Some Questions Concerning the Effects of Hydralazine on Cardiopulmonary Function in Canine Low-Pressure Pulmonary Edema

To the Editor—The report by Ghignone et al., suggests that hydralazine has a potential role in the management of noncardiac pulmonary edema.1 If confirmed in subsequent human studies, these data will be a useful adjunct to current management. However, before applying these results to clinical investigation, it may be worthwhile to discuss some methodologic questions that limit the applicability of the data to clinical practice.

First, the dose of oleic acid (0.8 ml/kg) reported in this study is greatly in excess of that usually employed to produce canine lung injury.2−4 If the reported dose is correct, it makes comparison of these data with those of other studies of oleic acid-induced pulmonary edema very difficult.

Second, the cardiovascular status of the animals is different from that which is usually observed in canine oleic acid-induced pulmonary edema. Cardiac output, as reported in this study, increased 90 min after the administration of oleic acid. Review of several other studies in which oleic acid was used to induce canine pulmonary edema suggests that oleic acid infusion reduces cardiac output.5−7 Apparently the increase in cardiac output in this study is associated with volume expansion before the administration of oleic acid and administration of fluid during the study to maintain a wedge pressure in excess of the baseline wedge pressure. It is possible that augmentation of cardiac output with volume administration increased Qs/Qt% before the administration of hydralazine, and therefore attenuated any increase in Qs/Qt% produced by the drug. The effects of hydralazine in animals that have not been volume expanded would be of considerable interest. If hydralazine supports cardiovascular function and reduces pulmonary microvascular pressure in ARDS without the need for preliminary fluid loading, it should be clinically invaluable.

Donald S. Prough, M.D.
Assistant Professor
William E. Johnston, M.D.
Assistant Professor
Department of Anesthesia
Wake Forest University
Bowman Gray School of Medicine
Winston-Salem, North Carolina 27103

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