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Vasopressor drugs and renal function in septic acute kidney injury

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Abstract : Vasopressor drugs are the primary treatment in volume resuscitated patients with septic shock. Noradrenaline is the most common vasopressor drug used, but there is increasing interest in the use of vasopressin and also angiotensin II as primary vasopressor agents, as well as in the treatment of patients with catecholamine-resistant hypertension.

Since these three drugs have different mechanisms of action they have contrasting effects on systemic haemodynamics and renal function. In a pre-clinical model of septic shock, that has a similar phenotype to human sepsis, the effects of noradrenaline, vasopressin and angiotensin II have been compared. Noradrenaline and angiotensin II had little effect on the increased cardiac output in sepsis, whereas vasopressin reduced cardiac output and maintained arterial pressure by greater levels of peripheral vasoconstriction, particularly in the mesenteric vascular bed. In further studies, to elucidate the role of increased nitric oxide release in sepsis, the effect of inhibition of renal nitric oxide was examined. Although a non-specific inhibitor of nitric oxide reduced the elevated renal blood flow to normal, it did not improve renal function. Further studies have examined the effect of noradrenaline and angiotensin II on renal microvascular blood flow and oxygenation. In sepsis there was a selective decrease in renal medullary perfusion and oxygenation, which was enhanced by noradrenaline, but not angiotensin II. These findings indicate that in sepsis global renal blood flow is not related to renal function, whereas changes in renal medullary perfusion and oxygenation precede the development of AKI. Developing therapies that improve renal medullary oxygenation may therefore reduce the development of septic AKI.