Correspondence

Accepted: August 30, 1984

Acta haemat. 1984;72:358

Reply

D.	Draga	Creter
----	-------	--------

Petah-Tiqva

Draga Creter, MSc, Department of Hemostasis, Hasharon Hospital, Petah-Tiqva 49382 (Israel)

We are grateful for the comments and think that these remarks will be of interest to us and to others.

As Prof. Girolami remarked, it is a short paper on factor VII deficiency, and therefore not all the determinations done on this family were included. We performed a large battery of tests including a coagulation screening with the quantitation of all the intrinsic and extrinsic plasma factors, a fibrinolytic screening with the determination of fibrinogen split products, plasminogen, SK complex, plasmin, thereafter the bleeding time, platelet count, and so on. All these tests included also the inhibitors of coagulation and fibrinolysis, especially the antithrombin which was done by two methods, a functional one using chromogenic substrate and an immunological one using RID plates.

In the paper, on page 61, we wrote that all the coagulation and fibrinolysis tests were within the normal range. The protein C was not included because the family was studied in 1981 when the commercially available kit was not on the market. We think that it would be interesting for us to study all the members of the family by this test as well.

In our study, 14 members of the family were studied, but table I represents only the results of those

members with pathological changes, as it is mentioned in the title of this table. The husband was included in the study, the results of his tests were found to be within the normal range, and he was not drawn on the family tree. The factor VII assay was done with human thromboplastin (Behringwerke).

In answer to the remark that it can be factor VII Padua defect, we thought so too, but we have neither experience with this variant, nor a control plasma from a family with factor VII Padua defect, so we concluded that what is important is to establish an accurate diagnosis of the deficiency, and especially the fact that a link was observed between this defect and the increased thrombotic tendency. The 'hyperactive' factor VII described by Prof. Girolami can be a possible explanation.