COMPUTERIZED MULTI-CHANNEL THROMBOELASTOGRAPH
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Widely used Thrombelastographs (TEG) made by
Helipage Co. measure the coagulation and fibrinolysis of blood by an optical method. The light
rays scan on the film according to the viscoelasticity of the blood. The result cannot be known
until the film is developed at the end of the experiment. It is troublesome to develop and
read the film. The purpose of the present work is to overcome this inconvenience. A silicon
photo diodes array is used as a photo sensor and placed in the position where the light hits.
The array includes 128 diodes, each of which has a corresponding memory (128 bit shift register).
These memories read in what parts of these diodes are exposed to the light when the rotary
socket in the main instrument change the direction. These memories are read out by synchronizing
clock pulse and counted in binary code. The signal in binary code are converted into analogous
signals which correspond to the envelopes of TEG patterns. A microcomputer is added in order to
calculate the maximum amplitudes of the envelopes. These amplitudes are assembled in the same
space as the recording cassette. Therefore, by replacing the recording cassette by the newly
made recording system and connecting to a dot printing recorder, we can get directly printed
envelopes of TEG patterns. We can also get the digital values of reaction times, coagulation
times and maximum amplitudes by connecting the recording system to a digital printer.
The newly developed recording system could overcome the inconvenience of TEG currently used
and save time. (This work was supported in part by a grant (140911) from the Ministry of
Education, Japan)

THROMBOLYTIC THERAPY WITH VIRGINIA PLASMIN: BIOCHEMICAL AND CLINICAL RESULTS.
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Preliminary results are presented on the systemic action of porcine plasmin which was used
as a thrombolytic agent. Patients suffering from acute arterial thrombosis (2) or deep
veinous thrombosis (6) received three plasmin (pp) in the following dose schedule: 2000 U
given as an initial bolus dose at 1 hour, the first 60 minutes, and 2000 U given as a maintenance dose
by continuous infusion within the following 8 hours. The maintenance dose was repeated at each of the
2, 3, or 4 consecutive days.

Investigations of the plasma showed a rapid drop of fibrinogen to 50% of the initial
value. FDP raised up to 300 μg/mL. There was no change of the mean plasminogen concentration.
Systemic fibrinolytic activity was very low and could only be demonstrated in traces during all
stages of the therapy. Analyses of the inhibitors showed a continuous drop of the alpha2-
macroglobulin to levels of 30-100 mg/L. Plasmin-antiplasmin complexes were detected in
considerable amounts.

The treatment was well tolerated by all patients. In one patient, a complete recanalization
of a femoral bypass of an iliac artery could be achieved. In the other patients, only partial
recanalizations could be demonstrated. A combination of porcine plasmin with streptokinase
therapy is possible.

FIBRINOLYTIC SYSTEM AND FIBRINOLYTIC INHIBITORS IN BECHET’S DISEASE. P. Asbeck,
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An increase of the inhibitory potential seems to be the generally accepted
cause for the suppression of the fibrinolytic system in Behcet’s disease. To
prove this theory, we investigated 5 male patients (31-46 yrs.) suffering from
severe Behcet’s syndrome. Additionally, three of them were treated with Stanozo-
loli (50 mg/d) and Phenformin (50 mg/d) during a period of one year. The follow-
ing parameters were investigated in detail: fibrinogen, fibrin (different fi-
brin plate assays), plasminogen, fast reacting antiplasmin, plasmin-antiplasmin
complex, alpha2-macroglobulin, C3-esterase inhibitor, alpha-antitrypsin, anti-
thrombin III (Different immunological methods).

In all patients, the concentration of the fibrinogen and alpha2-antitrypsin were
elevated. There were normal concentrations of plasminogen, fast reacting anti-
plasmin, plasmin-antiplasmin complex, alpha2-macroglobulin, C3-esterase inhibi-
tor, and antithrombin III. Using the venous occlusion technique, a marked re-
duction of the in-vivo activation of the fibrinolytic system could be demonstrat-
ed. During therapy with Stanozolol and Phenformin, a high fibrinolytic response
was induced by venous occlusion. The analysis of the different inhibitors, however,
could not explain this phenomenon. An increased production or release of
vessel activator(s), therefore, seems to be the mechanism of fibrinolysis in-
duction by anabolic steroids and Phenformin.