

CHAIRMAN'S INTRODUCTORY ADDRESS TO THE WORKSHOP OF TRENTAL

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Cardiovascular diseases possibly caused by and certainly followed by abnormal platelet aggregation are the major killers in developed countries (Ambrus, J.L., et al, 1975). It was hoped that suitable platelet aggregation inhibitors will be developed to prevent these problems including the vicious cycle of more platelet aggregation on new blood clots, followed by more platelet aggregation, more clots, etc. Some aggregation inhibitors appear to inhibit both the metabolic pathways leading to thromboxane A₂ formation and the pathway leading to cyclic AMP synthesis. Thus *in vivo* they may do as much harm as good. Pentoxifylline appears to act selectively inhibiting platelet aggregation (Gastpar, H., 1978). This drug has been used extensively as a vasoactive agent in cerebrovascular and peripheral vascular disease (Aschkar, D., et al 1972; Nauman, J.L., et al 1971; Hammer, O. Neuner, A., 1972; Mansfeld, H.G., 1972; Schafe, M.K., 1973). Platelet aggregation inhibition, reduction of blood viscosity and increased deformability of red cells may be important parts of the mechanism of action (Jauch, M., 1973; Gastpar H., et al 1978).

In our experience, thromboembolic and/or hemorrhagic complications are major causes of death in cancer patients (Ambrus, J.L. et al 1975). Platelet aggregation inhibitors appear to decrease this problem. Pentoxifylline was also found to inhibit metastatic distribution of cancer cells by inhibiting platelet aggregation on circulating cancer cells. This may open new horizons for the clinical use of these agents. For example, Gastpar et al (1978; 1977; 1974; 1972) found that platelet aggregation inhibitors given in connection with cancer surgery decrease recurrences due to tumour cell mobilization during surgery under experimental and clinical conditions.

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