Management of Patient Anticoagulant Therapy

Training Programme

Adapted from the original programme developed by Anticoagulation Team, Derby Hospitals NHS Foundation Trust
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INTRODUCTION

In order for practitioners to monitor and dose patients safely within the anticoagulation services a recognised training programme must be undertaken and the nurse assessed as competent. The need to have a certain standard of expertise is common to all health care professionals and is important to protect the public to ensure quality care and to establish the credibility of the profession.

This document contains research-based information to help gain or consolidate the theoretical background in support of safe dosing and monitoring of anticoagulant therapy. It builds on the principles outlined within the United Kingdom Central Council for Nurses, Midwives & Health Visitors (UKCC) advisory papers, ‘The Scope of Professional Practice’ (1992), and ‘Guidelines for Professional Practice’ (1996); and the Nurses and Midwifery Council ‘Code of Professional Conduct’ (April 2002). This programme also reflects the guidelines on expanding the scope of professional practice.

This document provides basic information required to provide this service and must not be seen as a comprehensive guide to anticoagulation management. The document assumes that each practitioner will only work within the scope of their Professional Practice and meet the requirements of codes of ethics and good practice guidelines issued by national bodies. Clinical management of patients will respect and adhere to the Derbyshire CCG guidelines on oral anticoagulation1 and appropriate national guidelines issued by the British haematology Society2 (BSH) and the NPSA. All providers should follow the recommendations of the NPSA Anticoagulation Patient safety alert 18.3 All service providers will need to name an individual as the clinical lead who will be responsible for ensuring that the service is delivered in accordance with the specification and any other guidelines issued by Derbyshire CCGs.

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3 National Patient Safety Agency alert 18, 2007 (www.npsa.nhs.uk)
SECTION 1

2.1 CRITERIA FOR ENTRY ONTO THE ANTICOAGULATION TRAINING PROGRAMME

Any practitioner who wishes to provide the Anticoagulation management level 4 local enhanced service and undertake the training will be required to fulfil the following criteria:

- Be a registered GP or Pharmacist
- Be on the NMC register for Nurses, Midwives & Health Visitors.
- Be employed within an organisation signed up and committed to providing the anticoagulation service.

For the purpose of this training programme anticoagulant therapy refers to oral anticoagulants (vitamin K antagonists) namely Warfarin.

1.2 TRAINING AND ACCREDITATION CRITERIA

Training should be undertaken to ensure all provider staff or those contracted by the Service providers to provide the service have the necessary skills and knowledge to conduct anticoagulation monitoring and use the equipment supplied. To provide the level 4 service the person(s) running the clinic will have to:

- Accept accountability for developing the necessary competencies
- Successfully undertake the required training
- Complete post-course certification. This can be either of the following:
  - Maintaining patients on anticoagulants: how to do it
  - Anticoagulation; managing patients, prescribing and problems. CPPE 2007. An open learning resource for pharmacists and pharmacy technicians.
    http://www.cppe.manchester.ac.uk
- Complete a period of clinical supervision and work place summative assessment within the anticoagulation clinic if not previously provided a level 4 service
- Meet all the criteria specified in the most up to date CCG specification.
1.4 LEARNING OUTCOMES OF THE PROGRAMME

Following theoretical and practical input and a period of supervised practice the practitioner will be competent in the management of patient anticoagulant therapy.

Objectives

On completion of the training and education programme the practitioner will have knowledge and understanding of:

- The importance of patient confidentiality and how to ensure that this is fully maintained.
- When to seek advise from the haematologists
- The principles of anticoagulant therapy
- Methods, principles and purpose near patient testing relating to oral anticoagulant management
- Relevant local guidelines, policies and procedures and who is responsible for these.
- Factors affecting dosage and time interval of testing for individual patients
- The interactions of anticoagulants with other drugs and substances
- Oral anticoagulant reversal
- Requirements and indications of non-compliance with management regimes
- Medico-legal issues associated with management regimes
- Possible complications or side effects of anticoagulant therapy and how to recognise them
- The use of clinical audit in relation to anticoagulant dosing
- Specific national/international guidelines

The theoretical elements of the course should ensure that the clinical lead of each primary care service has:

- The ability to safely manage a primary care based anticoagulation clinic using near patient testing for INR estimating, interpreting INR results and assessing the dose of oral anticoagulation in order to maintain results within their appropriate therapeutic ranges;
• A comprehensive understanding of the conditions requiring oral anticoagulation therapy and the target ranges for warfarin therapy;
• The ability to evaluate which target INR is required when treating different conditions;
• An understanding of the pharmacology of warfarin and determine the relevant medication, side effects, antidotes, interaction and dosing;
• The ability to critically analyse all aspects of anticoagulation management and therefore evaluation aspects for safe practice.

All service providers will be expected to meet the following appropriate Anticoagulation competencies 1,2,3, and 6 outlined by the NPSA⁵:

- Initiating anticoagulant therapy (1)
- Maintaining oral anticoagulant therapy (2)
- Managing anticoagulants in patients requiring dental surgery (3)
- Reviewing the safety and effectiveness of an anticoagulant service (6)

The practical aspects of the training will involve providing and testing the following competencies:

<table>
<thead>
<tr>
<th>Competency</th>
<th>Method of assessment</th>
</tr>
</thead>
<tbody>
<tr>
<td>The ability to take capillary blood samples demonstrating good finger-prick technique</td>
<td>Demonstration of good finger-prick technique in taking samples from 12 patients. Achievement of the desired standard in a formal assessment.</td>
</tr>
<tr>
<td>The ability to use Coagulometer correctly</td>
<td>Demonstration of competency by testing the INR of 12 patients. Achievement of the desired standard in a formal assessment.</td>
</tr>
<tr>
<td>The ability to use the computerised decision support software (CDSS)</td>
<td>Achievement of the desired standard in a formal assessment.</td>
</tr>
</tbody>
</table>

Prior to service roll out, service providers will need to ensure they have the facilities, medical experience, training and competence as is necessary to enable them to provide anticoagulation management and that they will be able to meet the criteria as laid down in the specification.
SECTION 2

Anticoagulation Workbook

Name of practitioner .................................................................

Name of clinical lead ..............................................................
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Introduction
The History of Warfarin
Historical

The discovery of warfarin was centered in Canada and the United States. In the early part of the 20th century, farmers in the northern prairie states of Canada and the USA began planting sweet clover plants imported from Europe.

Although the sweet clover proved to be nutritious when used as fodder, it also brought a fatal disease which decimated cattle herds and horrified farmers: sweet clover disease, in which affected cattle developed relentless, spontaneous bleeding.

Schofield, a veterinary pathologist in Alberta, reported in 1921 that the disease was caused by consumption of spoilt sweet clover hay.

The fresh plant was known to contain the compound coumarin, which was not pathogenic.

The mystery of why spoilt hay caused the disease was solved by Karl Paul Link and his co-workers in 1940: in mouldy hay, coumarin is oxidised to 4-hydroxycoumarin and then coupled with formaldehyde and another coumarin moiety to form dicoumarol, an anticoagulant. This was responsible for the disease. Dicoumarol was patented in 1941 and was therapeutically used as an anticoagulant.

 Renewed impetus to the development of oral anticoagulants came again from Link in 1946. Working on the problem of rodent control, he found that dicoumarol was too weak and unreliable as a rodenticide. The most potent compound Link and co-workers discovered was 3-(2-acetyl-1-phenylethyl)-4-hydroxycoumarin.
Patent rights were assigned to Link's research benefactors, the Wisconsin Alumni Research Foundation, from which the name warfarin was derived. In 1948, warfarin was launched as the ideal rat poison.

In 1951, a navy recruit unsuccessfully attempted suicide with 567 mg of warfarin. His surprising full recovery induced research into the anticoagulant potency of warfarin in humans. It was found to be far superior to dicoumarol. Clinicians quickly discarded dicoumarol in favour of “rat poison” warfarin: it was introduced commercially in 1954. In that same year, President Eisenhower was treated with warfarin following a heart attack.

Today, warfarin is the standard treatment for long term oral anticoagulant therapy.
2.1 Haemostasis

Contact Factor Pathway

- Kallikrein (HK)
- XII
- Prekallikrein
- XHa
- XI Ca
- X Ca
- IXa
- VIIIa
- VIII
- Ila
- V Ca
- IIa
- II

Tissue Factor Pathway

- Tissue Damage
- Tissue Factor
- Xa
- VIIa
- VII
- Xa
- IIa
- II
- Fibrinogen
- Fibrin Monomer
- Soft Fibrin Clot
- Hard Fibrin Clot
SECTION 2.1 Haemostasis

Aim  To understand the processes behind the ability of the body to control the clotting mechanisms of the blood.

After completing this section of the Anticoagulation Training Package you will;

i) Be able to define the term Haemostasis

ii) Be able to determine possible causes of blood clots forming within vessels of the body

iii) Be aware of the coagulation factors involved in the haemostatic mechanism

iv) Be aware and understand the action of the Clotting Cascade

v) Understand what is meant by the term International Normalised Ratio (INR)

vi) Understand how anticoagulants exert an effect within the clotting cascade
Haemostasis

Haemostasis is the ability of the body to control blood flow after vascular injury. It is a natural process that allows blood to thicken and form a clot to stop bleeding. Eventually, the clot helps form a protective scab over the healing wound, without this protective mechanism people would bleed to death from even minor cuts.

Sometimes, however, blood clots can form even though a person has not been wounded in any way. Under certain conditions, a clot can form in an artery, which could block the blood flow and cause a heart attack or a stroke. A clot could also form in one of the heart's chambers, travel through the blood stream and lodge itself in an organ or artery, cutting of blood supply from that point and thus causing an embolism.

Blood clots usually form when blood flow becomes sluggish, as when there is roughness or scar tissue along the interior walls of a blood vessel that slows blood flow.

Some underlying conditions that could contribute to the formation of blood clots include:

- Atherosclerosis
- Heart attack
- Trauma to a blood vessel due to accident or medical procedure
- Hypertension
- Atrial fibrillation
- Congestive heart failure
- Valvular heart disease

In addition, there are a number of risk factors that can increase the chances of developing a blood clot. These include:

- Smoking
- Obesity
- Prolonged lack of exercise
- Genetics
- Advanced age
- Oral contraceptive use

Oral anticoagulants such as warfarin are specially indicated for treatment of these blood clots occurring within vessels of the body.
**Blood Coagulation**

To understand how warfarin exerts its anticoagulant effect, a general understanding of the mechanism of blood clotting is needed.

A blood clot forms as a result of the concerted action of some 20 different substances, most of which are plasma glycoproteins and are designated by Roman numbers.

<table>
<thead>
<tr>
<th>Coagulation Factors</th>
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<tr>
<td>Factor</td>
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<td>XIII</td>
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</table>

Blood coagulation represents a series of sequential interactive events that lead to the repair of the vascular system following injury. The mechanism is traditionally distinguished in 2 pathways: **the intrinsic and extrinsic pathways**.

The intrinsic pathway is defined as a cascade that utilises only factors that are soluble in the plasma, whereas the extrinsic pathway consists of some factors that are insoluble in plasma, e.g. membrane-bound factors like Factor VII.

The characteristic feature of the coagulation pathway is that upon activation, the individual glycoprotein serves as an enzyme to convert the zymogen (inactive) form of the succeeding glycoprotein to its protease (active) form in the presence of Ca\(^{2+}\) and an appropriate phospholipid membrane. The activated form of a glycoprotein is
The goal of both pathways is to produce Factor Xa, which then catalyses a key transformation: the conversion of Prothrombin (factor II) to thrombin (factor IIa).

Factor Xa will catalyse this reaction only if prothrombin is bound to a phospholipid membrane in presence of factor Va and Ca$^{2+}$. Thrombin, in turn, cleaves fibrinogen, an inactive circulating plasma protein, to form soluble fibrin monomers. These monomers spontaneously aggregate to form an unstable network.

Thrombin also activates Factor XIII, which then causes cross linking between the fibrin molecules to form a stable mesh like structure which traps blood cells, forming a clot.
**Warfarin is a vitamin K antagonist.** It produces its anticoagulant effect by interfering with the vitamin K cycle. Specifically, it interacts with the KO reductase enzyme so that vitamin KO cannot be recycled back to vitamin K.

This leads to a depletion of vitamin KH$_2$, thereby limiting the γ- carboxylation of the coagulation factors mentioned above. Factors like prothrombin are not carboxylated, and cannot effectively bind to phospholipid membranes.

Its activation by Factor Xa is not effected. Thus blood coagulation is limited.

The laboratory test to determine the coagulation ability of blood is the Prothrombin Time (PT). This reflects the depression of vitamin k dependent factors. However, the results of this test depends on a variety of factors, including the actual reagents used.

A system of standardising the PT in oral anticoagulant control was introduced by the World Health Organisation (WHO) in 1983. It is based on the determination of an International Normalised Ratio (INR) which provides a common basis for communication of PT results and interpretation of therapeutic ranges.

The INR is calculated as:

\[
\text{INR} = \left( \frac{\text{patient PT}}{\text{control PT}} \right)^{\text{ISI}}
\]

where ISI = International Sensitivity Index and is the correction factor which includes effects of the reagent used, etc.

The anticoagulant efficacy of warfarin is influenced by pharmacokinetic factors.

Warfarin is a racemic mixture and is well absorbed orally. It is transported in the blood by albumin, a plasma protein. It is metabolised in the liver by the cytochrome P450 family of enzymes.
SECTION 2.1  HAEMOSTASIS

Q1.  How does an anticoagulant like warfarin work?
A.

Q2.  How soon after administration can warfarin be detected in the plasma?
A.

Q3.  When can peak concentrations of warfarin be detected in the plasma?
A.

Q4.  To which plasma protein does warfarin bind?
A.

Q5.  Where is warfarin metabolised and excreted?
A.

Q6.  Approximately how many days are required for the antithrombotic effect of warfarin to take effect.
A.

Q7.  Why does the effect of warfarin vary so much when first initiated?
A.
Q8. What is the goal of anticoagulant therapy?
A.

Q9. What is the average maintenance dose of warfarin?
A.

Q10. What is the role of Vitamin K in Blood Coagulation?
A.

Q11. List 2 other Oral anticoagulants used in the UK in addition to Warfarin
A.

Q12. Warfarin is able to pass through the placenta
   True or False

Q13. Warfarin is able to pass into breast milk
   True or False
I confirm that ………………………………………………………… (candidate’s name) has completed the exercises within Section 2.1. ‘Haemostasis’ to the required standard.

Signature ……………………………………………………………….. (clinical lead)

Date ………………………………………………………………………..
Section 2.2

Deep Vein Thrombosis & Pulmonary Embolism
SECTION 2.2 Deep Vein Thrombosis and Pulmonary Embolism

Aim To understand the causes, symptoms and possible complications of deep vein thrombosis and pulmonary embolism and the implications of treatment of with oral anticoagulants.

After completing this section of the Anticoagulation Training Package you will:

i) Be able to determine the causes of DVT and PE

ii) Recognise the symptoms of DVT and PE

iii) Be aware of the tests used to diagnose DVT and PE

iv) Be aware of how DVT and PE is treated in both the acute and chronic phase

v) Be able to educate a patient in the use of oral anticoagulation treatment in regard to these conditions

vi) Be aware of measures used which attempt to prevent venous thrombosis and pulmonary embolism following surgery or periods of immobility
Deep Vein Thrombosis (DVT) is a condition where there is a blood clot in a deep vein (a vein that accompanies an artery).

Deep Vein Thrombosis affects mainly the deep veins embedded in the muscles in the lower leg and the thigh. It involves the formation of a clot (thrombus) in the larger veins of the area. It causes permanent damage to the vein. This thrombus may interfere with circulation of the area, and it may break off and travel through the blood stream (embolize). The embolus thus created can lodge in the brain, lungs, heart, or other area, causing severe damage to that organ.
Q1. What do you think are possible risk factors for the development of a DVT?
A.

Q2. Identify the 3 factors which comprise Virchow's Triad
A.

Q3. Why do Oestrogens increase the risk of a clot forming?
A.

Q4. Explain what is meant by a "Silent DVT"
A.

Q5. Where would a Proximal DVT occur?
A.

Q6. Where would a Distal DVT occur?
A.
Q7. DVT’s on which veins (Proximal or Distal) are associated with a high risk of a Pulmonary Embolism (PE)?
A.

Q8. What are the usual clinical symptoms of a DVT?
A. i) 
   ii) 
   iii) 
   iv) 

Q9. List the various tests used for diagnosing a DVT
A. i) 
   ii) 
   iii) 

Q10. Which of these test is considered the most specific?
A.

Q11. What do D-dimers measure?
A.
Q12. What is the loading dose of warfarin within the Derbyshire county NHS Foundation Trusts for a person diagnosed with a DVT?

A. 1st Day
   2nd Day
   3rd Day

Q13. If a person is suspected of having a DVT should they be commenced on warfarin immediately?

A.

Q14. Why should Low Molecular Weight Heparin (LMWH) not be stopped when the warfarin is started?

A.

Q15. When a patient has completed their treatment duration how should treatment be stopped?

A.

Q16. How do TED stockings work?

A.
Q17. Having reached the end of their treatment a patient is reluctant to stop taking their warfarin as they fear they may suffer another clot. How would you advise them?

A.

Q18. You are contacted by a patient who has recently been diagnosed (4 weeks ago) with a DVT. They inform you that they are planning a long haul flight and want to know if this is alright. What would you do?

A.

Q19. The use of appropriate thromboprophylaxis can reduce the risk of developing DVT after hip replacement to between ............ and ..........% of patients.

<table>
<thead>
<tr>
<th>Range</th>
</tr>
</thead>
<tbody>
<tr>
<td>5 - 10%</td>
</tr>
<tr>
<td>10 - 15%</td>
</tr>
<tr>
<td>15 - 20%</td>
</tr>
<tr>
<td>20 - 30%</td>
</tr>
</tbody>
</table>

Q20. Until the recent introduction of guidelines produced by the Royal College of Obstetricians and Gynaecologists (RCOG), thromboembolism was the single biggest killer of which group of patients? .........................
TRUE or FALSE  (Please indicate)

1. Most DVT starts in the thigh.
   True        False

2. Calf DVT extends to the thigh in about 50% of cases.
   True        False

3. Isolated calf DVT usually causes symptoms.
   True        False

4. 75% of DVT after orthopaedic surgery occur in the operated leg.
   True        False

5. Most clots associated with surgery start 1st POD (Post Operative Day)
   True        False

6. Risk of DVT is about the same for various types of surgery.
   True        False

7. Prophylaxis facilitates spontaneous lysis of perioperative DVT.
   True        False

8. Clinical diagnosis of PE is established in the majority of patients who die from PE.
   True        False
9. The majority of patients with symptomatic proximal DVT and NO chest symptoms have PE on lung scans.

True  False

10. Cancer is an independent risk factor for DVT.

True  False

I confirm that .............................................................. *(candidate’s name)*
has completed the exercises within Section 2.2(i) ‘Deep Vein Thrombosis’ to the required standard.

Signature .............................................................. *(Clinical lead)*

Date ..............................................................
2.2(ii) Pulmonary Embolism

Pulmonary emboli usually arise from the thrombi originating in the deep venous system of the lower extremities; however, rarely they may originate in the pelvic, renal, or upper extremity veins and the right heart chambers. After travelling to the lung, large thrombi lodge at the bifurcation of the main pulmonary artery or the lobar branches and cause haemodynamic compromise. Smaller thrombi continue travelling distally, occluding a smaller vessel in the lung periphery. These are more likely to produce pleuritic chest pain by initiating an inflammatory response adjacent to the parietal pleura. Most pulmonary emboli are multiple, and the lower lobes are involved more commonly than the upper lobes.

Acute respiratory consequences of PE include increased alveolar dead space, pneumoconstriction, hypoxemia, and hyperventilation. Later, 2 additional consequences may occur: regional loss of surfactant and pulmonary infarction. Arterial hypoxemia is a frequent but not universal finding in patients with acute embolism. The mechanisms of hypoxemia include ventilation-perfusion mismatch, intrapulmonary shunts, reduced cardiac output, and intracardiac shunt via patent foramen ovale. Pulmonary infarction is an uncommon consequence because of the bronchial arterial collateral circulation.

Following the initiation of anticoagulant therapy, the resolution of emboli occurs rapidly during the first 2 weeks of therapy. Significant long-term nonresolution of emboli causing pulmonary hypertension or cardiopulmonary symptoms is uncommon.
Q1. What are the risk factors for Pulmonary Embolism?
A

Q2. How is a Pulmonary Embolism treated?
A

Q3. Is Pulmonary Embolism preventable if so how?
A

Q4. Which condition would you say is more dangerous to the patient, a DVT or PE?
A

Q5. Pulmonary Emboli result from Deep Vein Thromboses
True or False

Q6. When suspected of having a PE a patient will be immediately started on Warfarin
True or False
Q7. **What is the loading dose of warfarin within the Derbyshire county NHS Foundation Trusts for a person diagnosed with a PE?**

A. 1st Day
   
   2nd Day
   
   3rd Day

Q9. **Complete the Table shown below**

<table>
<thead>
<tr>
<th>Condition</th>
<th>Target INR</th>
<th>Duration</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pulmonary Embolism</td>
<td></td>
<td>6 months</td>
</tr>
<tr>
<td>Proximal Vein Thrombosis</td>
<td>2.5</td>
<td></td>
</tr>
<tr>
<td>Calf Vein Thrombosis</td>
<td>2.5</td>
<td></td>
</tr>
</tbody>
</table>

I confirm that ..................................................  (candidate’s name) has completed the exercises within Section 2.2(ii) ‘Pulmonary Embolis’ to the required standard.

Signature ..............................................................  (Clinical lead)

Date .................................................................
Section 2.3

Atrial Fibrillation
SECTION 2.3 Atrial Fibrillation (AF)

Aim To understand the causes, symptoms and possible complications of atrial fibrillation and the implications of treatment with oral anticoagulants.

After completing this section of the Anticoagulation Training Package you will;

i) Be able to determine the causes of AF
ii) Recognise the symptoms of AF
iii) Be aware of how to diagnose AF
iv) Be aware of how AF is treated
v) Be able to educate a patient in the use of oral anticoagulation treatment
vi) Be aware of both pharmacological and non-pharmacological methods of cardioverting AF patients back into normal sinus rhythm
v) Be aware of the possible complications associated with AF
Understanding a normal heartbeat

The heart has four chambers - two atria and two ventricles. The walls of these chambers are mainly made of special heart muscle. The chambers have to contract (squeeze) in the correct order for the heart to pump blood correctly with each heartbeat.

The sequence of each normal heartbeat is as follows.

- The sino-atrial node (SA node) in the right atrium is a tiny in-built 'timer'. It fires off an electrical impulse at regular intervals. (About 60-80 per minute when you rest, and faster when you exercise. This controls the heart rate.) Each impulse spreads across both atria. This causes them to contract and pump blood through one way valves into the ventricles.

- The electrical impulse gets to the atrioventricular node (AV node) at the lower right atrium. This acts like a 'junction box' and the impulse is delayed slightly. Most of the tissue between the atria and ventricles does not conduct the impulse. However, a thin band of conducting fibres called the atrioventricular bundle (AV bundle) acts like 'wires' and carries the impulse from the AV node to the ventricles.

- The AV bundle splits into two - a right and left branch. These then split into many tiny fibres (the Purkinje system) which conducts the electrical impulse throughout the ventricles. This makes the ventricles contract and pump blood through one way valves into large arteries.

The artery going from the right ventricle (pulmonary artery) takes blood to the lungs.

The artery going from the left ventricle (aorta) takes blood to the rest of the body.
Atrial Fibrillation (AF):

This is a rapid, irregular heart rhythm (arrhythmia) caused by abnormal electrical signals from the upper chambers of the heart (atria). The normal heart rate is 60-100 beats per minute. AF is marked by a rapid heart rate of 100-175 beats per minute. Instead of contracting normally, the atria quiver. There is a chance that blood may pool in the atria, which can lead to the formation of blood clots. These can break off and cause emboli or stroke.

Q1. List as many causes of Atrial Fibrillation as you can.
(There are as many as 30 see how many you can find)
Q2. What is the most common cause atrial fibrillation?
A.

Q3. What is the main purpose of treating Atrial Fibrillation with warfarin?
A.

Q4. What are the Risk Factors which may increase the risk of stroke in patients with Atrial Fibrillation
A.

Q5. For how long should the patient undergo warfarin therapy prior too and post Cardioversion?
A.

Q6. What do we mean by Paroxysmal AF
A.

Q7. What do we mean by Persistant AF
A.

Q8. What do we mean by Permanent AF
A.
Q9. How common is Atrial Fibrillation?
A.

Q11. List four symptoms of atrial fibrillation?
A.

Q12. What is the Main complication of Atrial Fibrillation?
A.

Q13. What are the less common complications of Atrial Fibrillation?
A.

Q14. There are 3 Categories which identify the risk of having a stroke, High, Moderate and Low. What characteristics place an individual in each group.

*High risk* means that, without treatment, you have about a 6-12 in 100 chance (sometimes higher) of having a stroke in the next year. People in the high risk group are those:

i)
ii) Moderate risk means that you have about a 3-5 in 100 chance of having a stroke in the next year. People in the moderate risk group are those:

i) iii) 

iv) 

Low risk means that you have about a 2 in 100 chance or less of having a stroke in the next year. People in the low risk group are all people;

Q15. List the 3 treatment options for Atrial Fibrillation?

A. 

i) ii) iii)
Q16. What drugs can be used to slow the heart down?
A.

Q17. How do these drugs work?
A.

Q18. How does Digoxin work specifically?
A.

Q19. What non pharmacological measures can be taken to treat Atrial Fibrillation?
A. i) ii) iii) iv)

Q20. In what situations is Cardioversion more likely to be considered?
A.

Q21. In what situation is Cardioversion not likely to be considered?
A.
Q22. How does DC Cardioversion work?
A.

Q23. Why is it important for a patient to be adequately anticoagulated prior to DC Cardioversion?
A.

Q24. The patient should be anticoagulated for a minimum of ________ weeks prior to the DC cardioversion and for a minimum of ________ weeks post cardioversion.

Q25. Can successful DC cardioversion be determined immediately?
Yes No (Please indicate)

Q27. Identify one drug which is used in what is called "Chemical Cardioversion"
A.

Q28. Give a definition of Myocardial Infarction
A.

Q29. Give a definition of Cardiomyopathy
A.
Q30. Give a definition of Rheumatic Heart Disease

A.

I confirm that ................................................................. (candidate’s name) has completed the exercises within Section 2.3 ‘Atrial fibrillation’ to the required standard.

Signature ............................................................................. (Clinical lead)

Date .......................................................................................
Section 2.4

Heart Valve Replacement
SECTION 2.4 Heart Disease and Heart Valve Replacement

**Aim** To understand the causes, implications and treatment of venous thromboembolism.

After completing this section of the Anticoagulation Training Package you will;

i) Be able to determine the causes of valvular heart disease

ii) Be aware the symptoms of valvular heart disease

iii) Be aware of the tests used to diagnose valvular heart disease

iv) Be aware of how Valvular heart disease is treated.

v) Be able to educate a patient in the use of oral anticoagulation treatment following heart valve replacement

vi) Be aware of the different types of replacement heart valve used
Valvular Heart Disease and Heart Valve Replacement

In anatomy, the heart valves are valves in the heart that maintain the unidirectional flow of blood by opening and closing depending on the difference in pressure on each side. The mechanical equivalent of the heart valves would be the reed valves.

There are six types of valves within the heart.

The Atrioventricular valves
- The Mitral Valve
- The Tricuspid Valve
- The Semilunar Valves
- The Aortic Valve
- The Pulmonic Valve

Atrioventricular valves

These are large, multicusped valves that prevent backflow from the ventricles into the atria during systole. They are anchored to the wall of the ventricle by chordae tendinae, that prevent the valve from inverting.

The chordae tendinae are attached to papillary muscles that cause tension to better hold the valve. Together, the papillary muscles and the chordae tendinae are known as the subvalvular apparatus. The function of the subvalvular apparatus is to keep the valves from prolapsing into the atria when they close. The subvalvular apparatus have no effect on the opening and closure of the valves, however. This is caused entirely by the pressure gradient across the valve.

Mitral valve

Also known as the bicuspid valve, the mitral valve gets its name from the resemblance to a bishop’s mitre (a type of hat). It prevents blood flowing from the left ventricle into the left atrium. As it is on the left side of the heart, it must cope with a lot of strain and pressure, this is why it is made of only two cusps, as there is less to go wrong

A common complication of rheumatic fever is thickening and stenosis of the mitral valve

Tricuspid valve

The tricuspid valve is on the right side of the heart, between the right atrium and the right ventricle.
Semilunar valves

These are positioned on the pulmonary artery and the aorta. These valves do not have chordae tendinae, but are more similar to valves in veins.

Aortic valve

The aortic valve lies between the left ventricle and the aorta. The aortic valve has three cusps. During ventricular systole, pressure rises in the left ventricle. When the pressure in the left ventricle rises above the pressure in the aorta, the aortic valve opens, allowing blood to exit the left ventricle into the aorta. When ventricular systole ends, pressure in the left ventricle rapidly drops. When the pressure in the left ventricle decreases, the aortic pressure forces the aortic valve to close. The closure of the aortic valve contributes the A2 component of the second heart sound (S2).

The most common congenital abnormality of the heart is the bicuspid aortic valve. In this condition, instead of three cusps, the aortic valve has two cusps. This condition is often undiagnosed until the person develops calcific aortic stenosis. Aortic stenosis occurs in this condition usually in patients in their 40s or 50s, an average of over 10 years earlier than in people with normal aortic valves.

Pulmonic valve

The pulmonic valve lies between the right ventricle and the pulmonary artery and also has three cusps.

The sound of the heart valves shutting causes the heart sounds.
Valvular heart disease is any dysfunction or abnormality of one or more of the heart's four valves. Heart valves act as gates and keep blood flowing in one direction.

Diseased valves, which cannot be repaired, are replaced.

Anticoagulants help prevent the body's natural response of forming blood clots around foreign objects such as the artificial valve. They thus prevent complications associated with blood clots and also reduce risks of valve malfunction.

**Mechanical Heart Valves**

Q1. Name 2 conditions which can cause damage to heart valves.
A. 

Q2. There are two types of artificial heart valves what are they?
A. 

Q3. Which of these two heart valves requires anticoagulation therapy for life?
A. 

Q4. Give two other names for a Bioprosthetic Valve
A. 

Q5. Which type of valve tends to be associated with regurgitation?
A.

Q6. What causes valve regurgitation?
A.

Q7. Mechanical Valves are more suited to young patients. Explain why you think this is.
A.

Q8. From what animal do we obtain some Bioprosthetic Valves?
A.

Q9. Where else do we obtain Bioprosthetic Valves?
A.

Q10. What do we mean by Stenosis?
A.

Q11. What do we mean by Insufficiency?
A.

Q12. Why is the pig valve chosen for a bioprosthetic valve replacement?
A.
Q13. Identify some causes of Heart Valve Disease.

i)

ii)

iii)

iv)

v)

vi)

vii)

viii)

ix)

x)
I confirm that ....................................................... (candidate’s name) has completed the exercises within Section 2.4 ‘Heart Valve Replacement’ to the required standard.

Signature ................................................................. (Clinical Lead)

Date .................................................................
Section 2.5

Antithrombotic Therapy in Special Circumstances
SECTION 2.5  Antithrombotic Therapy in Special Circumstances

**Aim**  To understand the causes, implications and treatment of thrombotic conditions and the effects of cancer and its treatment on oral anticoagulation.

After completing this section of the Anticoagulation Training Package you will;

i)  Be aware of the different types of thrombotic conditions  

ii) Be able to determine the causes of the various thrombotic conditions  

iii) Be aware the symptoms of the various thrombotic conditions  

iv)  Be aware of how the various thrombotic diseases are treated  

v)  Be able to educate a patient in the use of oral anticoagulation treatment with regards to his thrombotic condition  

vi)  Be aware of the effects of cancer and its treatment on anticoagulation therapy
Protein C Deficiency:

"An absence or deficiency in PROTEIN C which leads to impaired regulation of blood coagulation. It is associated with an increased risk of severe or premature thrombosis. (Stedman's Med. Dict., 26th ed.)"

Protein C deficiency is a rare genetic trait that predisposes to thrombotic disease. It was first described in 1981. The disease belongs to a group of genetic disorders known as thrombophilias.

The prevalence of protein C deficiency has been estimated to about 0.2% of the general population.

Protein C deficiency is associated with an increased incidence of venous thromboembolism, whereas no association with arterial thrombotic disease has been found.

The main function of protein C is its anticoagulant property as an inhibitor of coagulation factors V and VIII. There are two main types of protein C mutations that lead to protein C deficiency:

Type I: Quantitative defects of protein C (low production or short protein half-life).

Type II: Qualitative defects, in which interaction with other molecules is abnormal.

Primary prophylaxis with aspirin, heparin or warfarin should be considered in known familial cases.

Anticoagulant prophylaxis is given to all who develop a venous clot regardless of underlying cause. Studies have demonstrated an increased risk of recurrent venous thromboembolic events in patients with protein C deficiency. Therefore, long-term anticoagulation therapy with warfarin should be considered in these patients.

Homozgyous protein C defect constitutes a potentially life-threatening disease, and warrants the use of supplemental protein C concentrates.

Protein S Deficiency

Protein S deficiency is a disorder associated with increased risk of thrombosis. Decreased levels or impaired function of protein S, a vitamin K-dependent physiological anticoagulant, leads to decreased degradation of factor Va and factor VIIIa and an increased propensity to venous thrombosis.
There are three types of hereditary protein S deficiency:

Type I - decreased protein S activity: low levels of free protein S, normal levels of bound protein S

Type IIa - decreased protein S activity: low levels of free protein S, low levels of bound protein S

Type IIb - decreased protein S activity: normal levels of free protein S, normal levels of bound protein S

Decreased activity is present in an acquired form in vitamin K deficiency or treatment with warfarin. This generally also impairs the coagulation system itself (factors II, VII, IX and X), and therefore predisposes to bleeding rather than thrombosis. Protein S levels are also lower in pregnancy and liver disease.

Protein S deficiency is the underlying cause of a small proportion of cases of disseminated intravascular coagulation (DIC), deep venous thrombosis (DVT) and pulmonary embolism (PE).

Factor V Leiden

Factor V Leiden is not a disease, it is the presence of a particular gene that is passed on from your parents.

Factor V Leiden is a variant of the protein Factor V which is needed for blood clotting. People who have a Factor V deficiency are more likely to bleed badly while people with Factor V Leiden have blood that has an increased tendency to clot.

People carrying the Factor V Leiden gene have a five times greater risk of developing a blood clot (thrombosis) than the rest of the population. However, many people with the gene will never suffer from blood clots.

In Britain, 5 per cent of the population carry one or more genes for Factor V Leiden, which is far more than the number of people who will actually suffer from thrombosis.

The genes for the Factor V are passed on from our parents. As with all inherited characteristics, we inherit one gene from our mother and one from our father. So, it is possible to inherit:

- two normal genes or one Factor V Leiden gene and one normal gene
- or two Factor V Leiden genes.

Having one Factor V Leiden gene will result in a slightly higher risk of developing a thrombosis, but having two genes makes the risk much greater.
The overall estimated incidence (annual occurrence) of deep venous thrombosis is 1 episode for every 1000 persons. This figure does not separate patients who had predisposing conditions from those who do not.

At this time, the data available do not suggest any role between factor V Leiden and arterial thrombosis (stroke, heart attack).

**Antiphospholipid Syndrome**

The antiphospholipid antibody syndrome, also known as Hughes Syndrome, is a disorder characterized by multiple different antibodies that are associated with both arterial and venous thrombosis (clots).

The antiphospholipid antibody syndrome is associated with both arterial and venous thrombosis.

A risk of recurrent thrombi, both arterial and venous, is associated with the antiphospholipid antibody syndrome as well. Most studies suggest that patients who have a recurrent episode will have it in a similar blood vessel type. In other words, patients who have a stroke initially will most often have a stroke if they have a recurrence. However, patients are reported that have multiple different types of thrombotic events.

Treatment of the initial thrombosis in patients with the antiphospholipid antibody syndrome does not generally differ from treatment of patients with the same disorder who do not have the antiphospholipid antibody syndrome. Anticoagulation with heparin and then subsequently with oral anticoagulation is initiated. The duration of anticoagulation in patients without the antiphospholipid antibody syndrome is generally 3-6 months. In patients with the antiphospholipid antibody syndrome, the risk of recurrence is relatively high for both arterial and venous thrombotic events. As a result, patients are generally started on long-term (in some cases life-long) oral anticoagulation.

The use of long-term anticoagulation has risks associated with it (approximately a 3% chance per year of having a major haemorrhage, of which approximately 20% are fatal). Beginning long-term anticoagulation is influenced by the patient's overall risk of recurrent thrombosis balanced against the risks associated with long-term anticoagulation on an individual basis. http://www-admin.med.uiuc.edu/hematology/PlAPS.htm
Anticoagulation in Cancer Patients

Patients with cancer who are receiving antithrombotic therapy are thought to be at higher risk of bleeding than patients without cancer. For practical purposes, the recommended therapeutic levels of anticoagulation remain the same as long as patients are educated about the risks and the anticoagulation levels are strictly monitored.

Patients with cancer who develop a thromboembolism should be treated in a similar manner to patients without cancer. An initial period of therapeutic unfractionated heparin or low molecular weight heparin which is overlapped and followed by warfarin for a minimum of three months is recommended. Anticoagulation should be continued in patients who have active disease or who receive chemotherapy while these risk factors last. The dose should maintain an INR of between 2.0 and 3.0.

Patients with cancer are at a higher risk than noncancer patients of recurrence of thromboembolism despite adequate anticoagulation. Again, no strict evidence based guidelines exist for the management of these patients. The recommended options include maintenance of a higher level of anticoagulation (INR 3.0 to 4.5),

SECTION 2.5

Q1. Protein C is a Vitamin .......... dependent protein synthesised in the ................. ... and is an inhibitor of the procoagulant system.

Q2. It is synthesised in the liver as an inactive form. Activated Protein C functions to inactivate Factors .......... and .................

Q3. Protein C is activated to ...................... when thrombin binds to thrombomodulin. This binding alters the conformation of thrombin to a form that readily activates Protein C.

Q4. The activity of Protein C is markedly enhanced by its cofactor Protein .......... 

Q5. Protein C causes an increased risk of ......................... primarily ............................... and ..............................

Q6. Thromboembolic events begin in .......... to .......... teenage years. Homozygous patients often die of thrombosis in early infancy. Clotting is common in heterozygotes.

Q7. In addition to the deep veins of the lower extremities, thrombosis can also occur in the ......................veins, ......................veins, .............................. ....veins, ...................... ..........veins, and the ...................... vena cava.

Q8. Clotting may be further precipitated by what other factors ................. /

Q9. Arterial thrombotic events are rare.

True  or  False
Q10. A condition also associated with congenital Protein C Deficiency is Warfarin Skin ..........................

Q11. Treatment for the condition in the previous question requires stopping warfarin and instituting ..................

Q12. When starting warfarin, very ............ doses must be used initially to prevent skin and fat necrosis.

Q13. Long-term low dose subcutaneous .............. can be used as an alternative to warfarin.

Q14. Factor ........ concentrates also contain Protein C.

Q15. Protein S is a Vitamin K-dependent factor synthesized in the liver.
   True or False (Please indicate)

Q16. Arterial thrombosis is increased in the presence of Protein S Deficiency
   True or False (Please indicate)

Q17. After starting warfarin, Proteins C & S drop to .................% within 48 hours
   10 - 20%
   20 - 40%
   - -

Q18. Patients with cancer are nearly ................ as likely to die from pulmonary embolism in hospital as those with benign disease
   Twice
   Three times
   Four Times
Q19. The INR of a patient undergoing chemotherapy should be monitored ................. days after each administration of the chemotherapy and then ................. during the entire course of her cytotoxic treatment.

Q20. Patient undergoing chemotherapy prior to commencing warfarin should be started on a ................. loading dose.

Q21. Patients undergoing chemotherapy often have a central line or Hickman line in situ. What should you be aware of when evaluating the INR of these patients.

A.

Q22. What common side effects of chemotherapy administration may have an adverse effect on the patients INR?

A.

Q23. Patients undergoing chemotherapy are often commenced on concurrent prednisolone (steroid) what effect may this medication have on the patients INR?

A.
I confirm that ......................................................... (candidate’s name)
has completed the exercises within Section 2.5 ‘Antithrombotic therapy in
special circumstances’ to the required standard.

Signature .............................................................. (Clinical lead)
Date .................................................................
Section 2.6

Potential Side Effects of Warfarin
SECTION 2.6 Potential Side Effects of Warfarin

**Aim**  To understand the causes, implications and treatment of the various side effects of oral anticoagulants.

After completing this section of the Anticoagulation Training Package you will;

i) Be able to list the side effects of warfarin

ii) Recognise the symptoms of the side effects of warfarin

iii) Be aware of how side effects are treated or minimized.

iv) Be able to educate a patient in monitoring for the potential side effects of warfarin.

v) Be aware of measures used which attempt to prevent the side effects of warfarin
SECTION 2.6  Side Effects

Like any drug, warfarin has its set of side effects and risks.

Q1. List any side effects of which you are aware.

A. i) 

ii) 

iii) 

iv) 

v) 

vi) 

vii) 

viii) 

Q2. Can you think of 2 reasons why Warfarin may be contraindicated in pregnancy?

A. i) 

ii)
Q3. What do we mean when we say that Warfarin has a Narrow Therapeutic Range?

A.

Q4. How long does it take Vitamin K to act?

A.

I confirm that .............................................................. (candidate’s name) has completed the exercises within Section 2.6 ‘Potential Side effects of Warfarin’ to the required standard.

Signature ................................................................. (Clinical lead)

Date .................................................................
Section 2.7

Concurrent Medications and their Effects on Oral Anticoagulants
SECTION 2.7 Concurrent Medication and their Effects on Oral Anticoaguants

**Aim**  To understand the implications and effects of concurrent medications used with oral anticoaguants.

After completing this section of the Anticoagulation Training Package you will;

i) Be able to recognise the possible effects of various medications when used with oral anticoaguants

ii) Be aware of the possible effects of supplements when used with oral anticoaguants

iii) Be aware of how often to test the INR of patients taking various medications concurrent with oral anticoaguants.

iv) Be aware of how Valvular heart disease is treated.

v) Be able to educate a patient in the use of oral anticoagulation treatment while taking concurrent medication.
**SECTION 2.7**

Q1. Complete the following table by determining whether the 20 drugs listed below Increase or Decrease the effect of Warfarin.

<table>
<thead>
<tr>
<th>Drugs that Increase Warfarin Effect</th>
<th>Drugs that Decrease Warfarin Effect</th>
</tr>
</thead>
<tbody>
<tr>
<td>Simvastatin</td>
<td>Cranberry Juice</td>
</tr>
<tr>
<td>Vitamin C (large doses)</td>
<td>Oral contraceptives</td>
</tr>
<tr>
<td>Trimethoprim</td>
<td>Antihistamines</td>
</tr>
<tr>
<td>NSAID’s</td>
<td>Omeprazole</td>
</tr>
<tr>
<td>Spironolactone</td>
<td>Barbiturates</td>
</tr>
<tr>
<td>Carbamazepine</td>
<td>Vitamin K</td>
</tr>
<tr>
<td></td>
<td>Amiodarone</td>
</tr>
<tr>
<td></td>
<td>Clarithromycin</td>
</tr>
<tr>
<td></td>
<td>Aspirin</td>
</tr>
<tr>
<td></td>
<td>Antacids</td>
</tr>
</tbody>
</table>
Q2. What do you think will happen to the INR of someone who has stopped smoking recently?
A.

Q3. A patient has commenced on Garlic capsules what effect do you think this may have on the INR?
A.

Q4. A patient has commenced on Glucosamine capsules what effect do you think this may have on the INR?
A.

Q5. A patient has commenced on Cod Liver Oil capsules what effect do you think this may have on the INR?
A.

Q6. It is approaching the flu season and a patient informs you he has just received his flu jab what effect would you expect this to have on his INR?
A.

Q7. A patient informs you that she has decided to go on a diet and as a result has increased her consumption of green leafy vegetables and salads how do you think this would affect her INR? Explain why.
A.

Q8. What effect do you think Aspirin will have on a patients INR?
A.

Q9. What effect will binge drinking have on a persons INR?
A.
Q10. *What response do chronic alcoholics show to warfarin?*
A.

Q11. *What effect will Erythromycin have on a patients INR?*
A.

Q12. List 3 other anti-platelet medications in addition to aspirin which patients may be taking on commencement of warfarin.
A.

Q13. Give 2 other names for the oral anticoagulant Nicoumalone.
A.

Q14. What is the trade name for the oral Vitamin K used to reduce INR’s > 10.0?
A.

Q15. How much oral Vitamin K should initially be given to a patient with an INR > 10.0?
A.

I confirm that ......................................................... (candidate’s name) has completed the exercises within Section 2.7 ‘Concurrent Medications and their effects on oral anticoagulants’ to the required standard.

Signature ................................................................. (Clinical lead)

Date ..............................................................................
Section 8

Patient Scenarios

?
SECTION 2.8

Discuss these Patient Scenarios with your clinical lead or your peers

Scenario 1

A male patient aged 75 years diagnosed with atrial fibrillation is found to have an INR of 2.5 his previous INR was taken 3 weeks ago and at that time was 2.3 he is currently taking 3mg of warfarin daily. What dose of warfarin would you prescribe and when would you ask for a retest?

Scenario 2

A female patient aged 69 years diagnosed with atrial fibrillation has an INR of 4.1 she has recently commenced a 7 day course of antibiotics what action would you take?

Scenario 3

A male patient aged 84 has been found to have an INR of 6.4 what would your actions be?

Scenario 5

A patient who is on lifelong warfarin due to recurrent DVT’s is found to have an INR of >10.0 what would your actions be?

Scenario 6

A patient notifies you that he is to undergo a cataract operation next week what level should his INR be to ensure the operation can proceed?

Scenario 7

A patient is scheduled for a cardioversion in 4 weeks time how would you proceed?

Scenario 8

A 98 year old female who is taking warfarin for atrial fibrillation is reported as having fallen a number of times over the past few weeks. You learn this when you contact the Nursing Home to relay a dose change as her INR results have been erratic for a number of weeks. What would your actions be following receipt of this information?

Scenario 9

A 49 year old male who has a mechanical heart valve in situ with the range 3 – 4 is found to have an INR greater than 10.0. There is no indication of bleeding the patient is not ill but he has commenced a new medications recently. How would you proceed?
Scenario 10

A female patient with a mechanical heart valve and a range of 3 – 4 has been taking 4mg of warfarin daily. She is found to have an INR of 4.7 how would you dose this patient?

Scenario 11

A 60 year old female patient taking warfarin for atrial fibrillation is found to have an INR of 5.1 how would you proceed?

Scenario 12

A female patient taking warfarin for atrial fibrillation who has been stable for many months is found to have an INR of 1.1 Discuss the possible reasons why this might have occurred.

Scenario 13

You are about to commence a patient with atrial fibrillation on warfarin when you notice that his baseline INR is 1.5 what would you do?

Scenario 14

What possible reasons might there be for the patient to have a baseline INR of 1.5?

Scenario 16

List the various points you would discuss with a patient when commencing them on warfarin.

I confirm that ................................................................. (candidate’s name) has completed the exercises within Section 2.8 ‘Patient Scenarios’ to the required standard.

Signature ............................................................... (Clinical lead)

Date ...............................................................
Management of patient anticoagulant therapy

References

Any General Medical Textbook

Any General Physiological Textbook

British National Formulary, Current Edition

A "GOOGLE" search will bring up vast amounts of information on atrial fibrillation its cause and treatment.

ABC of antithrombotic therapy Antithrombotic therapy in special circumstances. II—In children, thrombophilia, and miscellaneous conditions Bernd Jilma, Sridhar Kamath, Gregory Y H Lip BMJ Volume 326 11 January 2003 bmj.com

ABC of antithrombotic therapy: Antithrombotic therapy for cerebrovascular disorders Gregory Y H Lip, Sridhar Kamath and Robert G Hart BMJ 2002;325;1161-1163

ABC of antithrombotic therapy: Anticoagulation in hospitals and general practice Andrew D Blann, David A Fitzmaurice and Gregory Y H Lip BMJ 2003;326;153-156


ABC of antithrombotic therapy: Valvar heart disease and prosthetic heart valves Ira Goldsmith, Alexander G G Turpie and Gregory Y H Lip BMJ 2002;325;1228-1231


ABC of heart failure: Acute and chronic management strategies T Millane, G Jackson, C R Gibbs and G Y H Lip BMJ 2000;320;559-562


ABC of antithrombotic therapy Antithrombotic therapy in special circumstances. II—In children, thrombophilia, and miscellaneous conditions Bernd Jilma, Sridhar Kamath, Gregory Y H Lip BMJ Volume 326 11 January 2003 bmj.com


Anticoagulation in atrial fibrillation: what is certain and what is to come
Management of patient anticoagulant therapy


Antithrombotic therapy in cancer A K Kakkar and R C N Williamson BMJ 1999;318;1571-1572

Current status of stroke prevention in patients with atrial fibrillation Philip M.W. Bath, Lian Zhao and Stan Heptinstall Institute of Neuroscience, University of Nottingham, D Floor, South Block, Queen’s Medical Centre, Nottingham NG7 2UH, UK Institute for Clinical Research, University of Nottingham, Nottingham, UK European Heart Journal Supplements (2005) 7 (Supplement C), C12–C18

Diagnosis, investigation, and management of deep vein thrombosis Clive Tovey and Suzanne Wyatt BMJ 2003;326;1180-1184


Hemorrhagic Complications of Anticoagulant Treatment: The Seventh ACCP Conference on Antithrombotic and Thrombolytic Therapy Mark N. Levine, Gary Raskob, Rebecca J. Beyth, Clive Kearon and Sam Schulman Chest 2004;126;287-310

High intensity anticoagulation for cardioversion of atrial arrhythmias? The shocking truth* Brian Olshansky, MDFACC J Am Coll Cardiol, 2002; 40:934-936 http://content.onlinejacc.org/cgi/content/full/40/5/934

Lip GYH & Lowe GDO. Antithrombotic treatment for atrial fibrillation. BMJ 1996 311; 45-9

Lip GYH, Watson RDS & Singh PS. Cardioversion of atrial fibrillation BMJ 1996 311; 112-15

Lip GYH & Watson RDS Differential diagnosis of atrial fibrillation BMJ 1995 311; 1495-8

Oral anticoagulation and risk of death: a medical record linkage study Anders Odén and Martin Fahlén BMJ 2002;325;1073-1075

Recent developments in atrial fibrillation M Bilal Iqbal, Anil K Taneja, Gregory Y H Lip and Marcus Flather BMJ 2005;330;238-243


Stroke Prevention in Atrial Fibrillation Study. Final results Circulation 1991;84;527-539

Thromboprophylaxis after replacement arthroplasty Duncan P Thomas BMJ 2001;322:686-687

Warfarin Anticoagulation and Outcomes in Patients With Atrial Fibrillation A Systematic Review and Meta analysis Matthew W. Reynolds, PhD; Kyle Fahrbach, PhD; Ole Hauch, MD; Gail Wygant, RN, MS; Rhonda Estok, RN, BSN; Catherine Cella; and Luba Nalysnyk, MD, MPH CHEST / 126 / 6 / December, 2004

Warfarin use in patients with atrial fibrillation David Sulch BMJ 1997;315:750 (20 September)

**Essential Reading**


House of Commons Health Committee The Prevention of Venous Thromboembolism in Hospitalised Patients Second Report of Session 2004–05


Management of patient anticoagulant therapy