be detected in healthy and anticoagulated blood. We compared the gel time (GT) required to form the incipient clot and the corresponding fractal dimension \(d_f\) against laboratory markers of haemostasis and thromboelastography (TEG).

**Methods** Blood samples were taken from 52 healthy adults and similarly 34 individuals whose blood had been anticoagulated with heparin over the therapeutic range.

**Results** The incipient clot in normal blood is established as a sample-spanning network cluster at the gel point [2]. The value of \(d_f\) in whole healthy human blood is 1.74 (±0.07), which indicates a high degree of branching in the fibrin network at criticality and is commensurate with that reported to arise in other biological systems. There was a significant reduction in the value of \(d_f\) and a corresponding prolongation in the GT in the heparin group as compared with the healthy group (Figure 1).

**Conclusions** We describe for the first time that the incipient clot formed at the gel point of whole blood is characterised by a fractal microstructure. The values of \(d_f\) and GT discriminate between clot structure in healthy and anticoagulated blood. The relationship between these new markers may provide a basis for exploring the relationship between coagulation pathways and clot quality.

**References**

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Acquired bleeding model induced by dilutional coagulopathy in the rabbit

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**Methods** Severe traumatic or intraoperative blood loss necessitates massive transfusion. This blood loss and the dilution of coagulation factors result in insufficient haemostasis. Treatments to enhance the haemostatic capacity need to be evaluated. An acquired bleeding model due to a dilutional coagulopathy was developed in the rabbit to estimate the contribution of PCC (Beriplex P/N; CSL Behring) on haemorrhage.

**Results** Rabbits were anesthetised using isoflurane and were mandatorily ventilated. The animals were instrumented to monitor the cardiovascular and respiratory system. Dilutional coagulopathy was induced by phase-d blood withdraw, salvaged erythrocyte retransfusion and volume substitution with hydroxyethyl starch.

After the dilutional procedure the bleeding was inflicted by cutting the lateral kidney pole. To characterise the model, animals were allocated to groups: I, sham operation (no dilution, placebo treatment; \(n = 5\)); II, negative control (dilution, placebo treatment; \(n = 5\)); III, PCC 25 U/kg intravenously (dilution, verum treatment; \(n = 6\)). Coagulation factor activity, thrombin generation (TGA), prothrombin time (PT) and the bleeding from the kidney wound were assessed.

**Conclusions** The use of aerosolized unfractionated heparin and N-acetylcysteine attenuates lung injury and the progression of acute lung injury in ventilated adult patients with SIJ.

**References**