Pathology

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The thrombus commences as a platelet aggregate. Subse quently , fibrin and red cells form a mesh until the lumen of the vein wall occludes. The coralline thrombus then progresses Hulusi Behçet , 1889–1948, Turkish dermatologist, described a disease of inflamed blood vessels in 1937. /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF as a propagated loose red fibrin clot containing many red cells (Figure 62.31). This is likely to extend up to the next large venous branch and it is possible for the clot to break o ff and embolise to the lung as a pulmonary embolus. In this situation the embolus arising from the lower leg veins becomes detached, passes through the large veins of the limb and vena cava, through the right heart and lodges in the pulmonary arteries. This may totally occlude perfusion to all or part of one or both lungs. This results in a clinical spectrum from tachycardia and pain, through respiratory failure (despite adequate ventilation) to cardiovascular collapse and death. Moderate-sized emboli can cause pyramidal-shaped infarcts on imaging.

(thrombophilia) that lead to an increased risk of venous thrombosis. Congenital De /f_i ciency of antithrombin III, protein C or protein S Antiphospholipid antibody or lupus anticoagulant Factor V Leiden gene defect or activated protein C resistance Dys /f_i brinogenaemias Acquired Antiphospholipid antibody or lupus anticoagulant

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