Improving outcomes from high risk surgery using functional haemodynamic monitoring-directed resuscitation

It is clear from numerous clinical studies that tissue hypoperfusion is bad and that avoidance of ischemia improves outcome from stress states including surgery. Thus, the rapid restoration of normal hemodynamics by conventional means, including fluid resuscitation and surgical repair, results in a superior outcome than inadequate or delayed resuscitative efforts. Since critically ill patients often have abnormal blood flow regulation, increasing oxygen delivery (DO_2) to supranormal levels theoretically may treat the lethal occult tissue hypoxia that is a hallmark of many forms of circulatory shock. Accordingly, interest centered on "hyper-resuscitation" such that DO_2 is exogenously increased to supranormal levels, levels often seen in subjects who spontaneously survive acute circulatory insults, the so-called "survivor levels of DO_2." Most studies that have aimed at augmenting DO_2 or VO_2 to "survivor levels" have documented that if DO_2 can increase, subjects do better. However, this improvement in survival appears to be independent of whether the subject was part of the group with intentional augmented DO_2. Furthermore, aggressive therapies aimed at augmenting DO_2 may actually increase mortality in experimental groups! Thus, a low DO_2 in a critically subject is probably a marker of critical illness, rather than a parameter of effective resuscitative therapy. Interestingly, the most impressive beneficial outcomes from clinical trials have all included prevention of hypoperfusion rather than resuscitation from shock. Finally, prospective clinical trials documented that both pre-operative, intra-operative and post-operative resuscitation strategies aimed at sustaining a relatively high DO_2 and MAP markedly decrease post-operative complications, length of stay and the associated organ dysfunction. Often these benefits are seen without measurable differences in VO_2 during therapy. Since there is no such thing as a normal cardiac output, just an adequate or inadequate one, relative to the varying metabolic needs of the subject, targeting DO_2 to some target elevated value relative to normal values for otherwise resting healthy individuals make no sense. A major benefit of aggressive resuscitation therapy would potentially be realized if efforts were directed at more rapid identification of subjects at risk for tissue ischemia from the general hospital population and the more rapid emergency resuscitation and definitive therapy of these subjects based on metabolic end points. Such end points include tissue CO_2 levels, lactate flux and organ system function. However, once circulatory shock and/or organ dysfunction has occurred there appears to be little additional benefit and real risk of harm from aggressive resuscitation therapies that increase DO_2 or VO_2 to levels above which would otherwise be considered normal. In those subjects maintaining a mean arterial pressure $\geq$ 65 mm Hg, a cardiac index $\geq$ 2.5 l/min/m^2 and a venous to arterial PCO_2 difference < 6 mmHg and a $ScvO_2$ >70% appear to be reasonable therapeutic goals.