Hemostasis in the oral cavity. D. König, Munich, West Germany.

Various factors and reasons can be responsible for acute, severe, or protracted hemorrhages in the oral cavity. Hemostasis with conventional methods frequently poses a difficult, dangerous, and involved problem. A special method is developed utilizing the chemical and physical properties of inorganic and organic compounds, and this is found to be efficient for rapid and safe hemostasis. It acts on the blood coagulation process by lowering the pH value of blood, as well as by its astringent and coagulant properties. Altogether, the result is hemostasis and favorable wound healing. With the use of bone cement, the initial clot is physically protected, thus contributing more to reliable hemostasis, particularly after dental extraction. The method used, that is, bone cement in combination with the developed hemostatic agent, can be used without additional treatment for severe primary, as well as secondary hemorrhages. Patients suffering from hemophilia and other disorders of blood coagulation were treated successfully.


Furosemide fibrinolytic activity, as measured by the fibrin-plate method, is enhanced after an intravenous injection of 40 mg furosemide. The effect is evident within 30 minutes of the injection and attains a peak after 6 hours. If a second injection of furosemide is given at this stage, the increased fibrinolytic activity persists, and a slightly higher peak than the first is obtained again 6 hours after this injection. After the furosemide injection, there is an initial decrease of urokinase excretion which returns to normal after 3 to 6 hours.

The decrease of urokinase excretion is attributed to a dilution effect during increased diuresis and the rise in fibrinolytic activity by furosemide is attributed to its vasodilatory activity and cyclic-AMP phosphodiesterase inhibitory capacity. It also appears related to the Hageman factor dependent pathway of fibrinolysis. In the treatment of high altitude pulmonary edema, furosemide restores factor XII and fibrinolytic activity, which are both depressed in this disease (I. Singh and J.S. Chohan, Int. J. Biometeor. 18, 33, 1974).