FIBRINOLYSIS IN RELATION TO LIPAEMIA AND INTRAVENOUS HEPARIN IN THE WHITE AND BANTU*

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Fibrinolytic activity was studied in 22 White and 20 Bantu subjects using the 1:10 blood clot lysis time (BLT) of Fearnley et al. as modified by Lackner and Goosen, as well as the euglobulin lysis time (ELT) described by Von Kaulla and Schultz. Subjects were tested fasting and 3 and 4 hours after breakfast.

Variations in fibrinolysis which occur 3 and 4 hours after the ingestion of 70 G. of butter fat were found to be identical with those occurring after a fat-free breakfast. It was therefore concluded that lipaemia did not appreciably affect fibrinolysis.

Spontaneous acceleration of fibrinolysis over the 4-hour period of observation was found to be statistically significant with respect to BLT in the Whites (difference from fasting sample being 5-1 hours), but not in the Bantu (difference from fasting sample 1 hour). With respect to ELT, statiscally significant acceleration occurred in both Whites and Bantu, but was found to be twice as great in the Whites, i.e. 0-8 hours as opposed to 0-4 hours. As a result of the diurnal variation manifested by the Whites, their mean lysis time tended to approach that of the Bantu during the course of the morning. However, this diurnal variation was unable to abolish the statistically significant difference in fibrinolytic activity between Whites and Bantu, which has previously been reported by us.4

Since it is well known that heparin has a marked fatclearing effect on lipaemic plasma, it was decided to test the effect on fibrinolysis of heparin given at the height of lipaemia. Accordingly 9 White and 9 Bantu subjects were given an injection of 75 mg. of heparin (10,000 units) in a volume of 2 ml. 3 hours after the ingestion of 70 G. of butter fat. Blood was taken immediately before and 1 hour after the injection, and the fibrinolytic activity of the 2 samples was compared. As a control the experiment was repeated on another day under identical conditions, but instead of heparin, 2 ml. of saline were given. Again the fibrinolytic activity of the blood sample taken immediately before and 1 hour after the injection was compared.

It was found that heparin significantly accelerated fibrinolysis as measured by BLT in Whites (acceleration 3.6 hours, p = 1%) and Bantu (acceleration 2.2 hours, p = 2%). As measured by ELT, the acceleration in the White subjects (0.5 hours) and in the Bantu (0.3 hours) was statistically significant at the 5% and 2% levels respectively. The effect of saline was variable and not statistically significant. On comparing the changes in fibrinolysis occurring on the heparin day with those occurring on the saline day, it was found that they were statistically significant only with respect to BLT. They failed to reach statistical significance with respect to ELT partly because of paucity of numbers and partly because the heparin effect appears to be more marked with BLT than with ELT.

The fibrinolysis-accelerating effect of intravenous heparin, previously shown by us to be present in fat-free plasma, has now been demonstrated in lipaemic plasma. The exact mechanism is obscure, but we do not feel that it is due to the actual removal of chylomicrons from the plasma.

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