

.....

The Renal Effects of Noradrenaline and Dopamine

Rinaldo Bellomo^a, *Claudio Ronco*^b

^a Department of Intensive Care, Austin & Repatriation Medical Centre, Heidelberg, Melbourne, Vic., Australia and

^b Divisione di Nefrologia, Ospedale San Bortolo, Vicenza, Italy

Introduction

Septic shock, systemic inflammation (trauma, major surgery, cardiopulmonary bypass and the like), or pharmacological vasodilatation (phosphodiesterase inhibitors, sedative drugs, epidural or spinal block) are usually associated with systemic hypotension despite normal or increased cardiac output [1]. Under these circumstances, hypotension may persist despite vigorous volume expansion. Potent systemic vasopressor agents, such as noradrenaline or so-called high-dose dopamine, can then be used to restore an acceptable mean arterial blood pressure [2–5]. Under these conditions, renal dysfunction is common (oliguria and/or a rising serum creatinine). In these patients, the addition of low-dose dopamine infusion is believed to improve renal blood flow and protect the kidney from further injury. However, these areas of clinical practice are fraught with controversy. In this chapter, we will review the evidence in favor or against the use of dopamine and/or noradrenaline in critically ill patients and seek to provide intensivists and nephrologists with a clinically relevant update aimed at helping clinicians make informed decisions in the care of their patients.

Why Use Vasopressors?

The rationale for vasopressor therapy in hypotensive states is based on the physiological knowledge that, in all regional circulations, including the renal, splanchnic, cerebral and coronary beds, blood flow is autoregulated. This means