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Prevention of ischemic injury in the small intestine

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Sir: The article by Nakamura and colleagues [2] focuses attention on methods of preventing the injury that occurs with ischemia-reperfusion in the rat small intestine using ascorbic acid. This concept could theoretically be applied to ischemia-reperfusion injury in other organ models. Ascorbic acid presumably acts as an antioxidant against the known peroxidative tissue injury caused by high-energy oxygen radicals, possibly by scavenging radicals and preventing the reactions that damage the cell membrane and perhaps intracellular elements as well.

Twenty years ago, almost identical experiments were performed in our laboratory, focusing on the phenomenology and the survival characteristics of rats subjected to acute intestinal ischemia. At that time, ischemia-reperfusion injury was already known, although the term was coined later, and the molecular basis was only suspected. Experiments in the late 1960s and 1970s were directed mostly towards organ protection from the vascular dynamic, physiological, and biochemical points of view. In our particular experiments [4], rat mortality and intestinal viability improved remarkably with the use of methylprednisolone injected intravenously in fed rats subjected to 1.5 h of ischemia. Antibiotic protection was also needed to improve mortality. Survivors presented normal histology of the small bowel when compared to the extensive destruction of the bowel wall (mostly the mucosa) that occurred in controls.

These experiments were part of our ongoing research efforts in organ protection, and the liver [3], lung [1], and re-implanted extremities [4] presented similar beneficial results with pharmacological protection. The mechanism of this beneficial effect was unknown, but we hypothesized vasodilatation, better tissue oxygenation, and cell and lysosomal membrane stabilization.

Efforts should continue to formulate biochemical strategies to reduce injury due to ischemia and reperfusion. Perhaps protocols and methods like the one described by Nakamura et al. should be tested clinically. The steroid protection studies from past literature should be revisited in the light of current knowledge of intracellular biochemistry.

References

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