Special Lecture 2.

Effect of prostaglandin E1 on cutaneous microcirculation of flap or replantation

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Both vessel arterial ischemia and venous congestion are main factors of tissue necrosis in the flap surgery. Vasodilatory and/or antithrombic agents have been used for salvage of flap necrosis. However, the therapeutic effects of those drugs are still not well elucidated.

Recently prostaglandins E1(PGE1) has been shown to ensure flap survival by producing vasodilatation of the peripheral vessels and platelet disaggregation. However, direct observation and detailed quantitative studies of the effects of PGE1 on the cutaneous microcirculation have not been reported. In the present study, we investigated microcirculatory changes in the rabbit ear chamber (REC) with an intravital microscope following intravenous administration of PGE1. The results obtained in this study indicate the PGE1 administered intravenously at a rate 200 /g/min might act directly on the vessel and cause dilatation of metarterioles and capillaries without affecting vasomotion and systematic blood pressure, resulting in the increased cutaneous blood pressure. Our experimental date may provide basic support for the therapeutic effect of PGE1.

Clinically in order to evaluate the effect of an intravenous administration of PGE1 on the cutaneous microcirculation, cutaneous blood flow, skin temperature and transcutaneous PO2 in the pedicle or free flap of operated patients were evaluated after the administration of PGE1 by the combination of several measurements. Those clinical data will be reported.

Special Lecture 3.

Blood "No Reflow" in Skeletal Muscle After Replantation = Kappa Delta Award 1994 =

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Over the past 15 years we have designed and completed a series of experiments to study the "no flow/no reflow" phenomenon in reconstructive microvascular surgery. We define "no flow" as a lack of blood flow across the anastomotic site despite technically satisfactory anastomosis, and "no reflow" as the failure of blood to

perfuse the distal tissue and return through the veins after successfully traversing the anastomosis site.

We have utilized direct videomicroscopy of the microcirculation of the rat cremaster muscle supplemented by animal models of replantation, vascular crushing, and muscle function following injury and recovery in an effort to understand the mechanism of this phenomenon.

Our studies have revealed that three events consistently occur coincident with the failure of reperfusion: 1) arterial vasospasm, 2) ischemia-induced interstitial edema and hemorrhage, and 3) thrombus formation in the venous system. Any one or all of these physiologic occurrences may induce "no reflow" in the revascularized tissue.

Experimentally induced prolonged ischemia in an animal model has enabled us to document the dynamic changes and histopathology of small arteries, including vasospasm, venous dilation, and vessel wall failure. With reperfusion after prolonged ischemia, disturbances in the microcirculation include vasospasm, vortex formation, regional stasis, interstitial edema and hemorrhage, and white blood cell and platelet aggregation. The deleterious effects of systemic acidosis, interstitial hemorrhage, denervation, and prolonged venous occlusion have been documented and published. A major effort must be directed toward preventing the no-reflow phenomenon, as we have yet to find a reliable means to reverse it.

Clinical relevance. Most of our fifteen year study of the "no reflow" phenomenon has focused on direct, highly magnified visualization of the muscle microcirculation under a variety of conditions. The application of the information gained from these laboratory experiments have resulted in contined improvement of our success rate in clinical microvascular surgery, especially in traumatic replantation of amputated limbs. The basis for nearly all of our modifications and decisions in patient selection, preoperative preparation, intraoperative regulation, surgical technique, timing of venous and arterial repairs, preservation of the ischemic part, and early postoperative magnagement emanates from our laboratory studies of the "no reflow" phenomenon.

Specifically, the application of a range of pharmacologic substances chosen for their potential for prophylactic or therapeutic influence on reperfusion has identified papaverine as a potent and reliable vasodilator. Additionally, because of the proven deleterious effects of delayed venous outflow following ischemia, we prefer to perform the venous anastomosis prior to the arterial repair in elective reconstructive procedures when feasible. In addition to intraluminal irrigation of the vessels to be repaired with warm, heparinized, balanced salt solution, we routinely work at elevated operating room temperatures and maintain patient rooms at a temperature slightly above normal due to our research findings of the negative effect of cold on replanted extremities. Since there is increasing evidence that oxygen free radical scavengers have promise in the alleviation of perfusion injury, current research efforts are now focused in this area, with a goal of eventual elimination of the occurrence of "no flow" in clinical cases.