DR. JEFFERY AND PARTNERS

VASCULAR UPDATE

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AIR-TRAVEL RELATED VENOUS THROMBO-EMBOLISM

Deep vein thrombosis and pulmonary embolism associated with air travel has received *a* ***lot*** **of recent** ***publicity. A class*** **action** ***lawsuit against*** **certain** ***Australian airlines and air authorities involving large numbers*** **of** ***patients has particularly highlighted the issue. This*** **is** ***however*** **not** *a* ***new problem.***

The association between sitting in cramped conditions and fatal pulmonary embolism was first described in civilians sheltering in air-raid shelters during the Second World War.
The relationship with prolonged air travel became increasingly recognized during the 1950's and 1960's. In 1974 President Richard M Nixon developed a DVT during a flight to the Soviet Union. He was anti-coagulated but continued the trip, suffering subsequently a number of recurrences and episodes of pulmonary embolism. His hospitalisation for treatment prevented him from testifying in the Watergate trial. A 1986 study based at Heathrow found that pulmonary embolism was the second most common cause of sudden death among long distance passengers. **In 1996** a study from Hawaii noted that 17% of all patients treated in hospital for venous thrombo-embolism had developed symptoms during or after a flight.

The real risk of air-travel induced symptomatic venous thrombo-embolism for adults over 40 has been estimated at 0.004% per traveller per flight. Sub-clinical clots are probably 25 times more common at about 0.01 %. Patients with risk factors for thrombo-embolism are clearly at greater risk than this. Important risk factors are a history of previous DVT or PE, age over 70, malignancy, hypercoagulable states, oestrogens, recent major surgery, congestive heart failure, severe varicose veins and leg oedema.

Approximately half of all cases can be demonstrated to have an underlying hyper-coaguable state. The most common of these are the Factor V leiden mutation, methyl tetrafolate reductase deficiency leading to high levels of plasma homocysteine and the prothrombin gene mutation. In the remainder there is no obvious underlying cause. The "economy class syndrome" is now well recognized: The conditions of poor, ill-designed cramped seating in subjects provided with excessive quantities of free alcohol, in a dehydrating atmosphere of low humidity and hypoxia who are discouraged from exercising by being exhorted to wear seat belts at all times undoubtedly contributes to the increased risk of thrombosis. Even the more expensive business class seats ensure that maximum pressure is applied to the back of the calves during sleep.
A number of publications have suggested that increased fluid intake, calf exercise and avoidance of alcohol reduces the risk

Sleeping tablets should be avoided. Compression stockings and subcutaneous heparin have all been recommended for subjects at increased risk.
There is, however no evidence that low dose aspirin will be helpful. Aspirin prevent the platelet-rich thrombi found in arteries but has little impact on venous thrombosis, which is largely driven by clotting factors rather than platelets.
In 1995 the Vienna Consensus Conference on Travel Thrombosis produced a consensus guideline. Below knee compression stockings (Grade i, ii) were recommended for all patients over 40 with a planned flight exceeding 5 hours duration. This was particularly so for obesity, heart failure, oestrogen use and varicose veins or leg oedema. High risk patients with previous thrombo-embolism, malignancy, known thrombophilia or recent (4 weeks) major surgery were recommended to have 40 mg of

enoxaparin (Clexane) or 5000 iu deltaparin (Fragmin) 2 hours before flying. We agree entirely with these recommendations.
The first UCT Vascular Symposium was held in April at Kingsbury Hospital and the Sports Science Centre, Newlands. International speakers included Professor Michael Horrocks from Bath, UK and Mr Jonathan Beard from the Sheffield Vascular Institute. The meeting commemorated the life and work of Robert Goetz, one of the twentieth century's foremost innovators in vascular surgery who died recently. He spent over 20 years in the Surgery Department at UCT and established the first vascular studies unit in he world at Groote Schuur Hospital. A day of live cases and simulations enabled 20 delegates to participate in endovascular repair of abdominal aortic aneurysms using stent grafts. This was made possible by Kingsbury's state of the art audio-visual unit, which provides a real-time audio and video link of exceptional quality between the vascular theatre and the Kingsbury House seminar room.

On the following day twenty papers were delivered to a much larger audience by a combination of the international guests and a local faculty. Subjects included endovascular aneurysm repair, advances in peripheral and renal angioplasty and stenting, carotid endarterectomy and stenting, thrombolysis, the management of acute stroke, thoraco-abdominal aneurysms, surgery for critical limb ischaemia and new concepts in exercise physiology.

The meeting proved to be an enormous success. We plan to make it an annual event with the next meeting in February 2002.

Kingsbury hosts Vascular Congress

International speakers with the local organizing committee:
James Tunnicliffe, Michael Horrocks, Peter Jeffery, Jonathan Beard, Philip Matley.

Minimally invasive treatment of peripheral aneurysms

Stent grafts for abdominal aortic aneurysms are now well established. Modern vascular stent technology now permits the treatment of a **wide variety of complex peripheral aneurysms using covered stents introduced percutaneously. A number of recent cases at Kingsbury Hospital illustrate this:**

Figure 1 illustrates a 6cm traumatic aneurysm of the proximal left subclavian artery arising 10mm from the thoracic aorta. Access for open repair here would have been extremely difficult; and would have required a thoracotomy. The "aneurysm was treated using a Haemobahn endoprosthesis introduced through the brachial artery. which reconstituted the subclavian artery and excluded the aneurysm **(Fig 2)**.

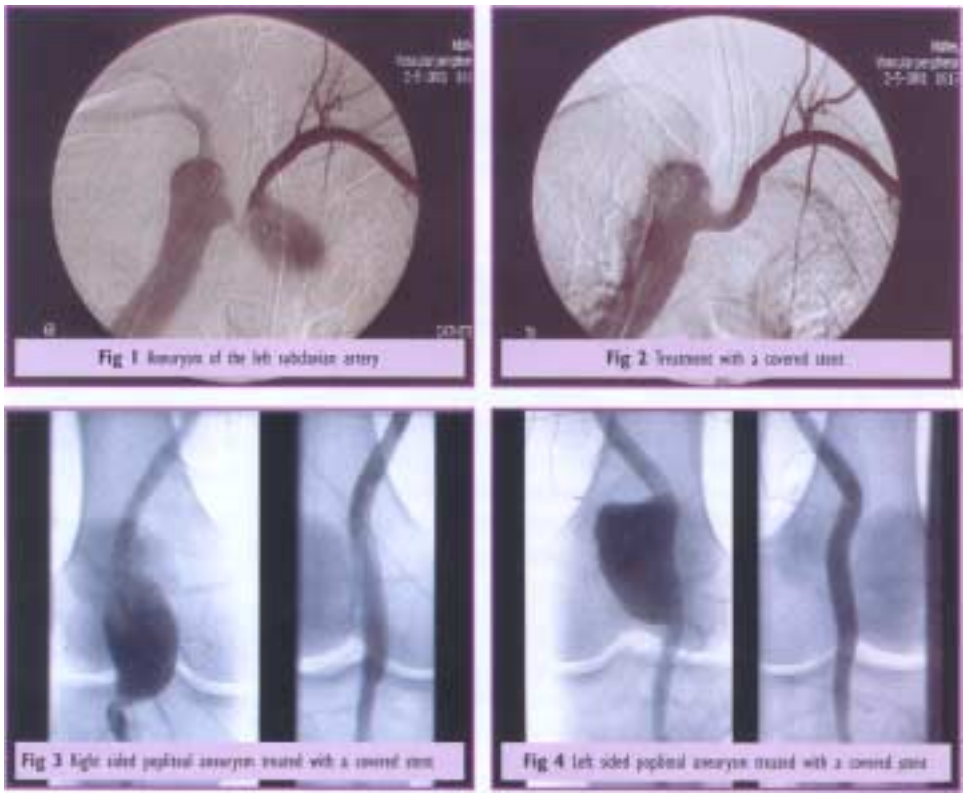
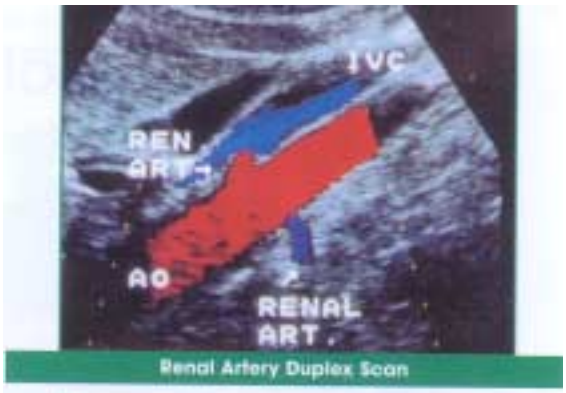
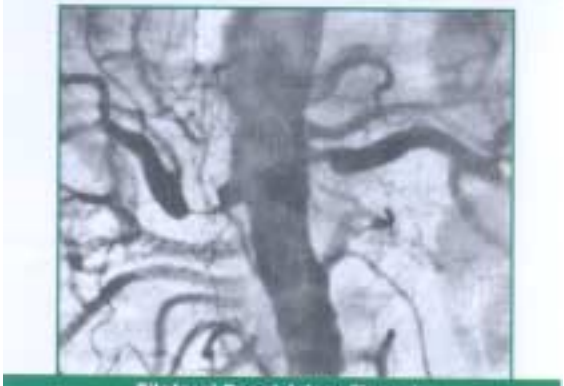


Figure 3 illustrates a right-sided popliteal aneurysm in a patient that had previously undergone an open repair of a popliteal aneurysm on the opposite side. He took a long time to recover from this and was keen to explore the possibility of a less invasive treatment for the second side. A Haemobahn endoprosthesis was inserted percutaneously through the femoral artery to successfully treat the aneurysm and restore a normal popliteal contour.
A similar case is illustrated in **Figure** 4.

Which hypertensive patients should be investigated for a correctable cause?



Renal Artery Duplex Scan


Bilateral Renal Artery Stenosis

Reno-vascular hypertension is a potentially correctable cause of hypertension but represents only 1- 2% of the hypertensive population. It is nevertheless important to identify these patients as the tests are usually non- invasive and the definitive treatment is usually renal artery angioplasty rather than open surgery. It is impractical to investigate every hypertensive patient.

The pointers to a possible reno-vascular cause are age of onset before 35, difficult to control hypertension, renal dysfunction after the introduction of an ACE inhibitor and the presence of an abdominal or renal angle bruit. Older patients with stable hypertension who become unstable frequently have an atherosclerotic renal artery problem.

The screening test of choice is a renal artery colour flow duplex scan. This is inexpensive and non-invasive but is very operator- dependant. In the hands of a vascular technologist or radiologist who does a large number of these studies the test is reliable as long as technical factors such as obesity or bowel gas do not interfere with the study. More sophisticated tests include captopril enhanced renal scintigraphy and magnetic resonance angiography.
Renal angiography may be required to make the diagnosis but the incidental finding of a renal artery stenosis during an angiogram performed to investigate the abdominal aorta or coronary arteries is of questionable significance. Many of these represent co- existent atherosclerotic disease in patients with long standing essential hypertension. Such lesions should not be treated unless there is compelling evidence that they are the likely source of the hypertension or of a recent deterioration in renal function.

THE SWOLLEN LIMB

Any excess tissue in any compartment of the limb may cause swelling. The mast common cause is *due* to *excess fluid - oedema. Careful attention* to *detail is required* to *correctly assess and diagnose the cause of leg swelling.*

Causes of lower limb swelling

ACUTE
Deep vein thrombosis
Superficial vein thrombophlebitis Cellulitis
Joint pathology with effusion Arthritis Fracture
Haematoma
Ruptured Baker's cyst Torn calf muscle
Acute arterial occlusion Hypo-proteinaemia

Clinical features of deep vein thrombosis including calf pain or tenderness, swelling, warm slightly cyanosed skin or prominent superficial veins may be present but the clinical diagnosis of deep vein thrombosis is inaccurate. Only in extensive occlusive ilio-femoral thrombosis does the clinical assessment have any reliability.
In only 35-50% of clinically suspected calf vein thrombosis is the diagnosis confirmed.
Non occlusive thrombosis may be entirely silent. In fact in the presence of deep vein thrombosis the fewer the symptoms the greater the threat of pulmonary emboli.

CHRONIC
Congenital vascular abnormalities
Chronic venous hypertension
Post thrombotic syndrome
Venous obstruction
Primary lymphoedema

Secondary lymphoedema
Infection
Trauma
Malignant infiltration
Heart failure
Paralysis and dependency

Venography has for many years been the gold standard. This is, however, invasive, costly, painful and potentially complicated by thrombosis with occasional extravasation of contrast and contrast reactions.
Recent advances in ultrasound technology have resulted in venography being largely superseded by colour flow duplex scanning and compression ultrasound. Ultrasonography has definite limitations in the assessment of recurrent deep vein thrombosis.

Endoscopic Treatment of Incompetent Calf Perforating Veins

The calf perforators are a system of medially and laterally situated veins, which provide channels of communication between the deep and superficial systems. To function efficiently they require competent valves. Incompetent perforating veins may lead to inflammatory panniculitis involving the subcutaneous tissue of the calf as well as to frank venous ulceration. This is particularly so if the deep veins are also incompetent.

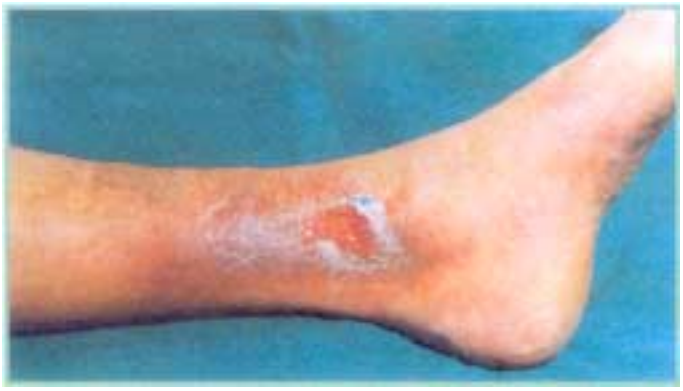
Colour flow duplex scanning enables the vascular surgeon to determine whether calf perforators are competent or not. When this problem is associated with incompetence at the sapheno-femoral or sapheno-popliteal junctions, control of venous reflux at these points assumes far greater importance than any perforator problem making surgical treatment of the perforator problem unnecessary in the majority of these cases.

Sub-fascial ligation of perforating veins has been an established procedure for many decades. However, until recently this operation required a long incision through very unhealthy tissue. A high incidence of wound necrosis and infection was the order of the day.

Modern endoscopic techniques now permit the clear visualization of these perforating veins using a telescope introduced through a standard laparoscopic port. The veins can then be interrupted by the application of metal clips. The procedure is easy to perform and requires no specialized equipment other than ports and instruments that are readily available in all theatres performing laparoscopic surgery.

An Approach to Leg Ulcers

Leg ulcers are a very common clinical problem. Correct management is vital to prevent unnecessarily prolonged treatment. The causes of leg ulceration can be seen in the attached table.



Vascular	**arterial**	pvd buerger's disease arterio-venous fistula collagen vascular diseases hypertension raynaud's phenomenon
	venous	chronic venous insufficiency varicose veins post injection sclerotherapy
	lymphatic	chronic lymphoedema
Infective		bone pyogenic miscellaneous specific / non specific
Systemic metabolic		diabetes mellitus sickle cell anaemia avitaminosis ulcerative colitis
Neoplastic	**primary**	scc/bcc/melanoma/leukaemia/kaposi
	secondary	
Traumatic		burns / decubitus / bites etc
Neurotrophic		cord lesions peripheral neuropathies

Practical management Of Leg Ulcers is summarised as **follows:**

Clean thoroughly, exclude venous hypertension remove slough from surface.
Exclude systemic conditions
Measure dimensions (**photograph).**
Exclude specific infections.
Assess surrounding skin.
Exclude neuropathy
Exclude malignancy.
Pus swab for mcs
Exclude arterial insufficiency.
Select appropriate dressing

Two of the most important problems are exclusion of arterial and venous insufficiency. The best way of doing this remains appropriate Duplex assessment after a vascular surgical opinion has been obtained. This is widely recognised, and has led to the development of Leg Ulcer Clinics. where a full assessment can be made at one visit. We currently offer this Leg Ulcer Service at all of our consulting rooms on a weekly basis.

Anti-platelet drugs are extremely useful for both preventing and treating atherosclerotic vascular disease and their use has become very widespread.
They have virtually no role in venous thrombosis or venous disease. Unfortunately for surgeons, anti-platelet agents are the most common cause of wound oozing.

ASPIRIN

Aspirin is still the most commonly used anti-platelet drug. It irreversibly blocks cyclo-oxygenase for the entire life of the platelet causing a mild increase in the bleeding time and a moderate decrease in platelet aggregation.
Aspirin reduces the risk of myocardial infarction in patients with stable angina and helps prevent secondary myocardial infarction. There is convincing evidence for its benefit in patients with TIA or ischaemic stroke. The ACE study has recommended a dose between 75 -325 mg/day.

DIPYRIDAMOLE

Dipyridamole (Persantin/Plato) blocks phosphodiesterase. Although it inhibits platelet aggregation in vitro the clinical performance has been disappointing. A recent study (ESPS2) showing a benefit when combined with aspirin has been severely criticized.
Because of side effects this drug should only be considered in patients with allergies to aspirin. In this regard it is being replaced by several superior drugs.

TICLOPIDINE AND CLOPIDOGREL

Thienopyridine drugs block the ADP receptors. They are better than aspirin for stroke prevention and maintaining the patency of coronary stents. Side effects of Ticlopidine including neutropenia and skin rashes have limited its usefulness but the recent CAPRIE study has validated ciopidogrel (Plavix) as effective in the secondary prevention of vascular disease without

significant side effects.
The drug is expensive and therefore should be *reserved* for specific situations where aspirin cannot be used or is considered to be of limited efficacy. Clopidogrel definitely increases surgical bleeding. Elective or semi-urgent procedures should be postponed until the drug has been stopped for at least 6 days.

IIB/IIIA INHIBITORS

Blocking the IIb/IIIA receptor renders, the platelet completely non- functional. The use of these agents has been largely limited to cardiology where they have been useful in unstable angina and in coronary angioplasty or stenting. Abciximab (Reopro) is a monoclonal antibody providing a non-reversible block. Tirofiban (Aggrastat) and Eptifibatide (Integrilin) have a shorter duration of action. There is a considerable risk of bleeding with these agents, which is particularly relevant to patients requiring urgent cardiac and vascular surgery.
Oral IIb/IIIA antagonists have been disappointing due to bleeding complications. These have necessitated the abandonment of three large trials (Symphony, Bravo and Excite). The clinical utility of intravenous IIIb/IIIa inhibitors in peripheral vascular interventions remains undetermined.