GERMAN-AUSTRIAN MULTICENTER TWO YEARS PROSPECTIVE STUDY ON THE PREVENTION OF SECONDARY MYOCARDIAL INFARCTION BY ASA IN COMPARISON TO PHENPROCOUMON AND PLACEBO.

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A multicenter clinical trial was conducted between Jan.1971 and March 1977 in seven hospitals in Germany and Austria on 945 patients who had survived a myocardial infarction for six weeks. The patients were randomly allocated within strata (hospitals,age,sex,secondary infarction,decompensation,hyperlipemia) to ASA (1.5g/day) placebo or phenprocoumon (ph) treatment. The trial was double blind regarding placebo and ASA and open regarding ph. The data of patients dying within the two years' observation period suffering secondary m.i. were reviewed by an expert panel of three cardiologists who did not participate in the study. There is no statistical relation between treatment and strata. 531 patients (56.1%) were followed throughout the study without complications, 12,2% had to be excluded for technical reasons, 10.5% did not continue the observation, 1.2% had intercurrent diseases. 6.3% dropped out because of side effects, 9.8% suffered a secondary infarction or died with sudden death, 3.9% died by other causes. 13 patients in the ASA group (317), 22 in the placebo (309) and 26 in the ph group (320) died after m.i. and by sudden death. 11 patients in the ASA group, 15 under placebo and 6 under ph survived a secondary m.i. 30 patients taking ASA dropped out of the study because of side effects, 17 in the placebo group and 13 in the ph group. The preliminary analysis of the data leads to the following conclusions: 1.under ph treatment more patients die than under ASA by m.i. plus sudden death. 2.Secondary m.i., death by m.i. and sudden death occured less frequent in the ASA group (24) compared with placebo (37) and ph (32) but side effects were more frequent in the ASA group.

EDWARD KOWALSKI MEMORIAL LECTURE

On the pathogenesis of "disseminated intravascular coagulation." Theodore H. Spaet, M.D.
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The following remarks introduce an alternative hypothesis to those conventionally held concerning the hemostatic changes of "disseminated intravascular coagulation (DIC)."

The concept that coagulant materials may enter the blood with sufficient rapidity to produce clotting within the vessels and reduction of various blood coagulation factors, particularly fibrinogen, has long been with us. In early animal experiments intravenous tissue factor or thrombin was administered; when these were given at sublethal rates, typical coagulopathies were observed. The next step was to identify experimental situations or clinical conditions in which blood changes resembled those produced in coagulant-injected animals. These were readily found, and many tests were developed to achieve increased sensitivity in diagnosis of the process. In general, the most widely used have been those which demonstrate loss of certain clotting factors, and those which identify fibrinogen or fibrin fragments. "DIC" is now a generally accepted syndrome.

Some neglected observations suggest an alternative interpretation of the blood changes in "DIC." These raise the possibility that in some, if not many or even most of these syndromes, the source of the blood changes is extravascular processing of the clotting factors. Products may return to the circulation via lymphatics. It is proposed that present methodology cannot resolve this alternative.

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