PHYSIOLOGY OF THE KIDNEY AND BODY FLUIDS

ROBERT F. PITTS

ROBERT F. PITTS, Ph.D., M.D.
Professor of Physiology, Maxwell M. Upson Department of Physiology and Biophysics, Cornell University Medical College

KIDNEY and BODY FLUIDS

An Introductory Text

THIRD EDITION

YEAR BOOK MEDICAL PUBLISHERS INCORPORATED
35 EAST WACKER DRIVE, CHICAGO

COPYRIGHT © 1963, 1968 AND 1974 BY YEAR BOOK MEDICAL PUBLISHERS, INC.

All rights reserved. No part of this publication may be reproduced, stored in a retrieval system, or transmitted, in any form or by any means, electronic, mechanical, photocopying, recording, or otherwise, without prior written permission from the publisher. Printed in the United States of America.

Reprinted, May 1964
Reprinted, March 1965
Reprinted, January 1966
Reprinted, January 1967
Second Edition, 1968
Reprinted, February 1969
Reprinted, February, 1970
Reprinted, February, 1971
Reprinted, January 1972
Third Edition, 1974

Library of Congress Catalog Card Number: 73-94395

Cloth: 0-8151-6702-4 Paper: 0-8151-6703-2

Preface to the Third Edition

THE RESPONSE of medical students and graduate students in the medical sciences to the first and second editions of this monograph has been gratifying to the author. Moreover, the book seems to have found a receptive audience abroad, for it has now been translated into Italian, French, German, Japanese and Spanish. In Madison Avenue jargon, "It seems we have been doing something right." This third edition continues in the tradition of the first two. Some errors have been corrected and some material has been added, most notably on glomerular hemodynamics, on the site of the glomerular filtration barrier, on the mechanism of amino acid reabsorption, on the buffering of acid by bone, on renal tubular acidosis, and on the mechanism and control of ammonia secretion. Two sections have been added on clinical problems: the intact nephron hypothesis and the tradeoff hypothesis; I have done so not because I consider myself at all qualified as a clinical nephrologist, but because of my interest in the physiologic implications. Finally, a new chapter on renal metabolism—a somewhat neglected field—has been added, in the hope that it will stimulate future work in this area. As in the second edition, Erich E. Windhager has revised Chapter 7, Mechanisms of Reabsorption and Excretion of Ions and Water.

I would advise the beginning student to read the following Preface to the First Edition, because it contains factual material not covered elsewhere in the text and because it more or less sets the stage for the ensuing discussion of renal functions.

R.F.P.

Preface to the First Edition

MORE THAN 100 years ago, Claude Bernard pointed out that the medium in which we exist is not the atmosphere which surrounds us but the blood and tissue fluids which bathe our muscles, glands and brain. He described this internal environment as a cosmos, elaborately isolated from and protected from the vicissitudes of the external world by a variety of physiological devices which operate to preserve and stabilize its physical properties, chemical constitution and volume. The partial pressures of oxygen and carbon dioxide, the concentrations of nutrients and wastes, the temperature, the hydrogen ion concentration, the osmotic pressure, the concentrations of the several cations and anions, and the volume of this internal fluid environment are all precisely maintained within narrow limits of normal.

The kidneys play a prominent role in regulating the concentration of metabolic wastes, and the osmotic pressure, the volume and the ionic composition of our internal environment. Claude Bernard argued that we have achieved a free and independent life, mentally and physically, by becoming relatively independent of our external environment. It may therefore be claimed, with some justification, that our present high station in the animal kingdom ultimately depends on our kidneys.

The kidneys are commonly described as excretory organs, but the assignment of such a limited role scarcely does them justice. They are primarily organs which regulate volume and composition of the internal fluid environment; their excretory function is incidental to their regulatory function. Primacy of regulatory function may be illustrated as follows: Consider two persons—one with normal renal function, the other a patient with long-standing, chronic, bilateral renal disease. Suppose both individuals ingest identical diets, containing 70 Gm of protein, 250 Gm of carbohydrate and 100 Gm of fat (2,200 Cal). Suppose each diet contains 5 Gm of sodium chloride, 2 Gm of potassium and 1 Gm each of phosphorus and sulfur; suppose fluid intakes are equal and generous. How, then, will the compositions of the urine of these two individuals differ? The answer is: they will not differ.

Each person will excrete per day 12 Gm of nitrogen, 5 Gm of sodium chloride, 2 Gm of potassium and 1 Gm each of phosphorus and sulfur. If fluid intake is high, the urine volumes will be the same. True, the patient with chronic renal disease may excrete a little protein, some red blood cells and casts, but the major excretory products will be the same.

Why, then, is the physician concerned about the patient with chronic renal disease? The answer is: the diseased kidney is deficient in its capacity to regulate the composition and, to a lesser extent, the volume of the internal fluid environment. The patient with renal disease will excrete each day 12 Gm of urea nitrogen derived from the 70 Gm of dietary protein exactly as does the normal

此为试读,需要完整PDF请访问: www.ertongbook.com

individual, but the plasma urea nitrogen concentration may be 100–150 mg per 100 ml, rather than the normal 10–15 mg per 100 ml. The patient will excrete 2 Gm (51 mEq) of potassium, but the plasma concentration may be 5–8 mEq per liter, instead of the normal 4 mEq per liter. The plasma concentrations of phosphate and sulfate may increase some 3–6 fold; that of calcium may be low. If salt or fluid intake is restricted, continued excretion may rapidly deplete salt and water reserves, leading to a far greater reduction in volume and to a much greater distortion of composition of the internal fluid environment than that suffered by a normal person. Salt restriction alone may lead to hyponatremia (reduced sodium concentration of the blood) and to a reduction in osmotic pressure of the body fluids. Metabolic acidosis gradually develops as a consequence of progressive failure of the mechanisms which regulate the bicarbonate concentration of extracellular fluid.

When, as a consequence of the inexorable progress of renal disease, of some added regulatory burden or of some intercurrent infection which precipitates renal failure, the regulatory capacities of the kidneys become so compromised that the composition and volume of the internal fluid environment deviate markedly from normal, vital organs fail and the individual succumbs. The success of the physician in prolonging the life and useful existence of the patient with chronic renal disease is in proportion to his ability to stay the progress of the disease and to his skill in helping the patient compensate for his renal regulatory deficiencies. Unfortunately, at present no specific therapy is available for the treatment of bilateral chronic renal disease. Regeneration of damaged renal tissue does not occur, and restoration of function by renal transplantation is successful only when the donor and recipient are immunologically similar.

The purpose of this volume is to describe the basic renal mechanisms of glomerular ultrafiltration, tubular reabsorption and tubular secretion; to clarify the operation of these mechanisms in the regulation of volume and composition of the internal fluid environment; and to provide a basis for an appreciation of the consequences of renal diseases and of the principles involved in therapy.

I am indebted to Miss Michelena Perrotta and Miss Sally Aleks for typing the manuscript, to Miss Jeannette de Haas and Mrs. Martha MacLeod for assistance in preparing the figures, and to Drs. Gerhard Giebisch and Richard Kessler for criticizing portions of the manuscript. I am also indebted to authors and publishers who permitted the reproduction of figures and data.

R.F.P.

Table of Contents

1

Anatomy of the Kidney

Gross Morphology of the Kidney

THE KIDNEYS are paired, somewhat flattened, bean-shaped organs, which lie retroperitoneally on either side of the vertebral column against the posterior abdominal wall. Together they weigh about 300 Gm and thus constitute 0.4% of the weight of the body. They are embedded in a mass of fat and loose areolar tissue. The upper pole of the right kidney rests on the 12th rib; that of the left kidney, on the 11th and 12th ribs. Each kidney lies posteriorly against the diaphragm, lateral lumbocostal arch, psoas major, quadratus lumborum and the tendon of the transversus abdominis (1).

The renal artery and nerves enter, and the renal vein, lymphatics and ureter leave, the kidney through the hilus, a longitudinal slit occupying somewhat less than the middle third of the medial border of the organ. The hilus opens into a more extensive but shallow and flattened C-shaped space, the renal sinus (Fig. 1–1), completely surrounded by kidney tissue. The renal sinus is largely occupied by the renal pelvis and its connecting major and minor calices. The chinks are filled with loose areolar and adipose tissue, through which course the renal vessels and nerves. The kidney is invested by a tough, collagenous and nearly indistensible capsule, loosely adherent to the underlying glandular tissue, from which it can be easily stripped. The capsule is reflected into the sinus through the hilus. The outer layer of this reflected area is anchored to blood vessels and the pelvis, and the inner layer forms a lining for the sinus.

The walls of the sinus are studded with

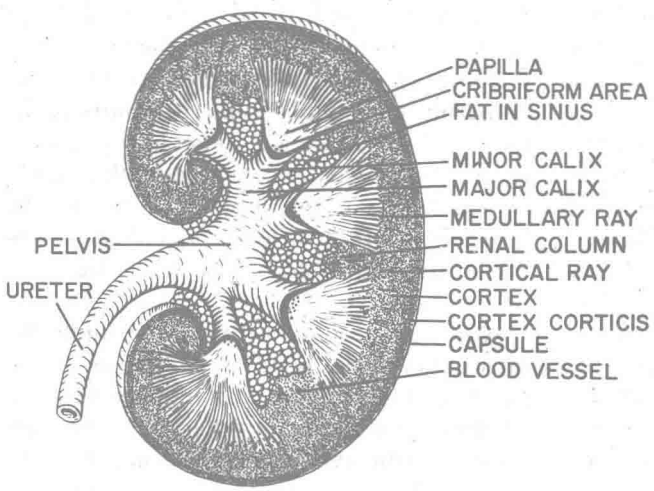

Fig. 1–1. — Longitudinal section of the kidney.

conical elevations, the renal papillae, which average 8–10 in number, although as many as 18 may be present. The papillae are flattened cones, 7–10 mm in height, with elliptical bases. The apex of each papilla—the cribriform area—is pierced by 18–24 minute orifices, barely visible to the naked eye; these are the openings of the papillary ducts of Bellini, which in turn are formed by the terminal fusion of many collecting ducts.

Each papilla is thrust into a terminal extension of the renal pelvis, a minor calix (Gr. *kalyx* = cup). Urine flowing from the orifices of the ducts of Bellini enters a minor calix, flows on into either the superior or the inferior major calix, and then flows into the renal pelvis and finally out, via the ureter, to the urinary bladder. The walls of the calices, pelvis and ureter contain smooth muscle, which contracts rhythmically and propels the urine along its course in peristaltic spurts.

The kidney of man is a multilobed organ. Each renal lobe is a pyramidal mass of tissue, the base forming the surface of the kidney, the apex forming the papilla. In the fetal kidney, lobulation is evident on the surface; but, in the adult kidney, fusion is so complete that no traces of external lobular markings persist. In insectivores and rodents, the entire kidney can be considered to be a single lobe, ending in a single papilla. The kidney of the dog is much like that of man, giving, on the surface, no evidence of its lobular structure. However, many mammals retain in their adult form the surface lobulation seen in the fetal human kidney.

The cortex, which is reddish brown in color, not only forms the shell of the kidney but also dips down between adjacent pyramids toward the renal sinus; these inward extensions are known as the columns of Bertin. The outermost layer of the cortex is finely granular, whereas the deeper portions are marked by radially arranged columns—medullary rays—extending outward from the pyramids. The uniform granularity of the outer cortex corticis is attributable to the fact that it is made up solely of highly convoluted proximal and distal tubules, which lie randomly in all planes. The radial columns of the deeper cortex are composed of straight segments of proximal and distal tubules, collecting ducts and blood vessels. Glomeruli are scattered throughout the granular cortex between the medullary rays but are absent from the outermost cortex corticis.

The medullary pyramids exhibit a fanlike striation, the rays of the fan spreading outward from the tips of the papillae, through the medulla proper, to penetrate the deeper layers of the cortex. The striation is attributable to the parallel course of loops of Henle and blood vessels. The color of the pyramids shades from reddish brown at the corticomedullary junction to gray-brown at the tips of the papillae. Often an outer zone of the medulla, made up of an outer and an inner stripe, and an inner zone, the papilla proper, can be distinguished. The differentiation of outer and inner stripe depends on the relative proportions of thick and thin limbs of Henle's loops. The only tubular elements of the inner zone are thin limbs of Henle's loops.

Gross Vascular Supply of the Kidney

Commonly, each kidney is supplied with blood by a single renal artery (Fig. 1–2), which arises from the abdominal aorta just below the superior mesenteric and middle suprarenal arteries. Either just before or just after entering the hilus, each renal artery divides into a series of branches, which pass dorsal and ventral to the pelvis through the areolar and adipose tissue of the sinus. The branches in the anterior group are the more numerous and supply some two thirds of the kidney. These arterial branches pass between the calices and penetrate the parenchyma between the pyramids. Within the parenchyma, these arteries are called interlobar, because they course between the lobes or pyramids.

At the junction of cortex and medulla, the interlobar arteries bend over the bases of the pyramids to form a series of incomplete arches, the arciform arteries. Interlobular arteries arise at right angles from the arciform arteries and run radially toward the periphery in the cortical medullary rays. In their course

此为试读,需要完整PDF请访问: www.ertongbook.com

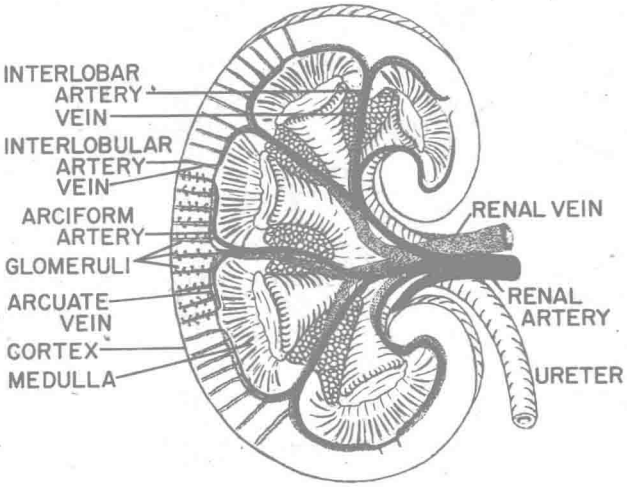

Fig. 1–2. – Gross morphology of the renal circulation.

through the cortex, they give rise to short lateral branches, the afferent arterioles, each of which supplies a glomerulus. Certain of these interlobular arteries continue through the cortex corticis to supply the capsule; some anastomose with the arterial supply of the perirenal fat and areolar tissue.

The pattern of the major venous drainage of the kidney corresponds in general to that of the arterial supply. Interlobular veins arise on the surface in stellate sinuses, which receive capillaries from the cortex corticis. These veins penetrate the cortex, receive additional cortical venules and the ascending venae rectae from the medulla, and join the arcuate veins, which arch over the bases of the pyramids. The arcuate veins form complete, or true, arches and anastomose freely. Interlobar veins drain the arcuate veins and emerge from the renal parenchyma in the columns of Bertin. In the sinus, they assemble to form the renal veins, which join the vena cava.

The details of the glomerular and peritubular capillary circulations are best considered along with the morphology of the nephron.

Microscopic Structure, Vascular Supply and Innervation of the Nephron

NEPHRON. – In man, each kidney is composed of 1 – 1¼ million units, all basically similar in structure and presumably also grossly similar in function. Each unit, which is termed a nephron (Fig. 1 – 3), consists of a renal, or malpighian, corpuscle (glomerulus and Bowman's capsule), a proximal convoluted tubule, a loop of Henle and a distal convoluted tubule (all described below). Many nephrons deliver their contents into each collecting duct, and in turn several collecting ducts join to empty through a duct of Bellini into a renal calix.

By definition, the morphologic unit, the nephron, excludes the collecting duct, for its embryologic origin differs from that of the remainder of the tubule. Until recently, the collecting duct was thought to play a passive role in urine formation; i.e., it was considered to serve only as a urinary conduit. Now it is known that it is involved in the processes of concentrating the urine, regulating acid-base balance, secreting potassium ions and salvaging sodium ions more or less completely from the urine in states of sodium depletion. Thus, because its role is allied with and complementary to that of the remainder of the tubule, the collecting duct should be included as a part of the nephron in a functional definition of the renal unit.

BOWMAN'S CAPSULE. – The nephron has its origin in the expanded blind end of the uriniferous tubule, termed Bowman's capsule, which together with its contained capillary tuft constitutes the renal corpuscle. The outer, fibrous layer of Bowman's capsule is

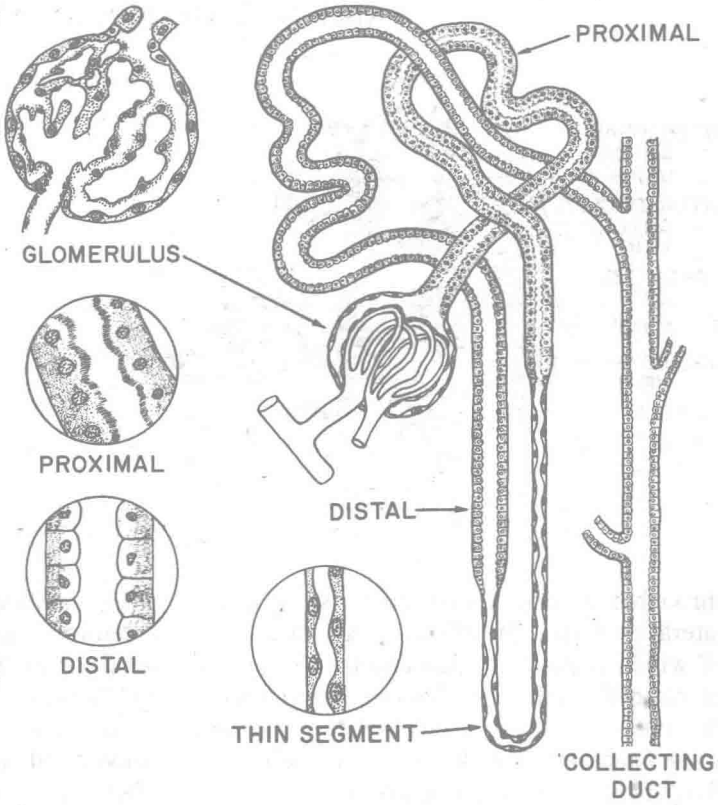

Fig. 1–3. – Relationship of component parts of the nephron. (Adapted from Smith, H. W.: *The Kidney* [New York: Oxford University Press, 1935].)

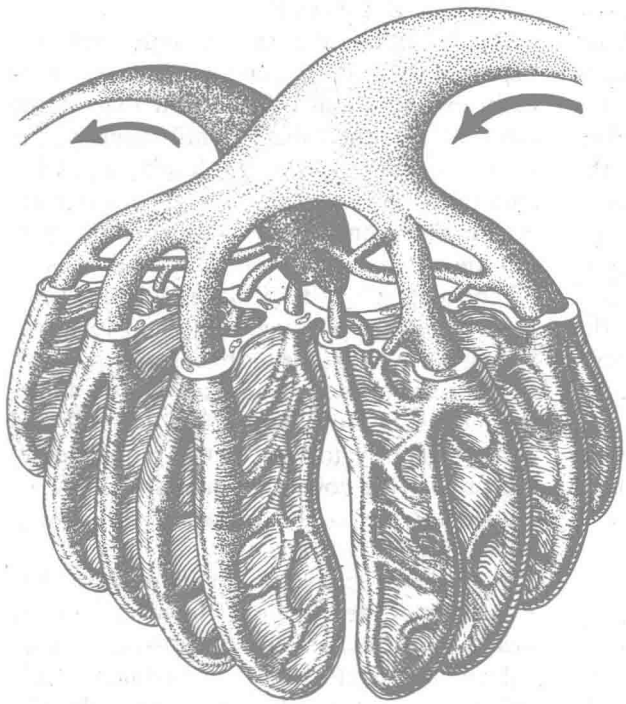

Fig. 1–4. – Anatomy of the glomerulus, illustrating the arrangement of capillary lobules derived from the expanded chamber of the afferent arteriole. The lower section illustrates the foldings of the glomerular basement membrane. (From Elias, H., Hossman, A., Barth, I. B., and Solmar, A. J.: Urology 83:790, 1960.)

continuous with the basement membrane of the tubule. Similarly, the flattened epithelial lining of Bowman's capsule is continuous with the cuboidal epithelium of the tubule. The renal corpuscles of man have an average diameter of 100 μ and, when engorged with blood, are just visible to the naked eye.

GLOMERULUS.—On the side opposite its attachment to the proximal convoluted tubule, Bowman's capsule is invaginated by a lobulated tuft of capillaries, the glomerulus* (Fig. 1–4). The basement membrane of the capsule is reflected over the capillary loops to form the much attenuated capillary basement membrane. It is also fused with the adventitia of the afferent arteriole supplying the glomerulus and of the efferent arteriole draining it. The epithelial lining of the capsule, where it is reflected over the capillary loops, forms the highly specialized podocyte layer of the capillary wall (see p. 55). The glomerulus nearly fills Bowman's capsule. Only small clefts remain, through which the filtrate expressed through the capillary walls flows into the tubular lumen (2).

As the afferent arteriole enters Bowman's capsule, it expands into a relatively wide chamber, which branches into 5–8 trunks. Each of these trunks subdivides, the subdivisions of each trunk constituting a separate glomerular lobule. In all, some 20–40 capillary loops are grouped into 5–8 or more lobules. Numerous anastomoses join the loops within a given lobule. The loops successively recombine to form the emergent efferent arteriole. The afferent and efferent arterioles, by virtue of the contractility of the smooth muscle elements encircling their lumens, constitute variable resistances to the flow of blood through the glomerular tuft and through the postglomerular peritubular capillaries. The cellular elements of the media and adventitia of the afferent arteriole become more numerous and modified in form and in staining properties as the vessel enters Bowman's capsule. These juxtaglomerular cells form a thickened cuff around the afferent arteriole known as the polar cuff or polkissen (Ger. *Polkissen* = pole cushion). A specialized segment of the distal tubule of that same nephron, known as the macula densa, is closely applied to the polkissen. Together, these two structures are termed the juxtaglomerular apparatus. The granular cells of the polkissen probably play an endocrine role, in that they appear to be the source of renin, an enzyme that splits an α_2 globulin of plasma to produce a powerful blood-borne vasoconstrictor. According to some, the macula densa is a sensing organ that responds to the sodium concentration of the urine in the distal tubule and controls the output of renin from its associated polar cuff (see section, Autoregulation of Renal Circulation, p. 167).

PROXIMAL TUBULE.—The proximal tubule is joined to Bowman's capsule by a short connecting segment. The tubule coils extensively in the neighborhood of the parent renal corpuscle, as the pars convoluta, and then enters a cortical medulla ray to penetrate the deeper layers of the cortex and outer medulla to a variable depth, as the pars recta. Throughout its length, the proximal tubule is composed of a single layer of cuboidal or truncated pyramidal cells resting on an enveloping basement membrane. The basement membrane is, no doubt, largely a supporting structure, although it is necessary for the proper organization of regenerating tubular cells following chemical or ischemic damage. This membrane must be permeable to water and solutes, for they traverse it in the course of reabsorption from tubular lumen into peritubular capillaries.

The proximal tubular cells are coarsely granular; their nuclei are large and basally located. The apical surfaces of the cells bulge into the tubular lumen, nearly obliterating its cavity and giving it an irregular contour. These surfaces are covered with numerous cytoplasmic filaments about 1 μ in length, which form the nonmotile brush border. The basal aspects of the cells exhibit a striated

*The term glomerulus is often used interchangeably with "renal corpuscle" to include the capillary tuft and Bowman's capsule. Properly, it refers only to the capillary tuft.

appearance, owing to the linear disposition of the mitochondria in narrow channels between multiple infoldings of the cell membrane. Adjacent cells are held together by basal trabecular projections, which interlock, and by terminal bars, which seal the chinks between adjacent luminal surfaces. The brush border, the coarse granularity of the cytoplasm and the basal striations, although present throughout the proximal segment, are less evident in the pars recta than in the pars convoluta.

HENLE'S LOOP.—The loop of Henle includes the descending thick limb, already described as the pars recta of the proximal tubule; the descending and ascending thin limbs; and the ascending thick limb, to be described below as a part of the pars recta of the distal tubule. The extent of loop development varies with the position of the renal corpuscle in the cortex. The nephrons that have renal corpuscles lying in the outer two thirds of the cortex (cortical nephrons) have relatively short loops. Some of these nephrons lie entirely within the cortex and have no thin segments at all. Others have loops that penetrate the medulla to varying depths; these generally have short thin segments. The nephrons that have renal corpuscles lying in the inner third of the cortex (juxtamedullary nephrons) have relatively long loops. The thin segments of some of these loops extend to the tips of the papillae. All intergrades of loop length are to be found in the human kidney, although cortical nephrons are much more numerous than juxtamedullary nephrons (ratio approximately 7:1).

In various species of mammals, the numbers of nephrons with long loops and the lengths of loop in proportion to total nephron length correlate well with capacity to form concentrated urine. Certain desert rodents that complete their life span without water, other than that present in relatively dry seeds and derived from the metabolism of fat, protein and carbohydrate, have only nephrons with loops that are very long in proportion to nephron length.

The hairpin configuration of the loop of Henle (i.e., descending and ascending limbs lying in close approximation) was once considered to be solely of morphogenic significance. The two ends of the tubule are fixed early in development—one end by its vascular attachment at the glomerulus, the other by its junction with the collecting duct. Growth at the two ends gives rise to the proximal and distal convolutions. Growth in the middle, along the axis of the pyramid, forms the hairpin loop of Henle. Although a morphogenic significance is not denied, a functional significance related to the concentration of urine is now accorded the hairpin configuration and the resultant countercurrent flow of fluid—toward the papilla in the descending limb, away from the papilla in the ascending limb (see pp. 124–135).

The thin segment of the loop of Henle arises abruptly from the descending thick segment of the loop, the pars recta of the proximal tubule. The diameter of its lumen is less than that of either the proximal or the distal segment. The cells are flattened and thin except in the nuclear region, which bulges into the lumen. The cytoplasm is clear, with only a few scattered mitochondria and granules. The cell borders are serrated and interdigitated, meshing together as the cogs of gear wheels. The thin segment may be confined to the descending limb of Henle's loop or may form the bend of the loop and continue for a variable distance up the ascending limb. As noted above, some nephrons of the human kidney have no thin segments at all.

DISTAL TUBULE.—The distal tubule arises abruptly from the thin segment as the thick ascending limb of Henle's loop. It continues in a nearly straight course to the region of the parent renal corpuscle, in the neighborhood of which it convolutes. The cells of the pars recta are cuboidal; those of the cortical convolutions are more columnar. In the region where the distal tubule makes contact with the afferent arteriole of the parent renal corpuscle, the cells are high, columnar and densely packed, forming a plaque called the macula densa. The cells of the distal tubule

lack a brush border, but they exhibit basal striation similar to that of the proximal tubular cells and due, as in the case of the proximal cells, to the accumulation of mitochondria in channels created by the infolding of the cell membrane. The distal tubule is shorter than the proximal tubule, and its convolutions are less complex. The distal tubule connects with a collecting duct through an arcade or transition segment made up of dark granular cells, presumably of distal tubular origin, intermixed with clear duct cells.

COLLECTING DUCT.—The collecting duct, formed in the outer cortex by the junction of two or more transition segments of distal tubules, receives additional contributions in its course through the outer and inner cortex. In the medulla, several collecting ducts fuse to form one of the many papillary ducts that drain into a minor calix. The cells making up the collecting ducts are cuboidal, sparsely granular and regular in size. The nuclei are round and uniformly placed within the cells. Cell boundaries are definite.

DIMENSIONS OF RENAL TUBULES IN MAN.—The proximal segments are 12–24 mm long; the thin limbs of Henle's loops, 0–14 mm; the ascending thick limbs of Henle's loops, 6–18 mm; and the distal convoluted segments, 2–9 mm. The total lengths of nephrons, excluding the collecting ducts, are 20–44 mm. The average length of the collecting ducts is 22 mm.

The external diameters of the proximal tubules are 50–65 μ; of the thin limbs of Henle's loops, 14–22 μ; of the distal tubules, 20–50 μ; and of the collecting ducts, as much as 200 μ in their terminal portions, the ducts of Bellini. Proximal tubules make up the bulk of the renal parenchyma (3, 4).

VASCULAR SUPPLY OF TUBULES.—The vascular supply of the tubules is essentially portal, the blood that perfuses the peritubular capillaries having initially traversed the glomerular capillaries. However, branches of afferent arterioles of cortical glomeruli feeding directly into the cortical capillary net are occasionally observed; and, similarly, direct connections between afferent arterioles of juxtamedullary glomeruli and the vascular loops descending into the medulla and papilla have been described. Nevertheless, any direct arteriolar supply of peritubular capillaries is insignificant in the normal kidney. In aged persons, and especially in patients with chronic renal disease, the number of anastomotic connections between preglomerular arterioles and peritubular capillaries is increased. Many of these connections result from the persistence of a single capillary loop in an otherwise degenerate glomerulus, the loop undergoing transformation into an arteriolar-like structure.

VASCULAR SUPPLY OF CORTICAL NEPHRONS.—The vascular supply of the cortical nephrons is quite different from that of the juxtamedullary nephrons (Fig. 1–5). In the cortical nephrons, the efferent glomerular arteriole, which is slightly smaller than the afferent, breaks up immediately into a rich, freely anastomosing network of capillaries that envelops the convolutions of proximal and distal tubules, and the cortical reaches of the ascending and descending thick limbs of Henle's loops and collecting ducts. The capillary net derived from one glomerulus communicates so freely with that of adjacent nephrons that no distinction is possible. The postglomerular blood is therefore widely disseminated among a group of nephrons. The capillary net recombines into venules that enter the interlobular veins.

VASCULAR SUPPLY OF JUXTAMEDULLARY NEPHRONS.—The juxtamedullary nephrons have a more complex vascular supply than that of the cortical nephrons. In the juxtamedullary nephrons, the diameter of the efferent glomerular arteriole is equal to or larger than that of the afferent arteriole. It gives off one or more side branches, which supply a capillary net enveloping the cortical convolutions of proximal and distal tubules—a net entirely analogous to that of the above-described network of cortical nephrons. The unique feature of the juxtamedullary circulation is the medullary blood supply, derived from the repeated branching of the descending efferent arteriole in the py-

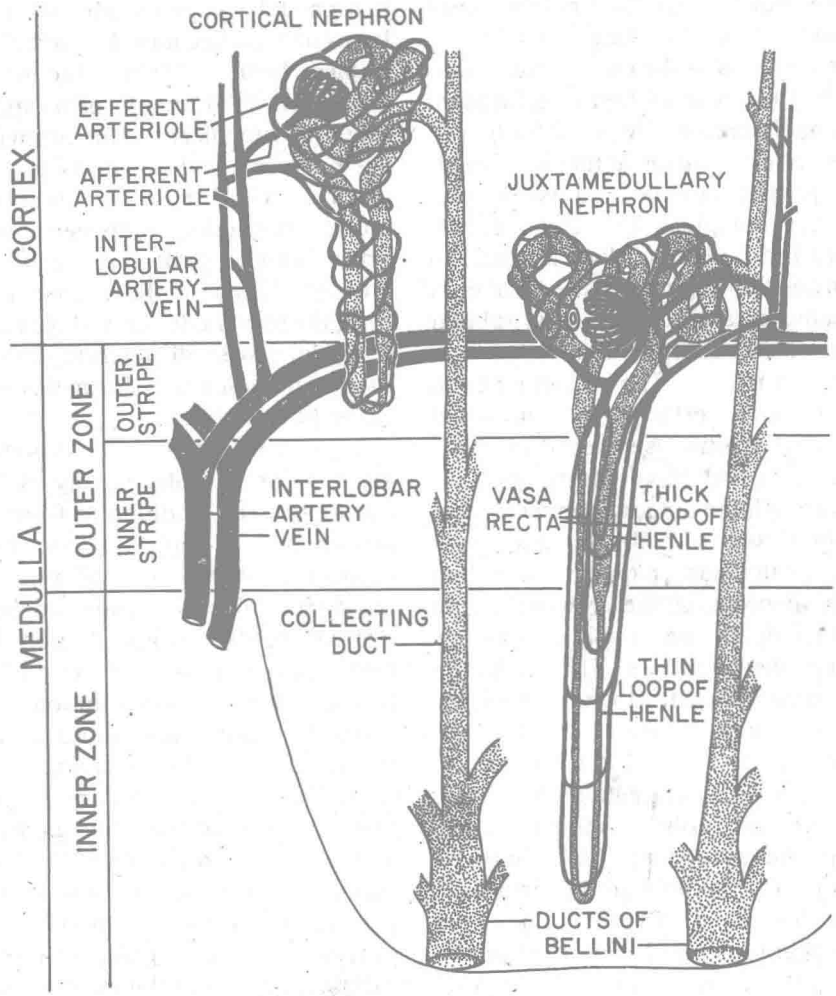

Fig. 1–5. – Comparison of the blood supplies of cortical and juxtamedullary nephrons.

ramid. These vessels are straight-bore tubes that follow the descending limbs of Henle's loops through the medulla and papilla, turn at the bend of the loop and return to reassemble into a venule that enters an interlobular vein close to its junction with an arcuate vein. The arrangement of descending and ascending limbs of these vasa recta (arteriolae rectae) is that of a countercurrent hairpin loop. The function of the medullary vascular loops as countercurrent exchangers is described on pages 130–131.

LYMPHATIC NETWORK. – A rich lymphatic network drains the cortex; no such network is found in the medulla and papilla. Two basic plexuses exist. One, subcapsular, drains the outer cortex and freely anastomoses with a perinephric system in the fat and areolar tissue surrounding the kidney. The other drains the deeper cortex through a series of channels, which follow the interlobular, arcuate and interlobar vessels and leave the kidney at the hilus. Both systems drain into lateral aortic nodes. Renal lymph from the subcapsular plexus has been studied; that from the hilar lymphatics has received less attention. The role of the lymphatic system in renal function is largely undetermined, but it is probably of minor significance.

NERVE SUPPLY. – The kidney is richly

此为试读,需要完整PDF请访问: www.ertongbook.com