Introduction

Trauma mortality has been described to have a trimodal or bimodal distribution **(Riou et al., 2001, Como et al., 2004).** Of trauma deaths 50% occur at the scene of injury because of massive head injury. There is then a second peak, which represents 30% of the deaths that occur early, half of which are due to uncontrollable hemorrhage **(Shackford et al., 1993, Sauaia et al., 1995).** A third phase of later deaths, related to multiple organ failure (MOF) associated with prolonged shock, massive transfusion, and infection, is also seen, although improvements in trauma care have seen figures fall. Life-threatening traumatic hemorrhage that occurs is often due to surgical and coagulopathic bleeding **(Velmahos et al., 2000, Kushimoto et al., 2003).**

The coagulopathy of trauma has remained problematic and its etiology is multifactorial, involving hypothermia, acidosis, consumption of clotting factors, and dilution **(Lawson et al., 2004; Martinowitz et al., 2005).** If a patient develops the lethal triad of hypothermia, acidosis, and coagulopathy, then surgical control is less likely to be effective alone **(Ferrara et al., 1990).**

Coagulopathy in trauma may also be due to traumatic brain injury, fat embolus syndrome. Attempts to minimize transfusion of blood and blood products have led clinicians to look at alternate means of restoring hemostasis **(Malone et al., 2003).**

Recombinant activated factor VII has been used to control life-threatening traumatic bleeding that has been uncorrected by other means. rFVIIa acts to amplify coagulation at the local site of injury where tissue factor and phospholipids are exposed, accelerating the tissue factor-dependent pathway and generating a thrombin burst along with platelet surface interactions **(Martinowitz et al., 2001; Levy, 2003)**.

In cases of injury, tissue factor (TF) is brought into contact with naturally occurring FVIIa, which is normally present in minute quantities, to initiate the coagulation pathway (Hoffman, 2003). At pharmacological, supraphysiological doses, rFVIIa are able to bind to activated platelets at the site of injury and activate factors IX and X directly, leading to a thrombin burst. As platelets are activated only at sites of TF exposure, it is believed that the action of rFVIIa is therefore localized to these sites **(Gabriel et al., 2004).**

No specific method is currently available to indicate the need for rFVIIa or to monitor its efficacy. Monitoring of rFVIIa efficacy should therefore be performed visually to assess the level of bleeding after rFVIIa administration, and by an assessment of the transfusion requirements after dosing **(Toschi V et al., 1997).**

rFVIIa should not be administered to patients who are unsalvageable according to the clinical evaluation of the medical team treating the patient. The risk: benefit ratio should be assessed in patients with coronary artery syndrome and in those with a presence or history of thromboembolic events. Unstable coronary plaques present TF on their surface **(Toschi V et al., 1997).** Treatment with rFVIIa may promote coagulation on these plaques, leading to acute complete coronary artery occlusion or myocardial infarction **(Ardissino et al., 1997; Badimon et al., 1999).**