This review on the role of adenosine in renal function is divided into three parts. In the first part, the action of exogenous adenosine on renal blood flow, glomerular filtration rate, and renin secretion is considered. In the second part, evidence that renal tissue levels of adenosine correlate inversely with those of ATP is presented. The third part describes adenosine as the metabolic mediator in the kidney that regulates glomerular filtration rate and renal renin secretion in response to the sodium chloride load of the thick ascending limb of Henle's loop.

**EXOGENOUS ADENOSINE**

The first observation of a renal vasoconstriction induced by adenosine was reported in 1929 by Drury and Szent-Györgyi [1]. This finding was confirmed by Thurau in 1964 [2] and later by other investigators [3–7]. Following intrarenal injection of adenosine, the renal vasculature responds immediately with marked constriction, as shown in Figure 25.1. However, during continuous intraarterial infusion of adenosine, renal blood flow decreases initially and then returns to preinfusion levels within 2–5 min. Following cessation of adenosine infusion, there is a short-lasting increase of renal blood flow (see also Fig. 25.4). Although renal vascular resistance is unchanged or even reduced during adenosine infusion, glomerular filtration rate is decreased by 10% to

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20% [5, 8]. Micropuncture studies in dogs [8] and rats [9] demonstrated that adenosine induces sustained afferent arteriolar vasoconstriction of the outer cortex (Fig. 25.2). Since whole kidney vascular resistance did not change, it was concluded that the vasculature of the deep cortex dilates in response to intrarenal adenosine infusion. In fact, deep cortical vasodilation due to adenosine was demonstrated by Spielman et al. [10], using radioactive labeled microspheres in dogs.

In this situation, the question arises of what the physiological response of the renal vasculature is to adenosine. To answer this question, one should keep in mind that single injections of adenosine unequivocally induce vasoconstriction. Also, endogenous adenosine, when transiently released by the kidney, induces renal vasoconstriction (see later discussion). At present, we do not know whether deep cortical vasodilation due to exogenous adenosine can also be elicited by endogenous adenosine. Under certain conditions, exogenous adenosine-induced vasoconstriction does not wane but persists during the entire infusion time in dogs. One such condition is elevation of ureteral pressure to 65 mm Hg [Spielman and Osswald, 1981, unpublished observation]; the other is furosemide-induced diuresis [11; unpublished observation].

Therefore, we can state that the vascular response of the kidney to intraarterial infusion of adenosine is characterized by outer cortical vasoconstriction and deep cortical vasodilation, and the deep cortical dilation is variable and probably controlled by other unknown factors.