END POINTS IN RESUSCITATION OF SHOCK: UNDERSTANDING THE EVOLUTION

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ABSTRACT

In the last two decades resuscitation strategies have gone through revolutionary changes. Ideas once held sacred have been questioned and the very foundations on which these strategies were based shaken. Resuscitation strategies basically revolve around three questions:

1. How much?
2. How fast?
3. When to stop?

Evolutions of shocked patient management concepts show three distinct phases.

- Phase 1 – Clinical parameter based.
- Phase 2 – Oxygen variables based.
- Phase 3 – Cellular perfusion based.

Accepted criteria for endpoints of resuscitation too have undergone similar changes in paradigm and can be grouped under similar phases.

Key Words: *Shock, Clinical parameter, cellular perfusion, resuscitation.*

INTRODUCTION

Most of the primary descriptive features of the shocked state are clinical in nature. They included bedside patient observations vital signs and urine output etc. Success or failure of resuscitation was measured by ability to normalize these parameters. In any group of patients presenting with severe shock approximately 12% - 18% mortality or significant morbidity will occur.¹ The first real challenge to the adequacy of clinical signs as end point of resuscitation was the landmark paper by Shires and Canizaro² who demonstrated a 20% mortality rate over long term in animals successfully resuscitated using fluid volumes based on direct measurements of intra and extra vascular fluid deficits. Important variables of shock like tissue acidosis and oxygen

debt cannot be measured by clinical parameters.³ Advances in the invasive and noninvasive monitoring systems now allow us sensitive and specific data on the organ and cellular level in established shocked states.⁴ Clinically evident outcomes alone have given us.

MATERIALS AND METHODS

Medline and Pubmed search was done using the key words, Shock, Resuscitation, End Points. Limitations set were English language and articles with full text. The authors independently gave points of importance to the list of articles obtained. 36 of the 165 articles listed thought to be the most relevant were selected. The WHO authorized HINARI web site was used to obtain full text access to the selected articles.

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Finding of the three distinct phases of shock and its resuscitation lead us to put or conclusions in the three phases.

PHASE I: CLINICAL PARAMETERS

Blood pressure: Higher success rates are attained if clinical index of suspicion is focused enough to recognize this state before the development of hypotension. Hypotension means that all the compensatory mechanisms have been overcome and should always be regarded as a pre morbid state.

It's very easy to presume that magnification of hypotension (Orthostatic hypotension) could be an early indicator of shock. 50% of normovolemic patients demonstrate positive orthostatic changes.⁵ The MAP (Mean Arterial Pressure) initially recorded in survivors was found to be only slightly higher than non survivors.⁶ The cuff pressures of shocked patients will poorly correlate with those obtained by direct monitoring of MAP using arterial catheter.⁷

Heart rate: Tachycardia usually accompanies shock. It must be remembered that age and medications will modify its response.⁸

Shock Index: Described by Allgower and Buri⁹ to overcome the inaccuracy of BP and HR is believed to bear an reliable inverse relation to LVSV (left ventricular stroke volume) in circulatory failure.¹⁰ It was concluded that even with apparently stable vital signs shock index (>0.9) is a specific indicator of acute circulatory failure which would later need intensive resuscitation therapy. It is not very sensitive though as normal index is recorded in normovolemic cases. MAP, HR and UO are potentially useful guidelines of volume status in the initial stages of resuscitation. If sole reliance is placed on them alone for endpoints many patients thought to be fully resuscitated will have reduced intravascular volumes and thus at risk of incurring morbidity if untreated shock.¹¹ This particular situation was termed as compensated shock by Fiddian-Greene.¹² Here clinical parameters are normal. The oxygen debt at the tissue and organ level has not been repaired, leading to continued morbidity and mortality unless mo.

PHASE II: OXYGEN CONSUMPTION AND TRANSPORT

Shoemaker¹³ reported in 1988 that MAP and cardiac output (CO) decreased while patients were in shock and increased immediately upon resuscitation. After this initial time period these variables did not bear a consistent relationship and many changes in CO and oxygen transport variables occurred that would have gone undetected without invasive monitoring. Bishop¹⁴ reported their findings on a series of 90 critically ill patients with 33% mortality all of whom had routine pulmonary

artery catheter inserted early in the course of their management. MAP was rapidly restored in all patients and mean values of MAP and HR was comparable in survivors and non survivors. Survivors had significantly higher cardiac indices and oxygen transport variables throughout the course of their stay. The conclusion made was that routine clinical observations can indicate the presence of shock, but their normalization cannot be used as the endpoint of resuscitation. Shoemaker¹⁵ and Bishop's base deficits are extremely attractive due to the ease and speed with which they can be analyzed. They have also been claimed to accurately reflect hemodynamic and tissue perfusion changes.^{18,19}

Abramson²⁰ et al have demonstrated 100% survival in patients who cleared lactate within 24h. Survival fell to 15% in patients who had persistent lactic acidosis. Most importantly levels of base deficits, anion gap and lactate did not correlate after the first 12h of intensive care. Reliance on these parameters alone would have resulted in wrong treatment in up to 33% to 50% patients. Normally global oxygen extraction is less than 25% of oxygen delivery. Increased extraction ratios mean decreased delivery and shock. Shoemaker must be credited for the concept of supranormal values of resuscitation where hemodynamic status is pushed from being flow dependent to flow independent. This will occur at:

- Cardiac index ≥ 4.5 L/min/m².
- Oxygen delivery ≥ 750 mL/min/m².
- Oxygen consumption ≥ 180 mL/min/m².

He has published many series where supranormal resuscitation leads to improved outcomes (mortality/organ failure). The only genuine criticism to his theory is the mathematical coupling phenomenon. This is an apparent but false correlation between two parameters in this case oxygen delivery and extraction that share common variables. This has been well put up by Phang²¹ and Hanique.²² All these cannot tell us about the specific tissue beds that may remain underperfused in spite of correcting the systemic indices of tissue perfusion. Now organ specific monitoring is promising to be the guide to ensure adequate resuscitation.

PHASE III: CELLULAR BEDS

Shock per se is inadequate perfusion at the tissue level, thus tests reflecting cellular metabolism will be of major value. Metabolic acidosis, elevated lactate and base deficits are markers of poor prognosis. Even though reversing them is not the perfect endpoint of resuscitation. Early in the shocked state blood flow is selectively shunted from splanchnic and subcutaneous tissues. Restoration of blood flow to more vital organs like brain, kidneys and heart cannot mean that blood flow to all the tissue beds has been achieved.²³⁻²⁶ This lets us

hypothesize that restoration of these two beds should indicate restoration of all other beds.²⁷⁻²⁹

Gastric Tonometry: All the prospective clinical studies in which gastric intramucosal pH has been examined as an index of tissue oxygen have found it to be a useful supplement. Roumenn et al³⁰ reported that all patients who maintained normal pH recovered without complications. 37.5% who did not developed major complications and 25% died. There was no significant correlation between pH values. Lactate levels, base deficits. APACHE II scores. Chang et al³¹ too report that uncorrected low pH in the first 24h period means high mortality and this does not correlate with other variables. There have been two clinical studies using gastric tonometry to guide resuscitation. Gutierrez³² showed that patients with normal gastric pH on admission but later developing mucosal acidosis improved with therapy to increase delivery of oxygen and decrease oxygen demand. If the patients had acidosis on admission then no benefit was seen, maybe due to irreversible shock. Ivatury³² compared global oxygen transport indices with ga. Calculated pH during the low flow shocked state will underestimate the mucosal acidity.³³

Tissue Oximetry: Resuscitation is improving tissue hypoxia. The most widespread research in tissue oximetry (measuring Po₂) has been in the wound healing field. Gosain³⁴ first compared global indices to sub and trans-cutaneous Po₂ during graded hemorrhage. They measured tissue blood flow at each stage of blood loss. S/C Po₂ was a sensitive predictor of volume loss to up to 40% loss. The important concept of "Oxygen Challenge Test" put forward by Johnson³⁵ needs mentioning. It is used to measure adequacy of tissue perfusion. Response of tissue Po₂ to increased inspired O₂ is measured. Hypothesis is that lack of response indicates increased tissue oxygen extraction due to inadequate perfusion. Of the 12 patients who demonstrated inadequate perfusion by this criteria 10 responded to fluid boluses by increasing their tissue Po₂. Hopf et al³⁶ used this method on trauma patients considered adequately resuscitated by global criteria. 75% failed the O₂ challenge test in the first 48h of hospitalization.

CONCLUSION

No single test or device can be relied in all the situations of the shocked state. Clinical parameters are irreplaceable to identify early the shocked state. Derangements of the clinical parameters imply decompensation, thus normalization of clinical parameters does not mean adequate resuscitation. Shock once defined at cellular level demand monitoring at the cellular level. Even with all this technology the best tool is a trained physician.

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