The fibrinolytic effects of intermittent pneumatic compression. 
Mechanism of enhanced fibrinolysis


Abstract

• A study using 6 healthy subjects and 6 post thrombotic subjects was undertaken to quantify;
  – Changes in fibrinolytic activity with 5 different Intermittent Pneumatic Compression (IPC) devices, including the FLOWTRON® System.
  – The mechanisms of action with respect to fibrinolysis and IPC.
  – Whether post-thrombotic patients have the same capacity for fibrinolytic enhancement as do normal subjects.
• The results indicated that IPC stimulates fibrinolytic activity in normal subjects and post-thrombotic patients, but the effect is reduced in post-thrombotic patients.
• Fibrinolytic activity is more related to the PAI-1 (plasminogen activator inhibitor) activity rather than amounts of tPA (tissue plasminogen activator) in the circulation.

Main Outcomes

1. Fibrinolysis is the result of activation of plasminogen to plasmin by one of 2 endogenous plasminogen activators – tPA and uPA (urokinase type plasminogen activator). This activation sequence is balanced by the presence of inhibitors. Of key importance is the relationship between tPA (activator) and PAI-1 (plasminogen activator inhibitor).
2. In the post thrombotic patients, baseline fibrinolytic activity was significantly less than that in the 6 healthy subjects and it was not until these subjects were stimulated by IPC that their fibrinolytic activity levels reached the levels seen in the normal subjects at baseline.
3. After 180 minutes of IPC, there was a significant increase in fibrinolytic activity in both groups. tPA levels only increased in normal subjects.
4. Decreases in PAI-1 were seen in both groups after IPC.
5. It is the balance of PAI-1 to tPA which determines fibrinolytic activity. IPC induces a reduction in PAI-1 which in turn increases the availability of tPA.