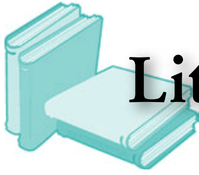

Literature Reviews

Arginine vasopressin in advanced cardiovascular failure during the post-resuscitation phase after cardiac arrest.

Mayr V, Luckner G, Jochberger S, Wenzel V, Ulmer H, Pajk W, Knotzer H, Friesenecker B, Lindner K, Hasibeder W, Dunser M. Resuscitation 72:35-44, 2007.

Reviewers:
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Abstract Excerpt:

Arginine vasopressin has received much interest in the fields of cardiovascular anesthesia and critical care. Researchers in Austria and Switzerland have done much to establish the value of a bolus of the drug as an aid to cardiopulmonary resuscitation (CPR). Now, they report that a sustained infusion of vasopressin may be an aid to recovery in the event of cardiovascular failure after initially successful resuscitation. Patients with inadequate perfusion following CPR were treated with intravenous fluid until stroke volume was optimized. If necessary, milrinone and then epinephrine infusions were started. If the mean arterial pressure (MAP) continued to be <65 mmHg, a norepinephrine infusion was added. In 23 patients, incremental increases in the norepinephrine infusion over two hours did not achieve target MAP, and an infusion of vasopressin was started at 4 U/hr. No bolus doses of vasopressin were given, and the infusion rate remained constant at 4 U/hr while the norepinephrine dose was adjusted to maintain MAP >65 mmHg. The supplemental vasopressin infusion significantly increased MAP and decreased norepinephrine and inotropic drug requirements in nearly all of the hemodynamically failing patients. Despite an apparent hemodynamic benefit of vasopressin, survival was observed in only 8 out of 23 patients. However, a lower survival was expected without the aid of vasopressin, and at least 7 of the 15 nonsurvivors who improved on vasopressin had incurred fatal brain damage at the time of cardiopulmonary arrest.

Reviewers' Comments

Vasopressin, also known as antidiuretic hormone, is an "old" drug. The pressor action of posterior pituitary extract was discovered in 1895. Impure preparations were clinically available before the 1950s, when du Vigneaud garnered a Nobel Prize in Chemistry for elucidating the structures of peptide hormones including vasopressin. The drug was little used as a clinical vasopressor after early reports of its adverse effects on myocardial perfusion. For instance, the drug was proposed as a stress test for coronary insufficiency in 1947, but "cardiac accidents" were reported soon thereafter (JAMA 1951;146:1126). However, interest was generally rekindled in 2000 when the American Heart Association guidelines endorsed consideration of a bolus of vasopressin as a pressor for CPR. Since then, prolonged infusions of the potentially dangerous drug have shown benefit in other desperate situations such as the "vasoplegia" syndromes that can complicate sepsis or develop during and/or after cardiovascular surgery. Furthermore, vasopressin is the most clinically reliable vasoconstrictor in the setting of hyposensitivity to norepinephrine. Interestingly, vasopressin sensitivity often increases when that to norepinephrine decreases (Crit Care Med 2007;35:33). This clinical report adds to the growing mountain of evidence indicating that vasopressin is an effective vasoconstrictor in clinical situations where traditional vasoconstrictors may be ineffective.