



Vascular hyporesponsiveness to vasopressors in septic shock: from bench to bedside

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Purpose To delineate some of the characteristics of septic vascular hypotension, to assess the most commonly cited and reported underlying mechanisms of vascular hyporesponsiveness to vasoconstrictors in sepsis, and to briefly outline current therapeutic strategies and possible future approaches. Methods Source data were obtained from a PubMed search of the medical literature with the following MeSH terms: Muscle, smooth, vascular/physiopathology; hypotension/etiology; shock/physiopathology; vasodilation/physiology; shock/therapy; vasoconstrictor agents. Results Nitric oxide (NO) and peroxynitrite are crucial components implicated in vasoplegia and vascular hyporeactivity. Vascular ATP-sensitive and calcium-activated potassium channels are activated during shock and participate in hypotension. In addition, shock state is characterized by inappropriately low plasma glucocorticoid and vasopressin concentrations, a dysfunction and desensitization of alpha-receptors, and an inactivation of catecholamines by oxidation. Numerous other mechanisms have been individualized in animal models, the great majority of which involve NO: MEK1/2-ERK1/2 pathway, H₂S, hyperglycemia, and cytoskeleton dysregulation associated with decreased actin expression. Conclusions Many therapeutic approaches have proven their efficiency in animal models, especially therapies directed against one particular compound, but have otherwise failed when used in human shock. Nevertheless, high doses of catecholamines, vasopressin and terlipressin, hydrocortisone, activated protein C, and non-specific shock treatment have demonstrated a partial efficiency in reversing sepsis-induced hypotension.

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