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(Accepted for publication December 13, 1991.)

Anesthesiology
76:487, 1992

Effects of Resuscitation Using Hypertonic Saline

To the Editor:—The report by Prough *et al.* on the use of hypertonic saline (HS) for resuscitation from hemorrhagic shock in the presence of increased intracranial pressure¹ seems a worthwhile contribution to a growing literature regarding the use of HS, and points once more to the potential clinical usefulness of this particular therapy. However, in reviewing their data, I noted that resuscitation with HS did not significantly increase cardiac output and in fact appeared inferior to resuscitation with normal saline in restoring cardiac output to baseline levels. This finding is not in keeping with multiple previous studies,²⁻⁴ which have consistently shown HS to be quite effective in restoring cardiac output to normal or supernormal levels after hemorrhage and in many cases to be more effective than normal saline in this regard. Given that cardiac output indirectly affects cerebral perfusion pressure and cerebral blood flow, how might this anomalous finding affect the validity of Prough *et al.*'s conclusions?

In addition, I was surprised by the authors' assertion that HS can increase myocardial contractility. The studies that are widely quoted to show such an effect actually looked at the effects of serum hyperosmolality induced by sucrose or urea on the heart⁵ and the effects of hyperosmotic sucrose on myocardial contractility.⁶ Later reports have shown that HS is, as opposed to other hyperosmotic agents, a direct myocardial depressant.^{7,8} Other authors have proposed a pulmonary/vagally mediated reflex resulting in systemic circulatory changes and increased cardiac contractility in response to HS^{9,10}; recent studies have cast doubt on that assertion.^{11,12} From the available data, I think it is safe to conclude that the improvement in cardiac output seen after resuscitation with HS is secondary to changes in preload alone and is not related to improved cardiac contractility.

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(Accepted for publication December 19, 1991.)