

# **THROMBOEMBOLIC COMPLICATIONS OF PREGNANCY**

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Thromboembolic disease is the leading non-obstetrical cause of maternal mortality, with an incidence of 0.05-0.3%. Early recognition and proper treatment dramatically improves outcome. The risk of thromboembolism during pregnancy and the postpartum period is 5.5 times greater than that for nonpregnant patients. Twenty four percent of untreated patients with deep venous thrombosis will have a pulmonary embolism, with a mortality rate of 15%. If treated with anticoagulants, embolization will occur in only 4.5%. The risk of thromboembolism is increased during pregnancy because the gravid uterus impairs the velocity of venous flow from the lower extremities. The risk of thromboembolism increases nine-fold with cesarean delivery as compared with vaginal delivery. Venous thromboembolism (VTE) refers to the formation of a thrombus within veins. This can occur anywhere in the venous system but the clinically predominant sites are in the vessels of the leg (giving rise to [deep vein thrombosis](#), DVT) and in the lungs (resulting in a [pulmonary embolus](#), PE). The pathophysiology of VTE in pregnancy appears to relate to the increased venous stasis noted during this period but other factors such as alterations in the balance of proteins of the coagulation and fibrinolytic systems have also been implicated. Its importance in obstetrics is highlighted by the statistic that PE is the most common cause of maternal death in the UK.

## **THROMBOEMBOLISM - Epidemiology**

It affects about 1 in 100,000 women of childbearing age. It is up to 10 times more common in pregnant than in non-pregnant women of a similar age. Occurs in about 1/1000 pregnancies in women under the age of 35. Occurs in 2.4/1000 pregnancies in women over the age of 35. Inherited [thrombophilia](#) is present in 30%-50% of women with pregnancy associated venous thromboembolism. 10-20% of VTEs are PEs which are the main contributors to VTE mortality. They are the leading direct cause of maternal mortality in the UK, being responsible for a third of maternal deaths.

### **Mortality rate**

The mortality rate is low but exact figures are not widely published. A recent study suggested a rate of 8.9 per million pregnancy years but this was in young women (the risk is highest in the over 35 age group). 62% of women with fatal VTEs die in the first trimester although the risk per day is actually greatest in the weeks following delivery. 71% of postpartum deaths from VTE occur following vaginal delivery. 10% of postpartum deaths from VTE occur following operative (interventional) vaginal delivery.

### **Presentation**

Presentation is similar to non-pregnant patients with DVT or PE. PTE occurs most often secondary to DVT, but also can occur after superficial vein, puerperal septic vein and puerperal ovarian vein thrombosis.

Superficial vein thrombosis with an incidence of 0.15% can occur during the antepartum period although the incidence increases 8 fold into postpartum period.

DVT with an incidence of 0.02 to 0.36% . Most of them occur between 15 to 20 weeks of gestation.

Puerperal ovarian vein thrombosis has a 0.025% and septic pelvic vein thrombosis has an incidence of 0.1%.

13 to 24% of pregnant patients with untreated DVT experience pulmonary embolism and the mortality is 12 to 15%. Treatment decreases the incidence to 0.7 to 4.5% and reduces the mortality to 0.7%.

Untreated septic pelvic vein thrombosis has a pulmonary embolism incidence of 33%

In spite of great advancement in controlling maternal mortality PTE accounts directly for 12 to 25% of maternal deaths.

### **Identification of risk factors**

There are a number of known risk factors, some hereditary and others acquired and in 80% of patients, at least one risk factor can be identified. Notably, the antenatal period is known to be a weak risk factor and the postpartum period a moderate risk factor. Often, more than one risk factor is present and these should be actively identified when assessing the patient for VTE during and post pregnancy.

- **Inherited factors:**
  - **Factor V Leiden mutation** (most common)
  - Prothrombin 20210 mutation
  - **Antithrombin III deficiency**
  - **Protein C deficiency**
  - **Protein S deficiency**
  - Hyperhomocysteinaemia
  - Dysfibrinogenaemia
  - Disorders of plasminogen and plasminogen activation
  - Strong family history
- **Acquired factors:**
  - Obesity
  - Immobilisation (> 4 days bed rest)
  - Previous thrombotic event
  - Trauma
  - Inflammatory disorders such as **inflammatory bowel disease**
  - Cancer
  - Oestrogen therapy (including **contraception** and **hormone replacement therapy**)
  - **Sepsis** including urinary tract infections)
  - Gross **varicose veins**
  - **Antiphospholipid syndrome**
  - **Nephrotic syndrome**
  - **Paroxysmal nocturnal haemoglobinuria**
  - Stroke
  - **Polycythaemia vera**
  - **Sickle cell disease**
  - Long haul travel
- **Factors specific to pregnancy:**
  - Venous stasis
  - Advanced maternal age
  - **Multiparity**
  - Gestation < 36 weeks
  - Instrument-assisted or caesarean delivery
  - Haemorrhage
  - **Pre-eclampsia**
  - Prolonged labour

### **ETIOLOGY**

Pregnancy results in a five to sixfold increase in relative risk of VTE. This the result of at least 3 factors

1. Increased venous stasis
2. Hypercoagulable state of pregnancy
3. Vascular injury associated with vaginal or cesarean delivery.

VENOUS STASIS occurs due to the compression of the IVC by the gravid uterus which increasingly becomes an abdominal organ as opposed to its original pelvic organ disposition. This results in venous stasis distal to the compression, in the pelvis and lower extremities.

HYPERCOAGULABILITY in pregnancy is due to enhanced platelet turnover, coagulation and fibrinolysis. This is further enhanced during parturition. There is an increase in concentration of coagulation factors particularly 1, 5, 7, 8, 10 and 12. Thrombin generation is also increased. So pregnancy represents a state of accelerated but compensated intravascular coagulation and the coagulation activity is increased relative to the fibrinolytic activity.

VASCULAR TRAUMA occurs during vaginal delivery and placental separation. This leads to a series of physiological changes that further accelerate coagulation. LSCS increases the risk of DVT and PE 7 to 8 fold over vaginal delivery.

OBSTETRIC CONDITIONS like preeclampsia and multiple gestation increase the risk of VTE.

COINCIDENTAL DISEASES mentioned above in risk factors further increase this risk.

## **PATHOPHYSIOLOGY**

The manifestation and prognosis of PTE depends on several factors including

1. Site and size of emboli
2. Concurrent cardiopulmonary function
3. Rate of clot fragmentation and lysis
4. Presence or absence of source for recurrent emboli

After PE, respiratory failure can occur due to cardiopulmonary decompensation due to extensive direct occlusion of pulmonary vasculature leading to severe pulmonary hypertension and right ventricular overload and failure. There is also a disruption of normal capillary integrity and aggressive IV volume resuscitation may increase the hydrostatic pressure and contribute to pulmonary edema which can again lead to respiratory failure.

**DVT:** leg pain and discomfort (the left is more commonly affected), swelling, tenderness, oedema, increased temperature and a raised white cell count. There may also be **abdominal pain**. The difficulty is that some of these symptoms may be found in normal pregnancies. The patient may also be asymptomatic with a retrospective diagnosis being made following a PE.

**PE:** **dyspnoea**, **pleuritic chest pain**, **haemoptysis**, faintness, collapse. The patient may have focal signs in the chest, **tachypnoea**, a raised JVP and there may be ECG changes (S1Q3T3). **Arterial blood gases** taken with patient sitting down may show respiratory alkalosis and **hypoxaemia**. There may also be symptoms or signs of a DVT.

## **Differential diagnosis**

**DVT:** swelling and lower leg discomfort are not unusual in a normal pregnancy. Other possibilities include muscle strain, a ruptured **Baker's cyst**, **cellulitis**, **superficial thrombophlebitis**, ruptured plantaris tendon and trauma.

**PE:** potentially extensive but specifically rule out **chest infection** and an intra-abdominal bleed (look for abdominal signs, shoulder tip pain from diaphragmatic irritation and a low **jugular venous pressure**).

## Investigations and diagnosis

Any woman with symptoms and signs suggestive of VTE should have objective testing performed promptly. Treatment with low-molecular-weight heparin (LMWH) should be started until the diagnosis is excluded by the objective testing, unless treatment is strongly contraindicated. Many hospitals have local policies regarding the management of these patients which may involve the obstetricians, haematologists, physicians and radiologists.

## Diagnosis

### A. Deep Venous Thrombosis

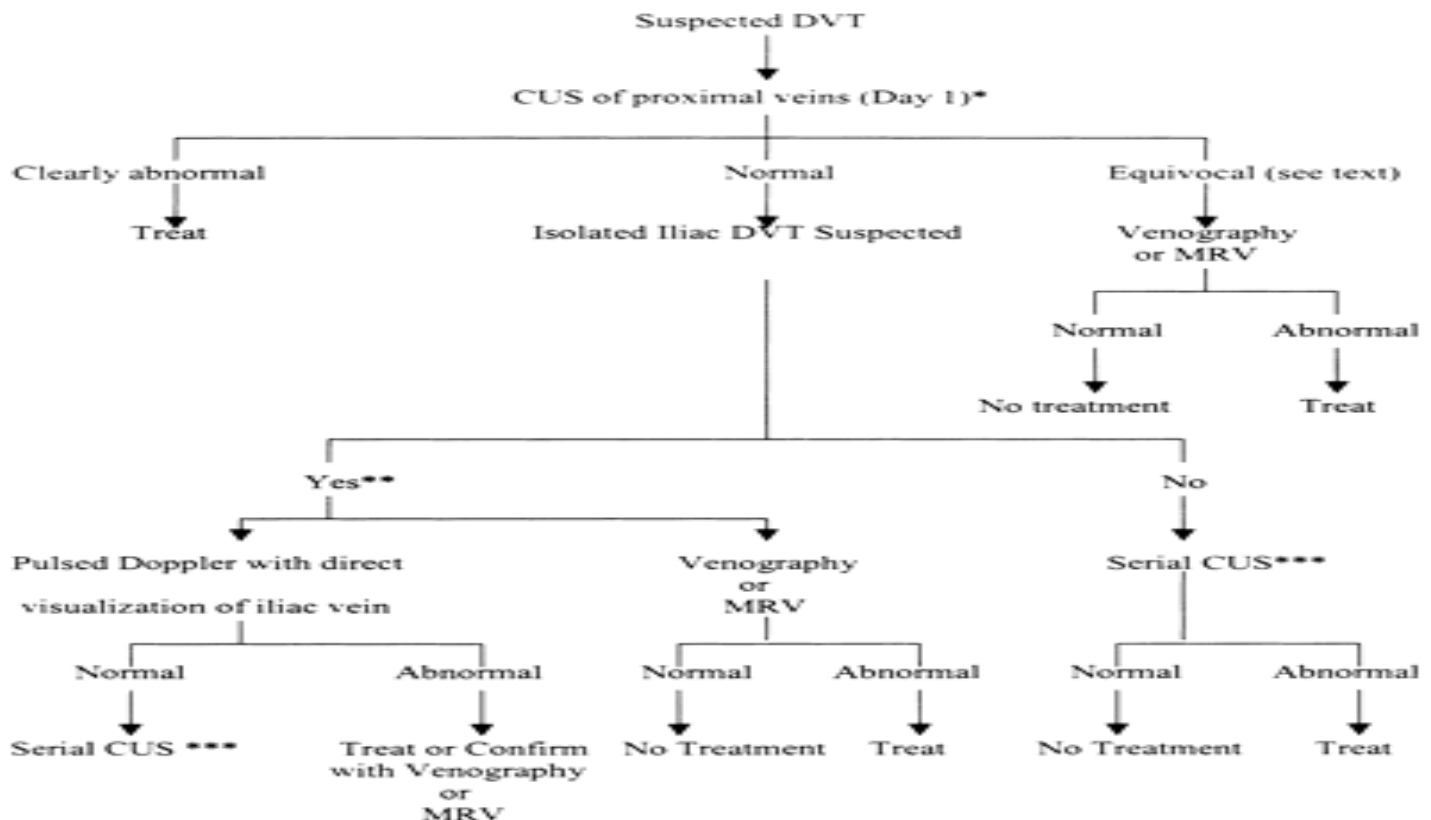
1. DVT most commonly manifests as pain and swelling. The physical exam may reveal tenderness, a difference in leg circumference, redness, and a positive Homan's sign. None of these signs or symptoms is specific, and DVT may also be completely asymptomatic.

2. **Venography** is an invasive procedure, and the contrast material can cause chemical phlebitis. Venography is useful when the results of other studies are equivocal.

3. **Doppler ultrasound** is the diagnostic study of choice in cases of suspected DVT. The sensitivity is 91% and specificity is 99%. When clinical findings are inconsistent with Doppler studies, venography is necessary.

4. **Impedance plethysmography** is an alternative technique for DVT diagnosis. Venous return in the lower extremity is occluded by inflation of a thigh cuff, and then the cuff is released, resulting in a decrease in calf blood volume. Any obstruction of the proximal veins diminishes the volume change, which is detected by measuring changes in electrical resistance (impedance) over the calf. If there is a clinical suspicion of a DVT, arrange an urgent compression duplex ultrasound scan. If this is negative and your suspicion is low, discontinue treatment. If it is negative but your suspicion is high, repeat the scan (or order an alternative imaging modality) one week later whilst keeping the patient anticoagulated. If this is negative, discontinue anticoagulation.

N.B. If you suspect an iliac vein thrombosis (back pain and swelling of the entire limb), magnetic resonance venography or conventional contrast venography may be considered.



## B. Pulmonary Embolism

1. Pulmonary embolism most frequently presents with dyspnea and tachypnea. Cough, anxiety, hemoptysis, cyanosis, diaphoresis and pleuritic chest pain may sometimes occur.

2. **Physical exam** may reveal only tachycardia or a few crackles. Massive pulmonary embolism may cause hypotension, syncope, right-sided heart failure with jugular vein distention, hepatomegaly, left parasternal heave, and accentuated and fixed splitting of the second heart sound. Eventually LV failure can occur due to poor LV filling and arterial hypoxemia.

<b>Clinical Findings in Pulmonary Embolism</b>	
<b>Clinical Finding</b>	<b>Pulmonary Embolism (%)</b>
Tachypnea	89
Dyspnea	81
Pleuritic pain	72
Apprehension	59
Cough	54
Tachycardia	43
Hemoptysis	34
Temperature >37°C	34

If there is a clinical suspicion of a PE, organise a chest X-ray and if this is normal, arrange a compression duplex doppler. If these are negative, the patient needs to have a ventilation–perfusion (V/Q) lung scan or a computed tomography pulmonary angiogram (CTPA) - discuss with the radiologist. If these are normal but the clinical suspicion remains high, continue anticoagulation and repeat the tests a week later.

Ideally, informed consent should be obtained before these tests are undertaken as there are risks associated with these investigations (V/Q scanning carries a slightly increased risk of childhood cancer compared with CTPA - 1/280,000 versus less than 1/1,000,000 - but carries a lower risk of maternal [breast cancer](#)).

## C. Laboratory Studies

1. **Electrocardiography** is abnormal in 90% of pulmonary embolisms; tachycardia is the most common abnormality. Nonspecific T-wave inversions occur in 40%; right axis shift with strain pattern occurs with large embolisms. P pulmonale and supraventricular arrhythmias may occur.

2. **Arterial Blood Gases.** A pulmonary embolism is unlikely with a PaO<sub>2</sub> of >80 mm Hg on room air. However, 11.5% of patients with pulmonary embolism have a PaO<sub>2</sub> of 80-90 mm Hg.

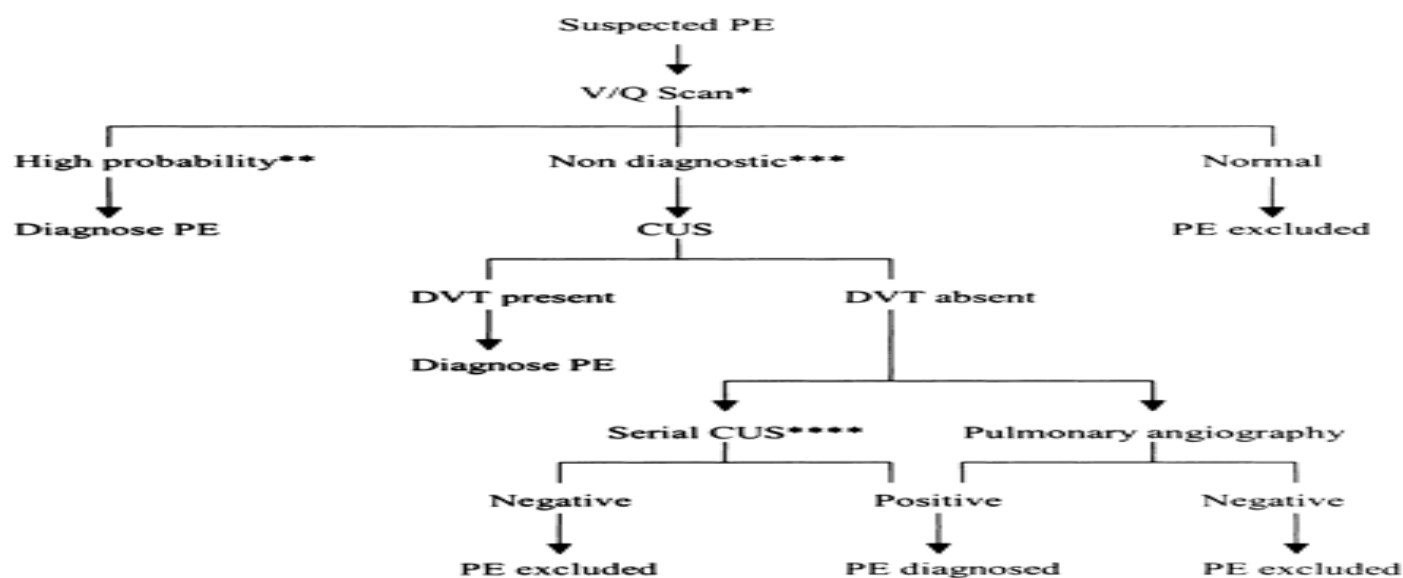
## 3. Technetium Lung Scanning

a. The perfusion scan is performed first, and a normal scan excludes pulmonary embolism. If the perfusion scan is abnormal, a ventilation scan is completed. Matching ventilation perfusion defects are not suggestive of embolism.

b. Almost all patients with pulmonary embolism have abnormal V/Q scan results (high, intermediate, or low probability). Unfortunately, most patients without emboli also have abnormal results (sensitivity 98%, specificity 10%). When clinical suspicion does not correlate with results of lung scanning, pulmonary angiography is necessary.

**4. Coagulation Studies.** If a family history of repeated is present; antithrombin-III, protein C.

**5. Invasive Haemodynamic Monitoring** shows 1) Normal to Low (< 15 mm Hg) PAOP. 2) Increased mean PAP (But < 35 mmHg). 3) Increased (>8 mmHg) CVP. Calculated PVR may be about 2.5 times the normal values.



### Blood tests

D-dimer is an unreliable test to carry out in these patients. In pregnancy, it can be elevated because of the physiological changes in the coagulation system and levels become 'abnormal' at term and in the postnatal period in most healthy pregnant women. Blood to check the full blood count, coagulation screen, [urea and electrolytes](#) and [liver function tests](#) before anticoagulant therapy is recommended. There is controversy surrounding the performance of a thrombophilia screen: it will not affect the immediate management of the patient and results are distorted by the pregnant state and by the presence of a thrombus. However, it can provide information that can influence the duration and intensity of anticoagulation. This is therefore best left to be discussed with everybody involved once the acute situation has been dealt with.

### Management

#### Massive life-threatening PE:

Nearly 10% of patients die in the first hour. Collapsed, shocked patients need to be managed by an experienced multidisciplinary team involving senior obstetricians, physicians and radiologists. Long term survival depends on rapid diagnosis and institution of therapy. Therapy focuses on 1) Adequate Maternal and foetal oxygenation 2) Support of maternal circulation including uteroplacental perfusion 3) Immediate anticoagulation or venous interruption to prevent recurrence of lethal PE.

- An urgent portable echocardiogram or CTPA within 1 hour of presentation should be arranged.

- If massive PTE is confirmed or, in extreme circumstances prior to confirmation, immediate **thrombolysis** should be considered. Both urokinase and streptokinase have been used. Urokinase therapy is started at 4400 IU / kg followed by 4400 IU / kg / hr. Thrombin time is a sensitive indicator to follow thrombolytic therapy. The thrombin time should never be greater than 5 times the normal value. This treatment definitely increases haemorrhagic risk
- Intravenous unfractionated heparin is the preferred treatment.
- Surgical embolectomy is an extreme measure

## General points

- In a woman with a past history of VTE or with a known inherited thrombophilia, it is best to refer her prior to a planned pregnancy for optimum prophylaxis throughout the pregnancy. Refer all women who are on **warfarin** as this will have to be stopped or replaced by heparin before the 7th week of conception, depending on her risk of VTE.
- Medical anticoagulation is the treatment of choice for acute VTE. Subsequently, surgical interventions may be considered: patients suffering from recurrent PEs despite adequate anticoagulation (or where there is an absolute contraindication to anticoagulation) may benefit from placement of a temporary caval filter and in those cases where there is limb or life threatening embolus, a surgical embolectomy or thrombus fragmentation may be attempted.
- Anticoagulation is by far the most common treatment option. Heparin is the most frequently used drug, being non-toxic to the fetus (it does not cross the placental barrier). However, its main disadvantages are that it has to be parentally administered and on the long-term, may give rise to heparin-induced osteoporosis and **thrombocytopenia**. In some patients, it can also provoke a painful, localised allergic reaction on administration. Warfarin is the other treatment option in the postnatal patient but it must be avoided antenatally as it is teratogenic and can also cause **placental abruption** and fetal / neonatal haemorrhage.
- In clinically suspected DVT or PE, treatment with unfractionated heparin or **low molecular weight heparin** should be given until the diagnosis is excluded by objective testing, unless treatment is strongly contraindicated.

## Initiating treatment

There are several different types of heparin to choose from:

- **Low molecular weight heparin:** this is the drug of choice. It has been shown to be more effective than unfractionated heparin with lower mortality and fewer haemorrhagic complications in the initial treatment of DVT in nonpregnant subjects. LMWHs are as effective as unfractionated heparin for treatment of PE. LMWH have greater antithrombotic (antifactor Xa) activity than anticoagulant (antifactor IIa) activity. aPTT is unaffected by this drug. The exact dose will depend on the manufacturer's recommendations but this is based on the patient's early pregnancy weight and should be administered subcutaneously twice daily. There should be clear local guidelines for the dosage of LMWH to be used
- **Intravenous unfractionated heparin:** this is an extensively used drug in the acute management of VTE, particularly massive PE with cardiovascular compromise. It is initiated with a loading dose of 5000 international units (80 U / Kg) followed by a continuous infusion of 15 – 20 iu / kg / hour depending on APTT measurements (daily - at least), the first of which is taken 6 hours post loading dose. aPTT is kept at 1.5 to 2.5 times normal. This corresponds to a circulating heparin level of 0.3 u / ml and antifactor Xa trough level of 0.7 u / ml. Thus, there is the benefit of accurate drug administration but it has been demonstrated that there are a number of difficulties with accurate APTT measurement, particularly late in pregnancy when interpretation of the results can be problematic. Prolonged use in pregnancy may give rise to the problems described above.
- **Subcutaneous unfractionated heparin:** this has been shown to be as effective as the intravenous form. It is administered as a 5000 iu bolus and subsequent 15,000 - 20,000 iu doses at 12 hourly intervals. The APTT needs to be checked and is best done mid-way between the 12 hourly doses, once every 24 hours. A target of 1.5-2.5 times the control should be aimed for.
- risk factors (surgery, long-haul flights), even several weeks after an uneventful vaginal delivery.

## Heparin dosing regimens

Dose	Regimen
Mini-dose UFH	UFH 5000 U subcutaneously every 12 h
Moderate-dose UFH	UFH sc every 12 h in doses adjusted to target an anti-Xa level of 0.1 to 0.3 U/mL
Adjusted-dose UFH	UFH sc every 12 h in doses adjusted to target a midinterval aPTT into the therapeutic range
Prophylactic-dose LMWH	Enoxaparin 40 mg once daily or 30 mg twice daily 1 mg = 100 u. Peak antifactor Xa activity occurs within 3 – 5 hours of administration and 50% activity disappears within 6 hours of stopping the drug. For therapeutic anticoagulation, Enoxaparin I used in doses of 30 to 60 mg twice daily. Tinzaparin 4500 U once daily Dalteparin 5000 U once daily
Weight-adjusted dose LMWH	Enoxaparin 1 mg/kg (100 U / kg) twice daily or 1.5 mg/kg once daily Thromboprophylaxis needs 2500 to 5000 U OD or BD dose Dalteparin 100 U/kg every 12 h or 200 U/kg every 24 h Tinzaparin 175 U/kg once daily

**U indicates units.**

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Additionally, the leg should be elevated and a graduated elastic compression stocking applied to reduce oedema. Mobilisation with graduated elastic compression stockings should be encouraged.

### Maintenance therapy

1. **During pregnancy:** [heparins](#) are the maintenance treatment of choice. Dose-adjusted subcutaneous, unfractionated heparin or subcutaneous LMWH are effective alternatives to [oral anticoagulants](#) in maintenance treatment of VTE.

Subcutaneous LMWH appears to have advantages over APTT-monitored unfractionated heparin in the maintenance treatment of VTE in pregnancy. The simplified therapeutic regimen for LMWH tends to be more convenient for patients, minimising blood tests (routine platelet counts are not required and levels of anti-Xa will only need to be monitored where there are extremes of weight: <50kg or >90kg) and allowing out-patient treatment. Women should be taught to self-inject and can then be managed as out-patients until delivery.

If unfractionated heparin is used, monitor platelet count at least every other day for the first 14 days or until treatment is stopped (whichever comes first). Seek specialist advice if the patient develops heparin-induced thrombocytopenia or a heparin allergy and requires continuing anticoagulant therapy. She should be managed with the heparinoid, danaparoid sodium or fondaparinux, under specialist supervision.

2. **Labour:** when the patient thinks she is going into labour, she should stop injecting and get in touch with the delivery ward staff who will manage the anticoagulation throughout labour and immediately post delivery. Heparin is discontinued during labour. If aPTT is abnormal and there is a risk of bleeding, incremental doses of protamine may be used titrated to surgical hemostasis. Alternatively, planned elective [induction of labour](#) or [caesarean section](#) at least 12 hours after prophylactic dose low molecular weight heparin or 24 hours after therapeutic dose low molecular weight heparin can be considered. As these patients are at high risk of haemorrhage, they will be managed with intravenous unfractionated heparin throughout this time. Regional anaesthetic or analgesic techniques should not be undertaken until at least 24 hours after the last dose of therapeutic LMWH.
3. **Postpartum period:** depending on the patient's individual circumstances, she may be managed with ongoing heparin treatment or warfarin postpartum. If she opts for warfarin, this needs to be avoided until at least day 3 post partum with an INR check at day 2 of warfarin treatment: aim for an INR between 2 and 3. Continue



heparin treatment until there have been two successive readings of an INR > 2. Although these drugs are detectable in breast milk, all are safe for use during breast feeding because warfarin metabolites are inactive and heparin is not absorbed through the gastrointestinal tract.

Postnatal review for women who develop VTE during pregnancy or the puerperium should, whenever possible, be at an obstetric medicine clinic or a joint obstetric haematology clinic.

### Stopping treatment

In theory, therapy should be continued for six months as would be the case for non-pregnant patients. However, the post-partum state is a period of physiological fluctuation of coagulation factors. Therefore, current advice is to continue therapy for at least 6-12 weeks post partum or until at least three months of therapy have been completed. At that point, the patient should be assessed for the presence of ongoing risk factors for a VTE prior to making the decision to stop anticoagulation therapy.

### Complications

#### Thrombophilia and placental vascular complications

- **Fetal loss:** although the figures are likely to be small (there are not many studies), there is thought to be a doubling of risk of fetal loss in women with genetic thrombophilia.
- **Intrauterine growth restriction:** a specific association between this and thrombophilia has not been identified but chronic abruption and extensive placental infraction have been noted to occur more frequently in these patients.
- **HELLP syndrome:** this may be associated with certain forms of thrombophilia.

#### Post thrombotic syndrome

Up to 60% of patients who have suffered from a DVT go on to have post thrombotic syndrome up to 12 months following the acute event. This arises from damage to the lumen of the vein following the presence of a thrombus. Subsequently, patients manifest symptoms and signs akin to those of varicose veins: aching, swollen legs, **pruritis**, **dermatitis** and **hyperpigmentation** of the affected area. Ulceration and cellulitis may complicate the picture. There is emerging evidence to suggest that compression stockings worn on the affected leg for at least 2 years after the acute event reduces the risk of developing post thrombotic syndrome. PE is the other complication of DVTs and is discussed above.

#### Other complications

Prolonged unfractionated heparin use during pregnancy may result in **osteoporosis** and fractures.

#### Prevention: prophylaxis

There are obvious risks associated with ante-natal anticoagulation and the decision to go ahead with prophylactic thrombolysis is one made jointly by the obstetricians and haematologists. Guidance suggested by the Royal College of Obstetricians and Gynaecologists suggests:

#### ACCEPTABLE REGIMEN FOR ANTICOAGULANT THERAPY IN OBSTETRIC PATIENTS

CLINICAL SITUATION	ANTICOAGULATION REGIMEN
Varicosities	None
Superficial Thrombophlebitis	None
Hypercoagulable states	Therapeutic
PREVIOUS DVT / PE	

Post trauma	None
Oral Contraceptives	Therapeutic
Antiphospholipid Antibody syndrome	Prophylactic or Therapeutic
Unexplained	Prophylactic
Recurrent	Prophylactic
DVT / PE Current Pregnancy	Therapeutic until 6 to 12 weeks postpartum or therapeutic for 4 to 6 months and then prophylactic until 6 to 12 weeks postpartum
DVT Prior Pregnancy	Prophylactic beginning in early pregnancy
PE Prior Pregnancy	Prophylactic or Therapeutic

- Regardless of their VTE risk, dehydration and immobilisation of the patient ante-natally, during labour and post-partum should be avoided.
- If a decision is made to go ahead with prophylaxis, this should be initiated as early in the pregnancy as possible (post-partum prophylaxis should commence as soon after the delivery as is practically possible).
- Women with a history of a VTE but no thrombophilia should be offered LMWH for 6 weeks post partum (there is some debate about the ante-natal period owing to conflicting evidence) unless the VTE was clearly associated with a (now fully resolved) risk factor. If she has had multiple VTEs or if there is a strong family history of VTEs in a first degree relative, ante-natal prophylaxis should also be offered.
- Women with a history of VTE and known thrombophilia should be offered LMWH prophylaxis ante-natally and for at least 6 weeks post partum.
- Women with inherited thrombophilia but no previous VTE may or may not qualify for ante/post natal prophylaxis depending on the nature of the thrombophilia and the whether there are associated risk factors.
- Patients with acquired thrombophilia (antiphospholipid syndrome) generally should receive prophylaxis throughout and after pregnancy in most but a handful of cases
- Women without previous VTE or thrombophilia: if there are three or more persisting risk factors, antenatal thromboprophylaxis should be considered through to 3-5 days post-partum. Notably, if the patient is over 35, has a BMI of over 30 or a body weight of over 90kg, prophylaxis is almost mandatory, especially in the immediate post partum period.

## **ANAESTHETIC MANAGEMENT**

In such cases the anaesthesiologist must consider the risk versus the benefits of RA. Even in healthy patients insertion of epidural needle causes some amount of bleeding in 5 to 40% of patients. However studies do exist reporting the safe use of CNB in patients receiving thromboprophylaxis.

The ASRA guidelines form the basis of safe practice of RA in these patients .

The University of Maryland Guidelines based on the ASRA recommendations use the following steps of management in patients with PTE and VTE

Patients receive full anticoagulation with SC heparin to maintain aPTT at twice normal values.

Heparin is discontinued at start of active labour. Wait till aPTT is normal. Till then opioid analgesia is offered for pain relief.

Use of LMWH precludes the use of any form of CNB at least 12 hours after the last dose. So it always better to substitute LMWH with unfractionated heparin as soon as possible.

Protamine may be administered in select patients needing emergency LSCS. Protamine has no role in patients on LMWH

### **RA IS ADMINISTERED ONLY IF COAGULATION PROFILE IS NORMAL**

If coagulation profile is abnormal give GA. There is a risk of airway bleeding.

Anticoagulation should be reinstated after delivery in consultation with obstetricians. LMWH is restarted after a delay of at least 6 to 8 hours after administration of CNB and 2<sup>nd</sup> dose to be given only 24 hours after 1<sup>st</sup> dose. Catheter removal must be undertaken 24 hours after last dose of LMWH. Detection of blood on needle / catheter insertion or removal must postpone LMWH dose by 24 hours.

Neuraxial catheters can be safely maintained in patients on OD dose of LMWH. Catheter must be removed 24 hours after last dose. Any dose after atraumatic catheter removal must not be initiated before 2 hours have elapsed.

Monitor the patient for signs of epidural hematoma which include 1) Severe unremitting backpain 2) Neurologic deficit including bowel or bladder dysfunction or radiculopathy 3) tenderness over spinous and paraspinal area 4) Unexplained fever. Suspicion of hematoma must lead to immediate imaging of spine and neurosurgical consultation.

To sum it up, in obstetric patients many embolic events occur intrapartum or postpartum and may lead on to mortality. The anaesthesiologist must be involved in the resuscitation and management of the patients. The key to effective management lies in early recognition, diagnosis and treatment.