3
The kidney and hypertension

It is difficult, considering the volume of literature available on the subject, to present a brief synopsis of the kidney and hypertension. The crux of the matter, however, is that the juxtaglomerular apparatus (Figure 22) consists of stretch receptors in the wall of the afferent arteriole working in conjunction with the sodium-sensitive cells of the macula densa, so that either (1) diminished renal blood flow or (2) a decrease in the sodium being delivered to the distal tubule will cause the release of renin (see Figures 6 and 23). This structure, described by Goormaghtigh in 1932, can be pictured as shown.

The JGA occupies the vascular pole of the glomerulus. Immunofluorescence confirms that its granules contain renin. There is a rich sympathetic nerve supply, and when there is increased sympathetic drive there is constriction of the afferent arteriole, so the pressure of the JGA is lowered and renin release enhanced. This happens in hypertension, cirrhosis, or shock, when there is raised intracranial pressure, or diminished flow due to obstruction to the circulation as in renal artery stenosis. It might also happen under the influence of ganglion-blocking drugs, for although the renal arterioles are dilated, the pressure at the JGA is low. On the other hand, if there is postural hypotension due to autonomic dysfunction (as in diabetic neuropathy), there is a failure to release catecholamines on standing. In this situation there is no
compensatory renin release, as occurs in the normal person in an upright posture. The JGA is therefore part of the baroreceptor system which includes the carotid body, aortic and atrial stretch receptors.

As the sodium flux down the tubules increases there is increased reabsorption by the ascending loop of Henle up to a limit, but inevitably the sodium concentration in the distal tubule must rise and this switches off renin release. Conversely in sodium depletion, renin–angiotensin–aldosterone generation will be activated, for it is the physiological sodium-conserving mechanism. Figure 23 summarizes renin and aldosterone control.

In normal persons and those with essential hypertension it is the sympathetic drive to the JGA which determines renin release. Yet the baroreceptor is relatively insensitive as the renal artery pressure has to drop by 50% before renin release is increased!

Figure 23. Renin and aldosterone control