Reprinted from ANGIOLOGY
Vol. 17, No. 9 *September, 1966*

THROMBOLYSIS IN MYOCARDIAL INFARCTION RESPONSE OF SERUM ENZYMES

DR. F. PRAETORIUS AND DOZ. DR. P. KÖRTGE

The revised 36 patients of our clinic (figs. 1, 2) are a part of the total collection presented by Dr. Poliwoda (8). It was possible to construct a mathematical curve of the average Serum Glutamat Oxalat Transaminase (SCOT) and Serum Creatine Phosphokinase (CPK) measurements at different times. A total of 349 measurements (SCOT: 208, CPK: 141) were taken in intervals of 3-24 hours. The two slopes of the curve following the point of maximum enzyme concentration are determined by biological Half Life Times. The Half Life Times depend initially on the extracellular and vascular distribution of the enzyme, later on metabolic processes, as shown by other investigators repeatedly (1-5,7). Therefore one cannot expect to change these slopes through our therapy.

The points in fig. 1B and 2B represent the average SCOT and CPK measurements of two groups. One group consists of patients who, in our estimation, received treatment at a sufficiently early time (1-3 hours after the onset of the infarction). This is compared with another group of patients in whom fibrinolytic therapy was started 3 hours or later after the begin of the infarction. With the exception of the early phase of both curves, the average enzyme measurements do not show any significant differences between the two treatment groups. During the early phase, there seems to be a definite difference with higher levels in the early treated group and the point of maximum occurring later in the later treated patients. But the difference was significant ($p < 0.01$, $p < 0.02$) only at a point 4 hours (CPK) respectively 6 hours (SCOT) after the onset of infarction. The high point at the end of the CPK curve may be due to a laboratory error, or possibly due to a re-infarction.

We believe that our findings correlate quite well with the results of Dr. Poliwoda's EKG investigations (6, 8). It should be noticed that the earlier increase of serum enzymes may demonstrate a measurable action of the fibrinolytic therapy. On the ground of the presented data we do not feel justified to draw more generalizing conclusions.

Dr. F. Praetorius
1st Medical Clinic
Free University of Berlin
Berlin, Germany

REFERENCES

1. Amelung, D.: *Fermentdiagnostik Interner Erkrankungen,* Georg Thieme Verlag, Stuttgart, pp. 11-29, 122 - 137, 1964.

2. Brüdigam, B., and Schneider, K. W.: Klinische Untersuchungen zur Pathogenese der

From the 1st Medical Clinic of the Free University of Berlin. (Head of the Clinic: Prof. Dr. H. Frhr. v. Kress).

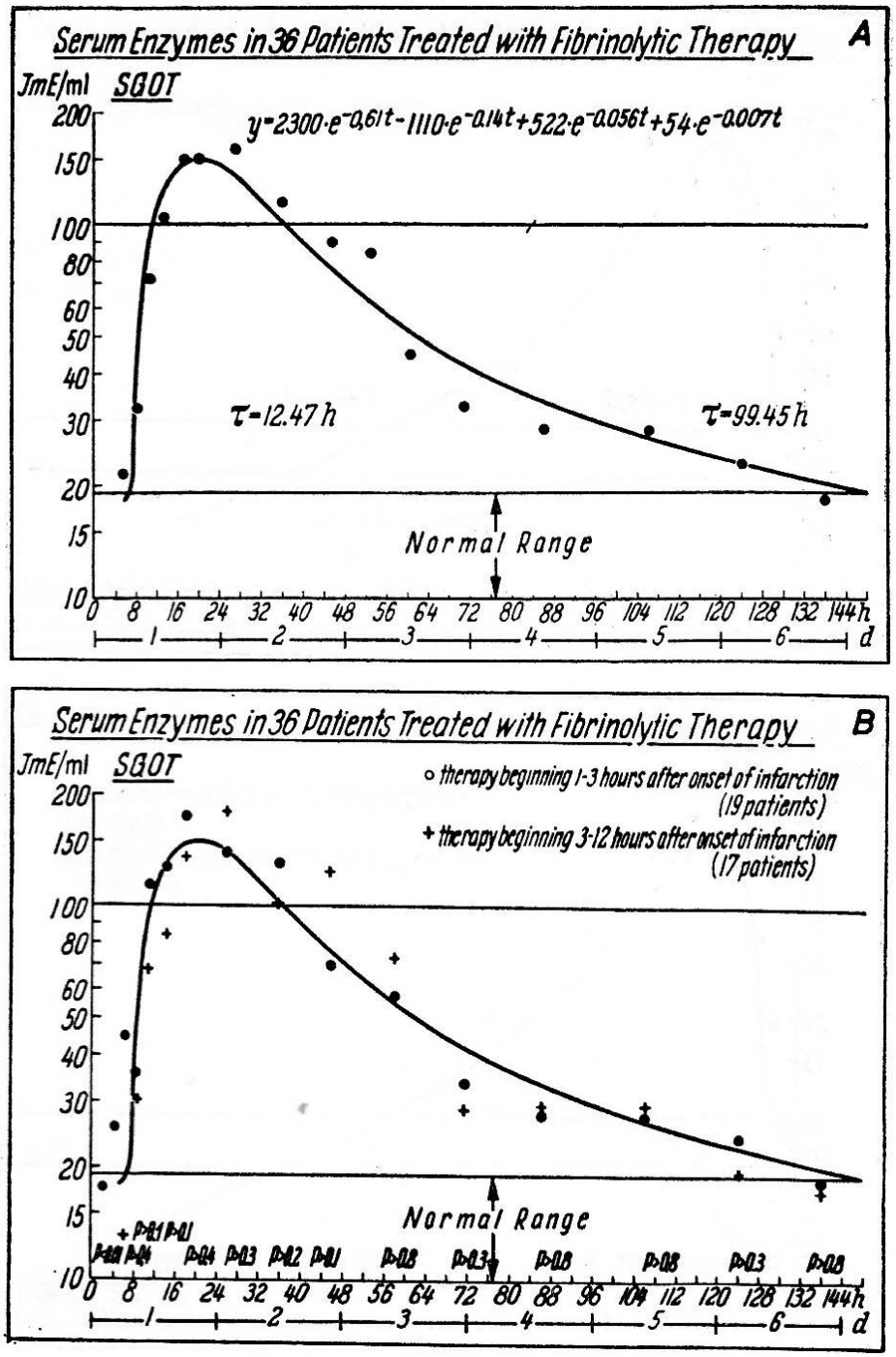

Fig. 1. A. Mathematically constructed curve of the total collection (average SGOT measurements). B. The two groups (early and late treated patients, see text), contrasted to the curve of fig. 1 A.

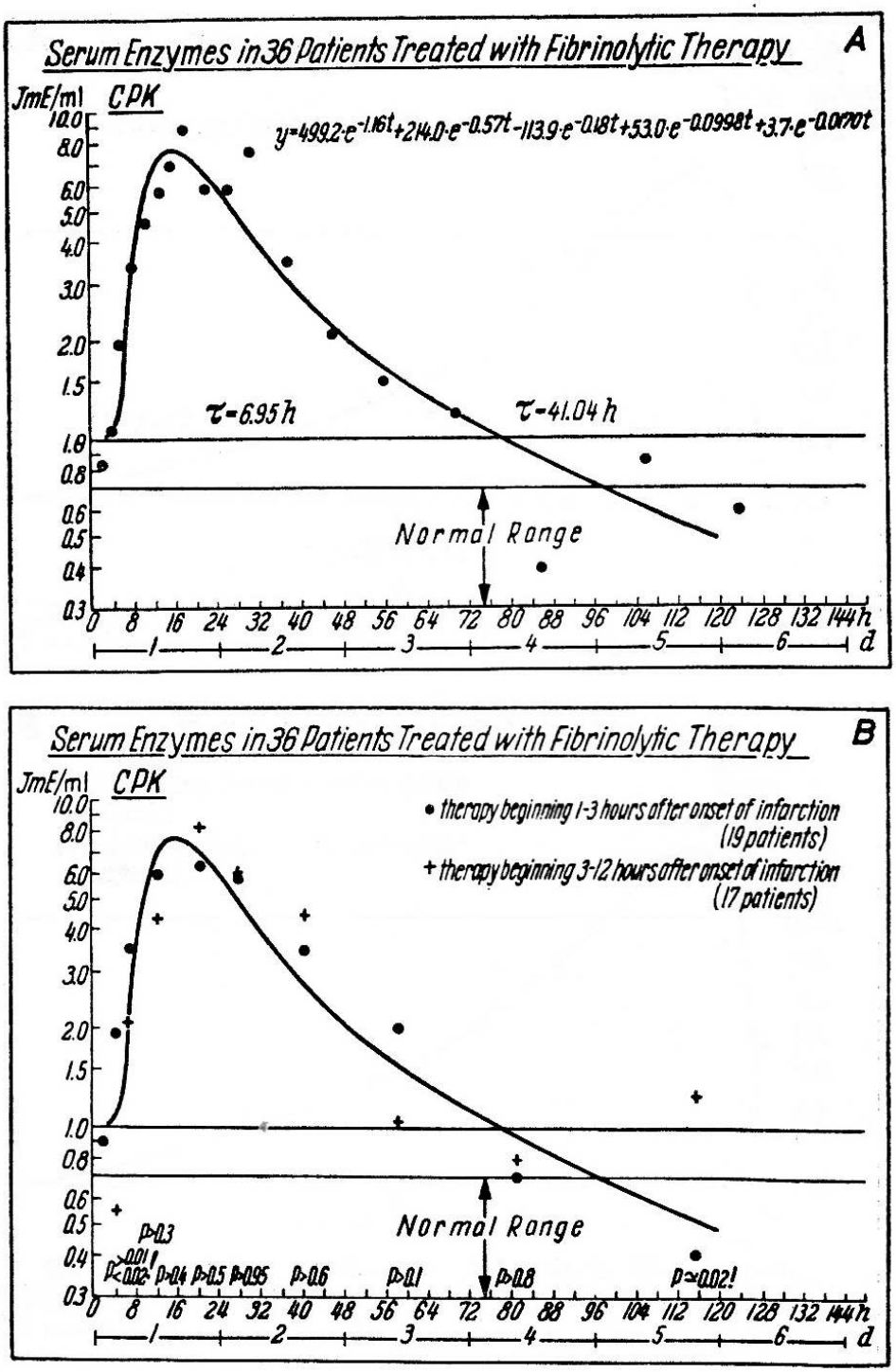

Fig. 2. A. Average CPK measurements, proceeding as in fig. 1 A.
B. The two groups, curve of fig. 2 A.

Fermentaktivitätserhöhungen der Glutaminsäure-Oxalessigsäure-Transaminase und Aldolase im Serum. Deutsches Arch. klin. Med., 206: 438, 1960.

3. Duma, R. J., and Siegel, A. L.: Serum creatine phosphokinase in acute myocardial infarction. Arch. Int. Med., 115: 443, 1965.
4. Forster, G., and Escher, J.: Die Kreatinphosphokinase in der Diagnostik von Herzinfarkt und Myopathien. Helvet. med. acta, 28: 513, 1961.
5. Hess, B.: *Enzyme im Blutplasma, p.* 48, Georg Thieme Verlag, Stuttgart, 1962.
6. Körtge, P., and Praetorius, F.: The value of streptokinase-induced fibrinolysis in the treatment of acute myocardial infarction. Sangre, 9: 235, 1964.
7. Neuhaus, G., Humpert, U., and Weller, H.: Das Verhalten der SGOT-Aktivität nach Leberpunktionen bei Patienten mit Schlafmittelvergiftungen. Deutsches Arch. klin. Med., 210: 87, 1965.

S. Poliwoda, H.: The thrombolytic therapy of acute myocardial infarction. Angiology, 17: 528, 1966.

9. Vincent, W. R., and Rapaport, E.: Serum creatine phosphokinase in the diagnosis of acute myocardial infarction. Am. J. Cardiol., 15: 17, 1965.