Lower-limb venous thrombosis

Deep-vein thrombosis and pulmonary embolism coexist to cause significant morbidity and mortality.

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Deep-vein thrombosis (DVT) and pulmonary embolism (PE) are major causes of significant morbidity and mortality which are frequently, but not exclusively, associated with surgery, injury and other reasons for hospitalisation. In the acute setting, PE may be fatal while in the long term pulmonary hypertension can develop, particularly from recurrent PE. Post-thrombotic chronic venous insufficiency, better described as post-phlebitic chronic lower-limb venous hypertension (CVH), occurs as a result of the DVT causing deep venous reflux and/or venous outflow obstruction and can cause swelling, skin changes (dermatoliposclerosis) and venous ulceration. With appropriate therapy the risk of both the primary condition as well as the consequences thereof can be reduced. As DVT and PE are inextricably linked the syndrome is usually referred to as venous thromboembolism (VTE).

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On the other hand, superficial venous thrombosis, often referred to as thrombophlebitis, is frequently an innocuous condition in which thrombosis occurs in one of the subcutaneous (superficial) veins of the lower limb. This is frequently a minor complication of varicose veins and as the risk of embolisation is small and only occurs if the sapheno-femoral or sapheno-popliteal junction is involved, it does not require any specific intervention other than symptomatic relief (which is generally not easily achieved). Recurrent superficial thrombophlebitis however, particularly in the absence of varicose veins, may be an important marker of a hypercoaguable condition.

Epidemiology

The incidence of the various components of VTE is:

- any DVT: ~ 2/1 000 person-years
- symptomatic non-fatal PE: ~ 0.2/1 000 person-years
- fatal autopsy-detected PE: ~ 0.5/1 000 person-years.

The prevalence of venous ulceration is at least 3/1 000 and approximately 25% of these are thought to be due to the complications of DVT.

Rates of VTE are higher in men than in women, and increase with age. Approximately half the cases of VTE are secondary to one or more underlying conditions. These most frequently are malignancy, immobilisation, surgery or major trauma, while in South Africa the association with HIV/AIDS is overwhelming.

Aetiology and pathophysiology

Virchow's triad, which describes the factors that predispose to VTE, consists of:

- lower-limb venous stasis
- a systemic hypercoaguable condition
- venous endothelial damage.

Usually it is necessary that at least two of the above factors coexist for VTE to develop. For this reason the clinical settings in which these conditions frequently occur are prolonged immobilisation, trauma, surgery, infection (including HIV/AIDS) and the postpartum period. Other factors that influence the risk are increasing age, obesity, malignancy, prior VTE, varicose veins, dehydration and oestrogen therapy. In the background is a systemic predisposition due to primary or secondary hypercoaguable (thrombophilic) conditions.

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In South Africa, HIV infection has been recognised as a hypercoaguable condition since the late 1980s. A 2005 systematic review of published studies indicated that the prevalence of VTE in an HIV-infected population can be as high as 18%.[1] Today the vast majority of patients treated for VTE in South African hospitals are HIV positive.

It is widely recognised that patients admitted to hospital are particularly at risk for VTE (Table I). This problem also continues after discharge.

Table I. The risk of developing DVT and PE when patients are hospitalised for the listed conditions without being given any form of thrombo-prophylaxis

Condition	DVT*	PE	Fatal PE
General medicine	**12% (10 - 14)**		
Patients in ICU	25% (19 - 32)		
Stroke	56% (51 - 61)		
General surgery	**25% (24 - 26)**	**1.6% (1.3 - 2.0)**	**0.87% (0.6 - 1.1)**
Multiple trauma	50% (46 - 55)		
Spinal cord injury	35% (31 - 39)		
Elective hip replacement	51% (48 - 54)	4.0% (3.0 - 5.1)	1.7% (0.4 - 2.7)
Elective knee replacement	47% (42 - 51)		
Traumatic orthopaedic surgery		6.9% (4.8 - 9.5)	
Hip fracture	44% (40 - 47)		4% (3.0 - 5.3)

*The number presented reflects all DVT (proximal and calf vein) found using objective methods of testing. The number for proximal DVT is approximately 25% of this. E.g. the rate of proximal DVT in general surgery patients is between 5.5% and 8.3%.

Adapted from Nicolaides AN, *et al.*[2]

Values presented are weighted means (95% confidence intervals).

Most DVTs arise in calf muscle veins, particularly within the gastrocnemius and soleus muscles (calf vein DVT). Many of these remain localised to the muscle and will not cause any clinical problem. If, however, the circumstances that initially caused the thrombus to develop persist, the thrombus can propagate into the proximal, larger deep veins of the lower limb (the popliteal veins and those craniad to these). The more proximal DVTs are at substantially greater risk of embolisation and the more proximal these thrombi are the greater the risk of mortality. For instance, it is estimated that the risk of PE from an extensive, newly formed iliac vein thrombus, in the absence of anticoagulation, is in the region of 70% and that the mortality is 5% per day while the patient is not anticoagulated.

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In the long term, most small DVTs are thrombolysed by intrinsic processes and therefore are of no consequence. If the thrombus load is large, however, and especially if the DVT involves proximal segments, the thrombus may only be partially broken down and therefore can cause both venous outflow obstruction and venous valvular dysfunction. This can cause CVH with varying degrees of lower-limb oedema, dermatoliposclerosis and venous ulceration. These complications tend to arise in the first 2 - 5 years after the DVT occurred.

Thromboprophylaxis

The risk of developing VTE in hospitalised patients can be reduced. Many guidelines for thromboprophylaxis have been published.[2,3] In order to achieve high levels of compliance simple recommendations are advisable. For this reason the authors use a combination of the patient's age, the risk of a procedure or disease and the risk of a hypercoaguable state being present to determine an individual's risk of developing VTE (levels of risk of developing VTE are reflected in Table II; individualised patient risk of developing VTE is determined using Table III). The need for thromboprophylaxis can then be determined (Table IV).

Table II. Levels of risk of developing VTE and its complications in hospitalised patients

Category	Frequency of DVT	Frequency of PE	Frequency of fatal PE
High risk	>40%	>10%	>1%
Moderate risk	10 - 40%	1 - 10%	0.1 - 1%
Low risk	<10%	<1%	<0.1%

From Nicolaides AN, *et al.*[2]

Table III. A simplified method of evaluating the risk of developing VTE in hospitalised patients

	Extent of illness or surgery	
Age	**Minor**	**Major**
>60	Moderate risk	High risk
40 - 60	Low risk	Moderate risk
<40	Low risk	Low risk

Additional factors: Previous VTE, known hypercoaguable condition, obesity, pregnancy, inflammatory bowel disease, malignancy, varicose veins and oestrogen therapy.

NB. The presence of one or more of these factors increases the risk from low to moderate risk or from moderate to high risk.

Major surgery: Any operation lasting longer than 45 min and all abdominal surgery and extensive bone and joint surgery.

Major illness: Any hospitalisation causing significant immobility or due to severe infectious agent or malignancy.

Modified from Nicolaides AN, *et al.*[2]

Table IV. Thromboprophylactic recommendations

Low risk	No thromboprophylaxis is recommended
Moderate risk	An effective form of thromboprophylaxis is recommended.
	This should be either in the form of an effective low-dose anticoagulant *or* an effective mechanical method (see below)
	The thromboprophylaxis should ideally be started before the risk commences and should continue as long as the risk persists (see comment regarding duration of thromboprophylaxis below)
High risk	An effective form of thromboprophylaxis is mandatory and consideration should be given to using a combination of low-dose anticoagulants and mechanical devices
	Continuation of thromboprophylaxis beyond the period of hospitalisation should be considered

All patients should be well hydrated and early mobilisation is mandatory.
Modified from Nicolaides AN, *et al.*[2]

Thromboprophylaxis is usually given for 5 - 7 days. In circumstances where the risk continues after discharge from hospital extended thromboprophylaxis to 1 month reduces asymptomatic DVT by a further 50 - 70%. For this reason individuals who remain immobile, or if the original condition causing risk has not resolved, are usually given extended post-discharge thromboprophylaxis.

The methods of thromboprophylaxis proven to be clinically effective and relevant are low-dose anticoagulants and mechanical devices.[2,3]

Low-dose anticoagulants

Low-dose unfractionated heparin (UFH) is given at a dose of 5 000 units subcutaneously, 8- or 12-hourly. In most clinical settings this reduces the risk of developing a DVT by approximately 60% with a similar reduction in PE-associated mortality. There is however a marginal increase in the risk of haemorrhage, particularly in patients undergoing surgery, but this is not associated with a rise in mortality.

Low-dose low-molecular-weight heparins (LMWHs) are as effective, if not slightly more effective when compared with low-dose UFH with fewer side-effects. As each LMWH is different the dosing is different for each preparation but they usually only require daily dosing (Table V).

A variety of other anticoagulants are used in specific settings, particularly in patients undergoing joint replacement surgery. These include the vitamin K antagonists, the newer pentasaccharides and thrombin inhibitors.

Mechanical devices

Graduated elastic compression (GEC) stockings and intermittent pneumatic compression (IPC) reduce the incidence of asymptomatic DVT by approximately 50 - 60% but the number of studies reported is too small to assess their effects on PE.

Table V. Commonly prescribed anticoagulants for thromboprophylaxis and treatment of VTE

Thromboprophylaxis	
• LMWH	
Enoxaparin	40 mg sc daily
	In high-risk patient 30 mg sc bd may be appropriate. The first dose should be given 12 hours prior to surgery or 6 - 8 hours after surgery if there is a concern regarding intra-operative haemorrhage
Dalteparin	2 500 IU sc daily
	In high-risk patient 5 000 IU is recommended
	First dose is given 2 hours prior to surgery
Nadroparin	2 850 IU sc daily
	First dose is given 2 hours prior to surgery
• UFH	5 000 IU sc 8- or 12-hourly
	The first dose should be given 1 -2 hours prior to surgery
Acute anticoagulation	
• LMWH	
Enoxaparin	1 mg/kg body weight sc 12-hrly (maximum recommended dose 100 mg)
Dalteparin	200 IU/kg body weight sc 12-hrly (maximum recommended dose 18 000 IU)
Nadroparin	Please see package insert
• UFH	100 IU/kg IVI stat as starting dose; thereafter 20 IU/kg hourly IVI as a continuous infusion
	Check the aPTT 3 hours after starting UFH; adjust the dose of the UFH infusion to prolong the aPTT to between 60 and 90 seconds
Chronic treatment	
• Warfarin	Initial dose 5 mg daily; dose adjusted thereafter to achieve an INR in the range of 2 - 3.5 in most patients. Particular care should be taken in patients who are old, wasted, or malnourished or have hepatic dysfunction
• Enoxaparin	1 mg/kg body weight sc 12-hrly
	In selected patients the antifactor Xa activity should be measured to ensure that the dose is in the therapeutic range

Note:
- Adjustment of doses in patients with renal dysfunction is required. Refer to package inserts for further information.
- Thromboprophylaxis using anticoagulants should not be given preoperatively if neuroaxial anaesthesia is being planned. These catheters should also not be placed or removed within 12 hours following a dose of LMWH.
- The safety of thromboprohylaxis using anticoagulants in pregnancy has not been established.

As aspirin only reduces the risk of developing a DVT by approximately 30% and of developing a PE by about 50% it is not usually recommended for thromboprophylaxis.

There is evidence that combinations of a low-dose anticoagulant and a mechanical device are more effective than using either on their own.

Most guidelines suggest that thromboprophylaxis should be started prior to the development of risk although there is evidence that introduction at a later stage still confers some benefit. For example, enoxaparin should be started 12 hours before surgery but when the risk of haemorrhage or the consequences thereof are substantial then a 6 - 8-hour postoperative start is justified. When such a compromise is made, use of a perioperative mechanical device should be considered.

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Diagnosis

The astute clinician maintains a high index of suspicion of VTE in all patients in order to make an early diagnosis of a DVT and/or PE. This is not only particularly of value in patients at high risk of developing VTE but also in all individuals who present with recent-onset lower-limb discomfort or swelling.

The symptoms of a DVT include swelling, pain and hyperaemia of the affected limb, yet many patients are asymptomatic. In addition, the correlation between the location of symptoms and the site of thrombosis is frequently not apparent. Symptoms only in the calf can be associated with major proximal vein involvement and *vice versa*. Physical examination may reveal calf tenderness, oedema or calf swelling, localised increase in temperature, erythema, and/or superficial venous dilation. There may be tenderness in the thigh along the course of the major veins. These symptoms are suggestive but not diagnostic of a DVT and require confirmation using an objective diagnostic modality. Importantly, Homan's sign (pain on dorsiflexion of the ankle) is unreliable and is not used.

Of patients with a clinically suspected DVT only some (±25%) will have one. The other reasons for the symptoms and signs include:

- muscle strains, tears, or twisting injuries to the leg
- other causes of lower-limb swelling such as cardiac, hepatic and renal pathologies
- lymphoedema
- chronic venous hypertension and its complications
- popliteal (Baker's) cysts
- cellulitis
- other knee pathologies.

The objective methods of making a diagnosis of DVT are venous compression ultrasonography, contrast venography, CT venography or MRI venography. In symptomatic patients compression venous ultrasonography is usually the first diagnostic modality used when available. This consists of B-mode imaging of the major veins in the groin and popliteal fossa evaluating compressibility of these vessels only. If the lumen can easily be obliterated with modest pressure on the ultrasound probe this implies that the lumen is not filled with a thrombus. If compressibility is not easily established Doppler insonation is also used to establish whether the lumen of the vein is patent or not. Extensive further evaluation of all veins in the affected lower limb does not significantly enhance the accuracy of venous ultrasound in such patients. A positive study in patients with a first episode of DVT usually establishes the diagnosis, with a positive predictive value for limited compression venous ultrasound of 94% (95% CI: 87 - 98%). If the initial study is negative and the clinical suspicion of DVT continues to be high, a repeat of the study should be obtained in 3 - 5 days as progression of the DVT will make it possible to detect the thrombus more easily. Ultrasonography is not as accurate in making a diagnosis of calf vein or pelvic vein DVT.

In asymptomatic patients in whom a DVT is suspected, compression ultrasonography can be preceded by a D-dimer assay as a negative D-dimer assay has a high negative predictive value for VTE (±94%). In some high-risk settings, such as in a casualty department, this negative predictive value may not be good enough. When such concern exists its value can then be enhanced by using some form of clinical probability score (e.g. the Wells score – Table VI). A review of 15 studies in which the Wells score was tested concluded that patients with low scores (0 or less) had a median negative predictive value for DVT of 96%.[5] The negative predictive value was further improved to 99% by the presence of a negative D-dimer assay.

The positive predictive value for DVT using D-dimer assays, with or without the use of a probability score, is low and therefore this test is not enough to establish the diagnosis of VTE. Further studies such as compression venous ultrasonography are therefore required.

Contrast venography or CT venography is currently only used on the rare occasions when the above non-invasive testing is not clinically feasible or the results are equivocal. These settings only occur in patients suspected of having pelvic vein DVT or recurrent DVT where the old DVT has caused fibrosis of the vein wall or where it has incompletely resolved.

The algorithm currently used to make the diagnosis of DVT in the authors' institution is depicted in Fig. 1.

Treatment

The primary objectives of treatment for DVT are to prevent further clot extension, to prevent fatal and non-fatal PE and to reduce the risk of recurrent thrombosis. The secondary objective is to reduce the risk of developing late complications of CVH and pulmonary hypertension. As anticoagulation

Table VI. Modified Wells score[4,6]

	Score*
Paralysis, paresis or recent orthopaedic casting of a lower limb	+1
Recently bedridden for longer than 3 days or major surgery in the last 4 weeks	+1
Localised tenderness of the deep lower-limb veins	+1
Swelling of the entire lower limb	+1
Calf swelling (3 cm > the other limb, measured 10 cm below tibial tuberosity)	+1
Pitting oedema of the symptomatic limb	+1
Collateral superficial veins (not varicose veins)	+1
Active cancer or cancer treated within the last 6 months	+1
An alternative diagnosis more likely	-2
Previous VTE	+1

* 1 or less – DVT unlikely; 2 or greater – DVT possible.

is extremely effective in achieving the first objective, rapid-onset anticoagulation is mandatory for all patients with proximal DVT. Rapid-onset therapy is important as pulmonary embolism will occur in approximately 50% of untreated individuals, most often within days or weeks of the event. Therefore patients with DVT or pulmonary embolism are treated acutely with an anticoagulant dose of one of the heparins (Table V). The need for anticoagulation in the treatment of an isolated distal DVT is controversial. Our approach is to treat such DVTs if they are symptomatic or if the patient is at high risk of developing pulmonary hypertension. All such patients are carefully followed up with a repeat ultrasound of the popliteal veins at 5 - 7 days to exclude thrombus propagation.

As LMWH is safer, more convenient and possibly more effective than UFH, the current standard of care is to use one of the LMWHs in all patients treated for VTE (unless they have a contraindication to the use of heparin). If a LMWH is not available unfractionated intravenous heparin should be given to rapidly achieve a doubling in the PTT. This ideally is converted to a LMWH as soon as possible. Treatment with LMWH should be continued for a minimum of 5 days and oral anticoagulation should overlap with LMWH for at least 4 - 5 days. Giving LMWH for a longer period (between 7 and 10 days) reduces the risk of the late post-phlebitic complications from developing.

In all patients receiving a heparin (including the LMWHs) a platelet count should be obtained at least once after 72 hours to detect the development of heparin-induced thrombocytopenia (HIT). The heparin should be stopped if there is a precipitous fall in the platelet count or if the platelet count falls below 100 000/µl.

In most patients warfarin should be given immediately after the first dose of LMWH, at an initial oral dose of approximately 5 mg/day. The INR is then carefully monitored from 72 hours onwards. A therapeutic range of between 2 and 3.5 is recommended. As most patients have variable responses over time to the warfarin regular monitoring (every other week once the INR is stable) is required. A low INR, particularly in the early phase of the disease, is associated with recurrence of thrombosis, while high INR levels are associated with haemorrhage and possible severe morbidity and even mortality. Elderly patients and those at high risk of bleeding or those who are malnourished or debilitated or have heart failure or liver disease should be given a reduced initial warfarin dose and the subsequent target INR should also be adjusted. If oral anticoagulants are contraindicated (mostly due to allergy, risk of haemorrhage or teratogenesis) or are inconvenient, long-term therapy can be undertaken with dose-adjusted LMWH.

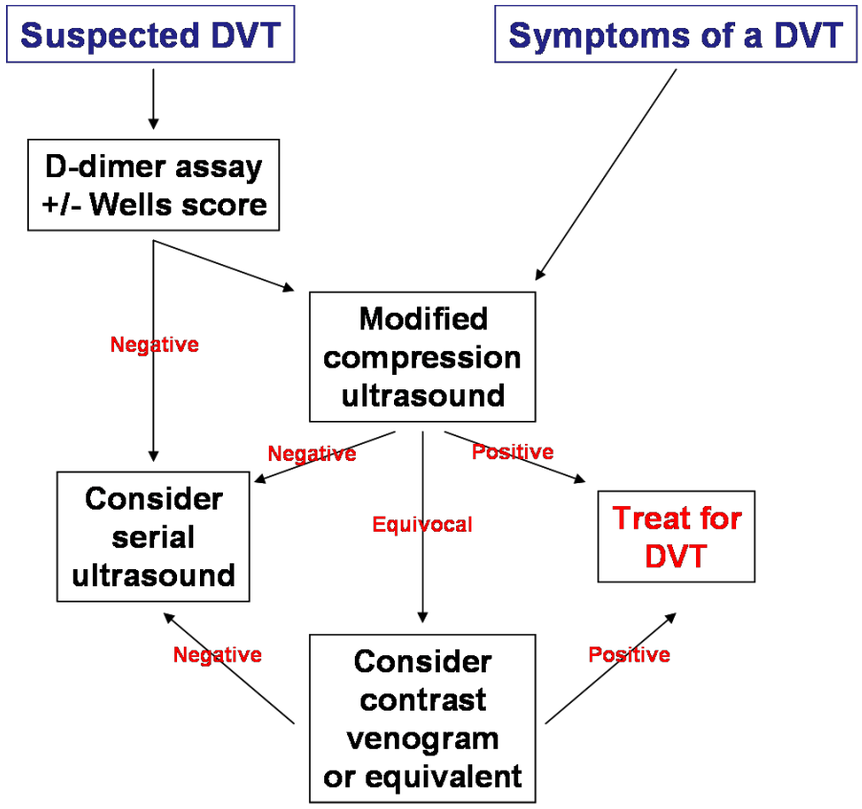

Fig. 1. Algorithm currently used to make the diagnosis of DVT in the authors' institution.

In selected patients, particularly young individuals with PE causing haemodynamic instability or those with a massive ilio-femoral DVT causing extensive swelling, pain and possibly venous gangrene and who are at low risk of haemorrhagic complications, other treatment modalities such as thrombolytic agents, surgical thrombectomy, or percutaneous mechanical thrombectomy should be considered. Inferior vena caval filters are used when there is a contraindication to, or a failure of, anticoagulant therapy in an individual with, or at high risk for, proximal vein thrombosis or PE. They are also recommended in patients with recurrent thromboembolism despite adequate anticoagulation and for chronic recurrent embolism with pulmonary hypertension.

In selected patients the initial anticoagulation can be safely administered in an outpatient setting. This results in reduced hospital costs and greater patient satisfaction. The conditions that need to be met for such outpatient treatment are:

- that the patients are able to understand and can administer the LMWH in a home setting
- that the patients are able to attend regular follow-up and have easy and rapid access to emergency medical care
- that no other problem exists that places the patients at increased risk of developing haemorrhagic complications.

The duration of anticoagulation therapy is a balance between the risk of recurrence of the VTE, the risk of haemorrhage from the anticoagulation and the patient's overall state of health and must therefore be individualised. In general the following applies:

- Patients with first-time VTE who have reversible and time-limited risk factors (e.g. trauma or surgery) should be treated for at least 3 months. If a significant thrombus load is still present at this time an extension of the anticoagulation to 6 months should be considered as the risk of recurrence is still high and because fewer post-phlebitic complications have been documented.
- Recent studies have suggested that once the anticoagulation has been stopped evaluation of the underlying thrombotic potential (as measured using a D-dimer assay, for example) at this stage can differentiate patients who have a high risk (approximately 20%) of developing a VTE within 18 months from those who have a low risk (<4%). Intuitively, selected individuals

who fall into the former group and who are at low risk from haemorrhagic complications of anticoagulant therapy should be considered for additional re-anticoagulation for another 18 months.

- Treatment is continued while the underlying hypercoaguable state persists. This applies not only to known hypercoaguable conditions but also to those patients presumed to have an unrecognised hypercoaguable condition on the basis of having had recurrent, usually unprovoked VTE.
- When there is significant risk associated with a further episode of PE, usually as a result of pulmonary hypertension, prolonged anticoagulation should be considered.

In all patients, once anticoagulation has been started and the patient's symptoms are improving, early ambulation is strongly advocated. During this initial ambulation, and ideally for the first 2 years following an episode of VTE, below-knee, grade 2 elastic compression stockings should be worn on the affected limb to reduce post-phlebitic complications.

Screening for underlying hypercoaguable conditions

Screening for such conditions is performed to predict the risk of recurrent VTE and who will require prolonged anticoagulation or to identify previously unrecognised diseases.

Screening for the inherited hypercoaguable conditions is most useful in the following situations:

- primary VTE in patients under 50 years of age
- a strong family history of VTE
- recurrent primary VTE and thrombophlebitis.

The clinical value of such screening is not clear as the majority of these patients will already have a good reason to receive long-term anticoagulation. When counselling family members of such patients, few conditions require additional treatment outside of what is identified by the presence of a family history of VTE. As the majority of the inherited hypercoaguable conditions are altered by an acute thrombotic event, testing is of little value soon after the development of a VTE and should only be performed 6 - 8 weeks after all anticoagulants have been discontinued. Notwithstanding these concerns we screen all high-risk individuals.

Similarly, the acquired hypercoaguable conditions are usually well advanced before they have a significant impact on the coagulation process. For this reason most are well established and known when the initial episode of VTE is recognised. On the other hand, these conditions can be of such importance that we believe that screening of most patients presenting with a VTE is of value. As no guidelines exist for the screening of such secondary causes the authors' recommendations are based on a pragmatic approach and have been modified for the South African setting. The following studies are obtained:

All patients:

- HIV
- Liver function tests
- CRP, ESR
- CXR
- Haemoglobin and MCHC

Selected patients:

- Antiphospholipid and anticardiolipin antibodies
- Abdominal ultrasound
- Stool for occult blood
- Urine analysis

Men:

- PSA

Women:

- Standard breast and cervical screening
- Ca125-5

Additional screening is performed as clinical circumstances demand.

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In a nutshell

- DVT is a common and potentially dangerous complication in all ill patients and in those individuals undergoing surgery.
- A high awareness of this condition and applying simple prophylactic measures when appropriate can substantially reduce the risk of such complications.
- Making an early diagnosis of a DVT and its consequences relies on the astute clinician having a high index of suspicion in all patients.
- The diagnosis is most frequently made using probability scoring and a D-dimer assay in individuals who have no symptoms or signs but are considered to be at high risk.
- In patients who have significant symptoms or signs or those in whom a DVT cannot be excluded using the above studies, the diagnosis of DVT is confirmed, usually using lower-limb venous compression ultrasound.
- Treatment of VTE usually consists of early and rapid anticoagulation using LMWH followed by the early introduction of warfarin.
- Therapeutic warfarin doses are given for a minimum of 3 months and recent advances have helped in identifying individuals who will benefit from longer periods of such treatment.
- It is important also to recognise those patients who are at risk of developing recurrent VTE on the basis of an underlying inherited or acquired hypercoaguable condition.
- Some of these individuals will benefit from 'lifelong' anticoagulation.
- It is essential not to overlook the late complications of DVT and to institute measures to reduce the risk of these occurring. Elastic stockings should be applied to affected lower limbs for approximately 2 years.
- The HIV/AIDS pandemic in South Africa has resulted in a massive rise in the number of patients being treated for VTE in local hospitals.