Coagulation, thrombosis, and embolism

Accidental trauma of any sort produces tissue damage in which many blood vessels are involved and from which haemorrhage may occur. These damaged vessels are crushed or ruptured, and in each vessel the normal haemostatic mechanism plays its role in reducing or arresting haemorrhage into or from the damaged tissues.

Vessel-wall injury promotes the adhesion of platelets to the area of injury probably by virtue of exposing collagen fibres below damaged endothelium. Within seconds, this is followed by aggregation of further platelets around these initial adherent platelets, this being a self-perpetuating process dependent on adenosine diphosphate (ADP) release from the initially injured platelets.

These platelet masses (white bodies) can form a complete plug in a small vessel or in the wound tract leading from the vessel. However, platelet aggregation is capable of reversal and, therefore, platelet plugs provide only a temporary effect. The progressive breakdown of the local ADP to adenosine and inorganic phosphate not only removes the direct aggregating factor but provides an increasing concentration of inhibitor to aggregation and this not only prevents further platelet aggregation but also promotes the breakdown of existing clumps. However, in damaged tissue while platelets clump in the injured area, factors are released into the circulation which promote fibrin formation in blood in the extravascular space and in damaged vessels where there is stagnant or turbulent flow. Such factors are tissue thromboplastin and platelet phospholipid.

The stimulus to activate these two mechanisms must be considerable and some limitation of the response must exist. The most important control exists in the fibrinolytic mechanism which limits fibrin formation and removes excess fibrin deposits. The activator of this mechanism can also be released from damaged tissue or injured vascular endothelium, and, while the lytic mechanism probably plays its main role locally, in tissue or on the vessel wall, after injury it can be shown to have a systemic effect throughout the body.

Thus, following severe trauma, changes occur in the blood which reflect platelet utilization, excess fibrin formation, and fibrinolysis. These must be a measure of the haemostatic response in any individual and the response has been found to vary in patients with similar degrees of injury.

If severe trauma takes place, the stimulus to induce the above changes may be so violent that the process may spread beyond the area of damaged tissue and induce platelet and fibrin deposits either in the vessels draining the damaged area or elsewhere. Thus, major vessel thrombosis may develop or platelet-fibrin microthrombi may be widely disseminated and involve tissues distant from the area of trauma, eg, lungs or kidney. If this occurs then the blockage of the circulation may alter the haemodynamic situation and hinder the restoration of normal blood volume by fluid replacement. If the process of intravascular coagulation is severe, consumption of platelets, fibrinogen, and other coagulation factors, together with the effect of active fibrinolysis can result in more severe primary or secondary haemorrhage into and from the damaged tissue. Further, complications such as haemolysis of blood in damaged tissue, or as a result of blood transfusion, infection, tissue anoxia, alteration in blood pH, can all accentuate this process. The problem facing the surgeon treating a patient with severe trauma and the haematologist trying to unravel the complex changes in the blood is to define what is happening in any individual patient at any one time. Does the clinical picture and the
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Changes in the blood simply reflect the haemostatic process in response to trauma, blood loss, or blood transfusion, or has the trauma and involvement of the blood systems been so great that local or widespread intravascular thrombosis has occurred to impede the circulation or to damage vital tissues?

From the available evidence, tests of blood coagulation are unlikely to unravel the problem. Initially, decisions on therapy must depend on clinical assessment. If, however, bleeding is excessive and laboratory tests show severe depletion of platelet numbers, fibrinogen, and the presence of large amounts of split products due to fibrinolysis in the circulation, then it can be deduced that an unusually severe reaction has occurred and that immediate complications are likely to arise.

Therapy lies, as Hardaway suggests, in the restoration of blood volume and the use of vasodilators to enhance flow, tissue perfusion, and oxygenation. If these fail to bring improvement and the clinical situation suggests that intravascular coagulation is hindering the restoration of the blood volume, then the possibility of anticoagulant therapy arises. From experimental studies it is evident that heparin or other prophylactic anticoagulants have little therapeutic effect unless given before injury and that the only effective therapy for established cases may be the use of fibrinolysis induced by streptokinase or urokinase. The wisdom of using such therapy in a patient suffering from multiple injuries, is of course, debatable.

A less dramatic but nevertheless serious complication is the high incidence of deep-vein thrombosis of the lower limb in the period after injury. Though not peculiar to this clinical state, some who survive their initial injuries and shock die later from pulmonary embolism. Prophylactic anticoagulant therapy using the long-term oral anticoagulants can reduce the incidence of this complication.

These problems are all discussed in detail in the papers that follow.

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