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Title : THE EFFECT OF APROTININ-ACD-BLOOD ON PULMONARY FUNCTION

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Introduction. The inhibition of aggregation in banked blood by Aprotinin results an improvement of blood quality. Apart from interrupting the formation of microaggregates, Aprotinin also inhibits the release of vasoactive mediators from the platelets. A prospective randomised double blind study was conducted to investigate the benificial effects of Aprotinin-acd-blood in patients after massive transfusion.

Methods. 48 patients aged between 20-83 years by whome surgical or polytraumatic bleeding disorders were manifested, were divided in two equal groups. The patients of both groups were comparable with respect to age, shock period, transfusion volume and age of stored blood. Standard filters of pore size 170 µ were used for transfusion. Generally, PEEP-ventilation with 10 cm WC and 30% O_2 was conducted during the immediate postoperative phase. A transfusion volume of not less than 10 units in 18 hours was a primary criterion for the admission in the study. Patients with pulmonary contusions, cranial and cerebral traumata were excluded. The banked blood was obtained from the German Red Cross Transfusion Service in Lütjensee/Hamburg, W-Germany. ACD-blood of groups 0 Rh pos. and A Rh pos. was specially prepared with or without addition of aprotinin. In order to differentiate between the batches with or without aprotinin the former were labelled "Hamburg" and the latter "Kiel". This labeling was unknown to the doctors and the patients. All patients gave written informed consent. The approvals for the study were obtained from the Federal Public Health Office, Berlin. Pulmonary function was investigated by assays of arterial oxygen partial pressure, pulmonary vascular resistance, shunt volume and dead space ventilation. Renal function was monitored by means of the creatinin clearance test. Haemostasis was checked by estimations of the PTT and the thrombin coagulase time. The different parameters were statistically tested by means of two-way analysis of variance.

Results. The patients of the two groups showed significant differences in pulmonary function after transfusion of, an average, 15 units. By similar cardiac outputs in both groups, the pulmonary vascular resistance was significantly decreased and the arterial oxy gen partial pressure was significantly increased in the patients of the Aprotinin group. In the control group the physiological dead space was raised to more than 200 ml and the arteriovenous shunt volume was increased to 20%. In contrast, both parameters remained in normal range in the Aprotinin group. A similar favourable perfusion effect was observed by investigating the renal function. The creatinin clearance was significantly higher in the Aprotinin group than in the controls. Whereas the activity of the plasmatic clotting system was not affected, the fibrinolytic system was slightly stimulated after massive transfusions. This was confirmed by estimations of the PTT and thrombin coagulase time as sensitive parameters for the clotting and fibrinolytic systems respectively.

Discussion. Massive blood transfusions do, in fact, lead to an increase in the pulmonary vascular resistance. As to be expected, the arteriovenous shunt volume is increased by almost 100%, the dead space ventilation is pathologically increased and the arterial oxygen partial pressure is decreased to a critical level in the patients of the controlgroup. In contrast, an uncomplicated perfusion was seen in patients receiving Aprotinin blood. None of the parameters of pulmonary function in this group was significantly impaired. Furthermore, the number and function of the platelets were significantly better in the Aprotinin group than in the control. Thus Aprotinin-ACD-blood completely satisfies the important clinical requirement of minimising the pulmonary and haemostatic complications caused by massive transfusions.

References.

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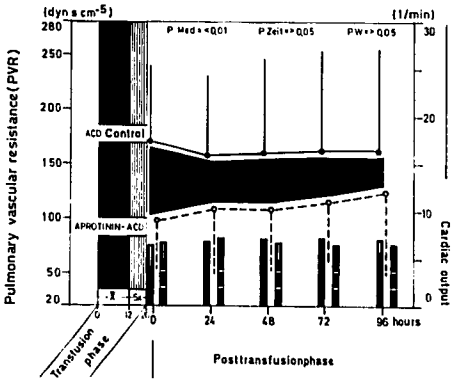

PVR after massive transfusions of ACD-Blood and Aprotinin-ACD-Blood.