Normal haemostasis is dependent on the interactions of blood vessel wall with circulating cellular elements and proteins. The vascular system is metabolically active organ system that act as transducer for substances, synthesis, prevent coagulation, respond to stimuli. The platelets are capable or responding to stimuli, secretion of substances from its granules, it helps haemostasis by aggregation and adherence. Protein coagulation system is formed of procoagulants, anticoagulants and the coagulation pathway. When the vessel is ruptured haemostasis is achieved by vascular spasm formation of platelet plug, blood coagulation and growth of fibrous tissue into the blood clot. The coagulation "clot" occurs via extrinsic and intrinsic mechanisms and depends mainly on calcium.

Haemostatic inhibitory system contains endothelial cell inhibitors, naturally occurring inhibitors of coagulation and fibrinolytic system.

Drugs that affect haemostasis are classified into the following groups :

- Anticoagulants such as calcium sequestrating agents, heparin and oral anticoagulants.

Heparin combined with-antithrombin III (ATIII) and increases its action given by subcutaneous and intravenous

routes as treatment or prophylaxis for thrombosis, contra-indicated if known hypersensitivity. It complications are mainly bleeding and thrombocytopenia.

Low molecular weight heparin inhibits mainly factor X. Oral anticoagulants inhibit vitamin K dependent factors and monitored by PT. These drugs have many mechanisms of interaction with other drugs.

Antiplatelet drugs have irreversible inhibition of platelet functions.

Thrombolytic drugs used to dissolve thrombi as in myocardial infarction.

Drugs used in haemorrhagic states are anticoagulant antagonists such as protamine, hexadimethrine, platelet factor 4 and vitamin K.

Protamine inhibits the action of heparin, it has non coagulant effects such as haemodynamic effects which are :
Type I : Systemic hypotension due to rapid administration.
Type II : Anaphylaxis.
Type III : Pulmonary vasoconstriction.

The coagulant effects of protamine in the form of transient thrombocytopenia, however, protamine may has an anticoagulant effect in non heparinized patients.

Vitamin K reverses the effect of oral anticoagulants through synthesis of vitamin K dependent factors II VII IX X. So, useful in emergency situations.

Clotting proteins also used in the form of fresh frozen plasma, cryoprecipitate, factor VIII, factor IX and antithrombin III.

Antifibrinolytic drugs may be synthetic (e.g. aminocaproic acid and tranexamic acid "EACA") or natural (as aprotinin).

Other drugs used in haemorrhagic disorders are desmopressin, ethamsylate, and haemostatic.

To monitor the haemostatic system we have to take careful history and do careful examination.

Preoperative screening tests are, bleeding time, and platelet count which monitor platelet while PT, APTT and TT monitor the coagulation.

Intraoperative assessment of anticoagulation can be done by ACT, heparin level and TEG.

ACT normally 90-130 seconds and must reach > 400 before initiation of CPB it is simple and reproducible test.

Heparin level and protamine titration also give an idea about anticoagulation during CPB but much costly and complicated technology.

Thromboelastography (TEG) is used mainly in liver transplantation and useful also in assessment of haemostatic state pre and post operatively.

Cardiac patient must stop medications that affect haemostasis before operation :

Oral anticoagulants must stopped 2-5 days before operation, and if urgent operation, vitamin K or FFP must be administrated.

Antiplatelet drugs should be discontinued 7 days before operation, and platelet transfusion is advised in emergencies.

Heparin only stopped 4-6 hours before operation.

If recently received thrombolytic therapy, it should respond to EACA or tranexamic acid.

Before cardiopulmonary bypass (CPB) adequate level of anticoagulation must be achieved by using heparin 300 IU/Kg then ACT should be done if less than 400 seconds additional doses of heparin may be needed or dose response curve may be done.

If heparin resistance occur additional doses of heparin most commonly prolong ACT if not respond, fresh frozen plasma

(FFP) is given to increase AT III level or AT III concentrate transfusion.

HIT may occur, if suspected the options are : delay the case, plasmapheresis or give alternative to heparin such as ancrod, hirudin, platelet receptor antagonists but non are equally effective as heparin, in the future non thrombogenic surface may be used. To reverse heparin effect protamine 1 mg: 100 IU hepazin is given t must be diluted. If side effects occur or suspected alternatives are given or omit neutralization, heparinase also may be used.

CPB has many effects on haemostatic system in the form of platelet dysfunction, thrombocytopenia, (dilution or mechanical distruction).

It also affects coagulation factors by dilution and mainly by consumption, fibrin degradation products (FDPs) also generated during CPB and act as circulating inhibitors of coagulation.

Some patients bleed excessively post operatively this may be due to surgical causes, patient factors (such as medical history of abnormal bleeding systemic diseases e.g. uraemia and hepatic impairment or medications) or the insult of CPB and the hypothermia which employed during it.

To prevent bleeding, doctors must identify any problem preoperatively and deal with. Also proper physical conditions

must be employed as regard to hypothermia, hypertension, cardiotomy suction, oxygenator type, priming volume and speed of surgery. Pharmacological factors also must be in mind or prevent bleeding as regard to heparin, protamine, desmopressin and antifibrinolytic (synthetic e.g. EACA. or natural e.g. aprotinin) administration pre & intra operative.

Postoperative bleeding could be classified into five categories :

1. Surgical defect in which the patient must be re-explored and complete surgical haemostasis must be done.
2. Circulating anticoagulants which may be :
    - Residual heparin or heparin rebound that may need additional protamine.
    - Protamine effect or FDPs that need palliative treatment and maintain cardiac output and microcirculatory flow.
3. Platelet abnormalities either in number < 60.000 or in function, should treated by platelet transfusion.
4. Deficiency of circulating procoagulants may be due to consumption or dilution and treated by FFP, cryoprecipitate, or fresh donor blood.
5. Fibrinolysis which may be rarely primary that treated by EACA or secondary in which increase FDPs and it gives a worse prognosis inspite of aggressive therapy by EACA and blood transfusion.