossroads Institute of South Africa. The Crossroads Institute is an international cardiovascular education foundation based in Brussels. In May 2009 he presented a paper at Euro-PCR, an international cardiovascular congress in Barcelona. The title of the paper was "Duplex-guided angioplasty of the SFA and Popliteal arteries" and was based on original research conducted at the Kingsbury Vascular Unit in conjunction with our chief vascular technologist, Greta Blacker.

JAMES TUNNICLIFFE has been appointed to the executive committee of VASSA.

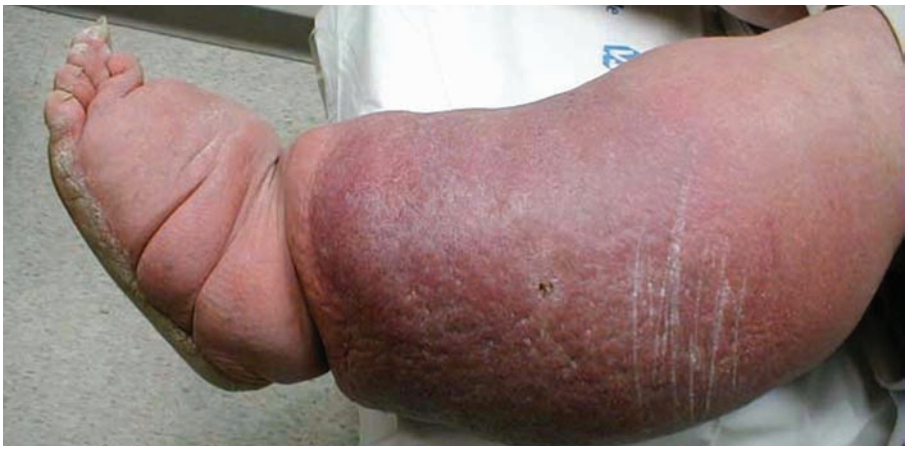
MARTIN FORLEE has been invited to join the speaker faculty of the Crossroads Institute of South Africa.

BOB BAIGRIE has been elected President of the South African Society of Endoscopic Surgeons (SASES).

We are pleased to announce that we will be taking on a new partner in early 2010. He is **MARC HEWAT**, a specialist Hepato-biliary and pancreatic surgeon. Marc is currently working in one of the busiest Hepato-biliary departments in the UK, at the Freeman Hospital in Newcastle-on-Tyne.

PETER JEFFERY is enjoying his retirement on the family farm in Stellenbosch but still assists the vascular partners in theatre from time to time.

DIAGNOSTIC APPROACH TO THE PATIENT WITH A SWOLLEN LIMB



History

Key elements of the history include

- What is the duration of the oedema (acute vs. chronic)? If the onset is acute, deep vein thrombosis should be strongly considered.
- Is the oedema painful? Deep vein thrombosis and reflex sympathetic dystrophy are usually painful. Chronic venous insufficiency can cause low-grade aching. Lymphoedema is usually painless.
- What drugs are being taken? Calcium channel blockers, prednisone, and anti-inflammatory drugs are common causes of leg oedema.
- Is there a history of systemic disease (heart, liver, or kidney disease)?
- Is there a history of pelvic/abdominal neoplasm or radiation?
- Does the oedema improve overnight? Venous oedema is more likely than lymphoedema to improve overnight.
- Is there a history consistent with sleep apnoea? Sleep apnoea can cause pulmonary hypertension, which is a common cause of leg oedema. Findings that may increase suspicion of sleep apnoea include loud snoring or apnoea noted by the sleep partner, daytime somnolence, or a neck circumference > 43 cm.

Physical Examination

Key elements of the physical examination include

- Body Mass Index. Obesity is associated with sleep apnoea and venous insufficiency.
- Distribution of oedema: unilateral leg oedema is generally due to a local cause such as deep vein thrombosis, venous insufficiency, or lymphoedema. Bilateral oedema can be due to a local cause or systemic disease, such as heart failure or kidney disease. Generalized oedema is due to systemic disease. The dorsum of the foot is spared in lipedema but prominently involved in lymphoedema.
- Tenderness: deep vein thrombosis and lipedema are often tender. Lymphoedema is usually nontender.
- Pitting: deep vein thrombosis, venous insufficiency, and early lymphoedema usually pit. Myxedema and the advanced fibrotic form of lymphoedema typically do not pit.
- Varicose veins: leg varicosities are often present in patients with chronic venous insufficiency, but venous insufficiency can occur without varicose veins.
- Kaposi-Stemmer sign: inability to pinch a fold of skin on the dorsum of the foot at the base of the second toe is a sign of lymphoedema.
- Skin changes: a warty texture (hyperkeratosis) with papillomatosis and brawny induration are characteristic of chronic lymphoedema. Brown haemosiderin deposits on the lower legs and ankles are consistent with venous insufficiency. Reflex sympathetic dystrophy initially leads to warm tender skin with increased sweating. Later the skin is thin, shiny, and cool. In the chronic stage, the skin becomes atrophic and dry with flexion contractures.
- Signs of systemic disease: findings of heart failure (especially jugular venous distension and lung crackles) and liver disease (ascites, spider hemangiomas, and jaundice) may be helpful in detecting a systemic cause.

Differential Diagnosis of Swollen Limb

Acute (< 72 hrs)	Chronic	
	Systemic Causes	*Local Causes*
Deep vein thrombosis Cellulitis Superficial thrombophlebitis Ruptured bakers cyst Trauma/haematoma Acute arterial occlusion Insect bite	Cardiac failure Renal failure Hepatic failure Hypoproteinemia Hypothyroidism Pulmonary hypertension Allergic disorders Idiopathic cyclic oedema Hereditary angio-oedema Drugs *antihypertensives* *steroids* *anti-inflammatories* *mono-amine oxidase inhibitors*	Chronic venous insufficiency Lymphoedema Lipedema Congenital Vascular Malformation Arteriovenous fistula Trauma Infection/inflammation Paralysis and dependancy Soft tissue tumour Post re-vascularization oedema Malignant infiltration

Diagnostic Studies

Laboratory Tests. Most patients over age 50 with leg oedema have venous insufficiency, but if the aetiology is unclear, the following tests will help rule out systemic disease:
Full blood count
Urinalysis
Electrolytes, creatinine
Blood sugar
Thyroid-stimulating hormone
Albumin. A serum albumin below 20 mg/L often leads to oedema and can be caused by liver disease, nephrotic syndrome, or protein-losing enteropathy

Imaging Studies.

- Duplex doppler examination is the investigation of choice to rule out deep vein thrombosis or chronic venous insufficiency
- Patients over age 45 with oedema of unclear aetiology should have an echocardiogram to rule out pulmonary hypertension.
- Lymphoscintigraphy can be helpful to distinguish lymphoedema from venous oedema and to determine the cause of lymphoedema. ***MF***