Tubular salt and water transport in hydrated dogs studied with push-flow technique

A new technique for studying distal tubular transport processes in water diuresis in dogs is presented: Pelvic urine is sampled continuously in small portions during sudden increase of urine flow by rapid intravenous infusion of hypertonic mannitol. Marked and consistent changes in urine composition are observed more than one minute before mannitol appears in urine: Inulin concentration falls sharply in the first samples, indicating reduction of fractional water reabsorption due to shortened exposure time in the distal nephron. In later samples sodium concentration rises steeply, whereas inulin concentration remains steady, indicating reduction of fractional sodium reabsorption from a relatively water impermeable segment. It is concluded that considerable amounts of water, up to 20% of GFR, may be re-absorbed without sodium from distal nephron segments (collecting ducts and distal convolutions) also in the absence of ADH.

The low sodium concentration of the renal medulla in water diuresis as compared to hydropenia is probably explained by the diluting effect of water reabsorption from the collecting ducts. Studies on hypertonic diuresis revealed even higher distal water permeability, to an extent which makes the method unsuited to study tubular water transport in the presence of ADH.

Author’s address: Dr. K. Aukland and Dr. J. Kjekshus, Institute for Experimental Medical Research, University of Oslo, Ullevaal Hospital, Oslo (Norway).

Renal tubular reabsorption of bromide compared with chloride

A theoretical kinetic analysis of renal tubular reabsorptive processes suggested that two similar solutes passively reabsorbed throughout the same portions of the nephron should exhibit a power function relationship between their excretion fractions. Simultaneous clearances of radiobromide and chloride were measured in dogs to test the applicability of this relationship to these halides.

Bromide and chloride clearances were varied over a wide range by salt depletion and by infusion of various solutes. The relationship: excreted/filtered bromide = (excreted/filtered chloride)k was found adequate to describe the results, using values for k in each of 19 experiments varying from 1.02 to 1.19. A computer program was used to analyse the results statistically; k was unaffected by filtration rate, plasma chloride concentration, or infusions of thiocyanate, nitrate, perchlorate, bicarbonate, or hydrochloric acid; k varied somewhat with flow, plasma bromide or iodide concentration, and infusion of sulphate.

Extremely low bromide and chloride clearances were observed, indicating that active reabsorption of the last traces of both ions in the tubular fluid can occur.

Author’s address: Dr. M. Walser, Department of Pharmacology, Johns Hopkins University, Baltimore, Md. (USA).
Proximal tubule micropuncture studies were carried out in hydropenic and saline-loaded dogs with and without renal artery constriction. Proximal sodium reabsorption was determined by the change in tubule fluid to plasma inulin concentration ratio (TF/Pin), plotting this ratio as a function of distance along the proximal tubule. Renal artery constriction did not cause a significant difference in TF/Pin ratios compared to control ratios in hydropenic dogs at a comparable distance in the proximal tubule. Isotonic saline infusion, however, caused a marked depression of TF/Pin ratio with variable changes in glomerular filtration rates. The saline-induced TF/Pin depression occurred also in the renal artery constriction group. This depression of proximal tubule fractional sodium reabsorption was great enough that it was not necessary to implicate distal segment sodium rejection in order to explain the natriuresis following isotonic volume expansion.

Author’s address: Dr. J. F. Watson, Department of Medicine, State University of New York at Buffalo, Buffalo General Hospital, Buffalo, N.Y. (USA).

Using the ventral skin of the European toad (Bufo bufo) in the Ussing model the influence of the diuretics Furosemide and Ethacrinic acid on the membrane potential and the short-circuit-current of toad skin were investigated. A marked difference in response of the two parameters was observed in regard to the side of the membrane (inside-outside) to which the drug was added. In addition the results indicate that aside from the influence of the two drugs on active sodium transport changes in the permeability of the skin for passively moving ions have to be assumed.

Author’s address: Dr. H.H. Edel, I. Medizinische Universitätsklinik, Ziemsenstraße 1a, 8000 München 15 (Germany).

Intrinsic renal mechanisms for the regulation of body electrolytes in man

Intrinsic renal mechanisms for the regulation of body electrolytes in man


Previous communications have demonstrated sodium balance in normal man is regulated through 2 to 5 day cyclical fluctuations in urinary sodium excretion [J. lab. clin. Med. 55: 362 (1960)]. These periodic changes do not result from fluctuating adrenal steroid secretion [Metabolism 12: 1032 (1963)]. Present studies reveal these cyclical fluctuations are derived from comparable variations in mean daily GFR, both in normal subjects and in an adrenalectomized cortisone-maintained patient. Small transitory increments in the serum concentrations of sodium and the anion of the loaded sodium salt contribute to this filtered load, and participate in the excretion response to loading. During sodium phosphate ingestion, the fluctuations of urinary sodium excretion were significantly correlated with daily phosphate excretion. Comparable fluctuations in urinary sodium and phosphate excretion can be demonstrated in a parathyroprivic patient maintained with vitamin D therapy, indicating that the urinary phosphate fluctuations are not derived from changing parathormone secretion. Unlike sodium, urinary potassium excretion during potassium salt loading is unrelated to mean daily GFR. Daily potassium excretion is also unrelated to daily urine volume. By contrast daily sodium excretion in subjects drinking water ad libitum is significantly correlated
with daily water excretion at all but minimal sodium intakes, a correlation that persists even
during potassium loading.

Authors’ address: Dr. D. Baldwin, Dr. A. F. Nibbe and Dr. T. B. Schwartz, Section of
Endocrinology and Metabolism, Division of Medicine, Presbyterian-St. Luke’s Hospital,
Chicago, Ill. (USA).

Peritoneal sodium transport: enhancement by pharmacologic and physical agents
The mechanism of peritoneal sodium transport was investigated with the use of isolated rabbit
omentum suspended in a lucite diffusion chamber. Sodium movement across the membrane was
evaluated by use of Na22. Temperature, vasopressin, calcium concentrations and dinitrophenol
were used in an attempt to influence sodium transport. Temperature increase (27-37° C),
vaseopressin and omission of Ca++ from the bath all increased the rate of sodium movement
across the membrane. Dinitrophenol on the other hand had no effect. No definite conclusion
could be reached as to active sodium transport which, if present, must be different from that
found in systems like the toad bladder or frog skin. It was concluded, however, that the rabbit
peritoneum is not an inert diffusion barrier and peritoneal sodium transport can be modified by
pharmacological and physical agents.

Author’s address: Dr. K.G. Barry, Department of Metabolism, Division of Medicine, Walter
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Antidiuretic properties of chlorothiazide in diabetes insipidus dogs
The mechanism of the paradoxical antidiuretic action of chlorothiazide in diabetes insipidus has
not been explained. In an attempt to better define the mechanism of the antidiuresis,
chlorothiazide was given to diabetes insipidus dogs, which showed the decrease in urine volume
and increased urine concentration observed in patients. There was no change if GFR, RPF,
TmPAH, plasma volume, extracellular fluid or total body water. The one consistent altered
function was a decrease in ‘free water’ clearance. Diazoxide and mercuhydrin were not
antidiuretic. It is believed that the antidiuretic action is dependent on the chlorothiazide-induced
saluresis. Although the mechanism of this antidiuresis has not been clearly defined, it seems best
explained by postulating a decrease in filtrate reaching the distal nephron (giving a decreased
final urine volume) coupled with inhibition of solute reabsorption in the distal tubule (preventing
the selective reabsorption of solute in the distal tubule that is normally seen, accounting for the
increase in urine concentration). The possible role of the mineralocorticoids in the antidiuretic
response to chlorothiazide therapy was investigated in diabetes insipidus and adrenalectomized
dogs. Administration of sodium-retaining steroids to diabetes

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insipidus dogs did not reproduce the urinary changes associated with chlorothiazide therapy. In
addition, the antidiuretic response to chlorothiazide was not altered by adrenalectomy or
spironolactone administration. Therefore, the presence of the sodium-retaining steroids are not
essential for the antidiuretic response of chlorothiazide in diabetes insipidus.

Author’s address: Dr. J.Y. Gillenwater, University of Virginia School of Medicine, Charlotte
ville, Va. (USA).

Influence of posture and diurnal rhythm on the renal excretion of acid: observations in normal
and adrenalectomized subjects
The influence of posture and diurnal rhythm on the renal excretion of acid was examined in 4 normal and 2 adrenalectomized subjects. Assumption of the recumbent position in normal subjects resulted in inhibition of acid excretion, the extent of inhibition being influenced by the diurnal rhythm. Postural inhibition of acid excretion was also observed at high levels of acid excretion following methionine loading. In adrenalectomized subjects assumption of the recumbent position resulted in postural natriuresis but not in any decrease in acid excretion. The postural inhibition of acid excretion occurring in normal subjects could not be restored in adrenalectomized subjects by administration of hydrocortisone, methylprednisolone or aldosterone. It is suggested that (1) postural inhibition of acid excretion is dependent on adrenal function but not directly on the presence of glucocorticoid hormone or aldosterone, (2) that the mechanism of postural and diurnal variations in acid excretion may share a common factor.

Author’s address: Dr. P.R. Steinmetz, Harvard Medical School, Beth Israel Hospital, 330 Brookline Avenue, Boston, Mass. 02115 (USA).

The effects of combined renal vasodilatation and pressor agents on renal hemodynamics and the tubular reabsorption of sodium
Unilateral renal arterial infusions of vasodilators and systemic or renal arterial infusions of pressor agents were carried out in 21 anesthetized dehydrated dogs. Renal hemodynamic and electrolyte excretion measurements were made. Renal vasodilatation induced by either acetylcholine bromide, kallidin or bradykinin was accompanied by increases in sodium excretion associated with increased total and non-cortical renal plasma flow, the latter estimated by decreases in P.A.H. extraction ratios. The superimposition of pressor infusions of either angiotensin or norepinephrine resulted in further rises in sodium excretion despite decreased total and non-cortical plasma flow. It is suggested that these pressor agents may cause natriuresis indirectly via increments in renal perfusion pressure. It is postulated that there may be a pressure-sensitive portion of the renal vasculature that indirectly influences sodium reabsorption. If so, the renal vasodilating agents employed may have permitted a more complete transmission of the existing perfusion pressure to the capillary circulation resulting in increased cortical interstitial volume. This increase in interstitial volume could result in a decreased sodium reabsorption by producing some degree of tubular collapse.

Author’s address: Dr. L.E. Earley, Boston City Hospital, 818 Harrison Avenue, Boston, Mass. (USA).

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Renal ammonia excretion during acetazolamide or sodium bicarbonate administration
Proximal and distal micropuncture studies on ammonia excretion were carried out in a group of 49 rats. Thirty-nine animals were evaluated during administration of acetazolamide and 11 rats were studied while receiving an infusion of sodium bicarbonate. The results obtained suggest that the proximal tubule is a quantitatively important source of urine ammonia and that large amounts of ammonia added to proximal fluid may be reabsorbed within the loop of Henle. This reabsorbed ammonia could then be added to either collecting duct fluid or vasa recta blood. Calculated estimates of PNt in tubular fluid suggest that diffusion equilibrium exists for molecular ammonia throughout the renal cortex.

Author’s address: Dr. R. R. Robinson, Department of Medicine, Duke University Medical Center, Durham, N.C. (USA).
The mechanism of action of ethacrinic acid. II. The influence of ethacrinic acid on the distal nephron in man. Der Einfluß der Etacrynsäure auf das distale Nephron des Menschen


In five healthy individuals, one patient with decreased renal function and one patient with hereditary nephrogenic diabetes insipidus the influence of ethacrinic acid (50 mg intravenously) on T\textsubscript{\(\text{\textcircled{H}}\)} under conditions of osmotic diuresis (constant infusion of 10% Mannitol and 5-10 mU/h ADH) was investigated: in all cases the value of 7\textsubscript{\(\text{\textcircled{H}}\)} became negative indicating a hypotonic diuresis increment. These results suggest that ethacrinic acid causes a water impermeability at the distal part of the nephron in addition to its well known natriuretic effect. In the patient with nephrogenic diabetes insipidus however, the characteristic hypotonic diuresis increment disappeared, the diuresis increment was isotonic and the final urine remained hypotonic.

Author’s address: Dr. H.H. Edel, Medizinische Universitätsklinik, Ziemssenstraße 1a, 8000 München 15 (Germany).

The renal response to acid loads in metabolic alkalosis; an assessment of the mechanisms regulating acid excretion


Seventeen balance studies were carried out on 13 dogs given a chloride-free, low electrolyte, synthetic diet supplemented with neutral phosphate salts of sodium and potassium. Metabolic alkalosis was then induced by gastric drainage with plasma bicarbonate levels rising to 30 to 35 mEq/L. Acid loads were then fed, either as hydrochloric acid (the chloride ion being readily reabsorbable) or nitric acid (nitrate being poorly reabsorbable). The administration of hydrochloric acid led to a marked retention of sodium and potassium accompanied by an equivalent loss of acid, urinary acidification and increased acid excretion occurring even when plasma bicarbonate concentration and pH were still at frankly alkaline levels.

In companion studies in which retention of sodium and potassium was prevented by removing these cations from the diet before hydrochloric acid was administered, renal acid excretion did not increase, and alkalosis was fully corrected by the administration of a far smaller quantity of acid.

When nitric acid was administered to alkalotic animals, acid excretion also increased, not in conjunction with cation retention, but rather in parallel with nitrate excretion.

It would thus appear that in these studies acid excretion occurred in response to the need to preserve electroneutrality during cation conservation or anion loss; cellular acidity would seem to be important only to the extent that it influences the amount of hydrogen which will be contributed when there is a demand for cation at the exchange site.

Author’s address: Dr. W.B. Schwartz, New England Center Hospital, 1171, Harrison Avenue, Boston, Mass. 02111 (USA).

The significance of Na+-transport, -concentration and direction of supply for renal tubular transport of glucose and PAH.


The tubular transport of glucose by isolated kidneys of Rana ridibunda is Na+-dependent in this way: that the more Na+ is transported the more glucose is transported and vice versa. Only Na+ on the lumen side is essential for the transport of glucose. The Na+-transport is lower when it is
given as $\text{SO}_4^{2-}$ than as $\text{Cl}^-$ because of the relative impermeability of the tubule cell for the $\text{SO}_4^{2-}$ anion and the diminished Na$^+$ transport.

Also more PAH is transported (secretion) when much Na$^+$ is offered and vice versa. The best PAH-transport was observed when Na$^+$ was given from the lumen- and the bloodside. Na$^+$, offered only from the bloodside, decreases the transport of PAH but not as much as it was seen for the decrease of the glucose-transport.

Under the conditions of Na$^+$ supply only from the bloodside the Na$^+$ content of the kidneys was considerably diminished. The kidneys probably lose intra-cellular Na$^+$ with the consequence of missing activation of the membrane-ATPase. Na$^+$ may be linked with transport of substances by intracellular Na$^+$ concentration as an essential parameter of Na$^+$.

Authors’ address: Prof. Dr. G. Vogel, Biologisches Institut Madaus, KBln (Germany).

Renal gluconeogenesis in acidosis, alkalosis and potassium deficiency: Its possible role in regulation of renal ammonia production


Renal cortical slices from rats with metabolic acidosis induced by ammonium chloride were found to have an increased capacity to produce glucose from glut-amine, glutamate, and $\alpha$-ketoglutarate, whereas cortical slices from rats with metabolic alkalosis induced by sodium bicarbonate were noted to have a decreased capacity to produce glucose from these substrates. Potassium depletion induced by dietary restriction also was associated with increased gluconeogenesis from these substrates. Both metabolic acidosis and potassium deficiency are commonly associated with increased urine ammonium excretion. It is postulated that both disturbances cause increased conversion of glutamate and $\alpha$-ketoglutarate to glucose, and that this leads to a decrease in the intracellular concentration of these substrates resulting in accelerated synthesis or activation of glutaminase I and glutamine trans-aminase by a negative feedback system. The increased synthesis or activation of these enzymes would then result in increased ammonia production from glutamine.

It is suggested that such a mechanism also may play a role in the diminished glucose tolerance seen in metabolic acidosis and in potassium deficiency.

Author’s address: Dr. A. D. Goodman, Subdepartment of Endocrinology and Metabolism, Department of Medicine, Albany Medical College, Albany, N.Y. (USA).

The significance of the ionic environment for renal tubular transport of glucose and of glucose supply for the transport of Na$^+$/ Untersuchungen zur Abhängigkeit des renal tubulären Glucose-Transportes vom Ionen-Angebot sowie des Na$^+$-Transportes vom Angebot an Glucose


The transports of Na$^+$, Ca$^{2+}$ and glucose of isolated kidneys of Rana ridibunda are significantly decreased during perfusion without K$^+$. The K$^+$-deficiency seems to inhibit the activity of the Na$^+$-K$^{2+}$-dependent membraneATPase.

Na$^+$-transport as well as glucose-transport are dose-dependent inhibited by 12,0 and 1200,0 mg/l Furosemide. The inhibition of Na$^+$-transport is more pronounced than the inhibition of glucose-transport. Mersalyl 1000 mg/l also inhibited both Na$^+$ and glucose-transport, but the inhibition of glucose-transport is more pronounced. Because extracellular Na$^+$-concentration is not altered whereas Na$^+$-transport is diminished the results indicate the Na$^+$-transport to be the essential parameter for the connection of sodium supply and glucose-transport.

Phloridzin only inhibited the glucose-transport, the reabsorption of Na$^+$ was not influenced.
The partially blocked Na+-transport (Furosemide 10 mg/l) cannot be stimulated by the addition of 0.833 and 8.33 mM/1 glucose. This demonstrates together with the results of the Phloridzin experiments, that the glucose-transport is Na+-dependent but not vice versa, that means the Na+-transport is not dependent on glucose.

Author’s address: Prof. Dr. G. Vogel, Biologisches Institut Madaus, Köln (Germany).

The role of aldosterone and vasopressin in the postural changes in renal excretion in normal subjects and patients with idiopathic edema


The effects of the upright posture on urinary excretion were studied in 13 normal subjects, 13 patients with idiopathic edema, 5 patients with untreated diabetes insipidus (D.I.) and 6 patients with cortisol-treated adrenal insufficiency, during ingestion of dilute saline every half-hour. Leisurably walking for 2 h induced immediate and sustained oliguria and fall in free water clearance, in all groups except

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patients with D.I. In these patients, and in the other subjects when they were given 25 ml absolute ethanol, antidiuresis was delayed until after the first 30 or 60 min upright, suggesting that vasopressin release caused the immediate but not the delayed orthostatic antidiuresis. Walking reduced sodium excretion slightly (not significantly) in the normal subjects, moderately in patients with adrenal insufficiency—indicating that aldosterone was not required for this response—and severely in patients with idiopathic edema. Spironolactone and dextroamphetamine reduced but did not abolish the orthostatic retention of Na, suggesting that hyper-aldosteronism might contribute to the excessive Na retention in the patients with edema. However, factors other than vasopressin and aldosterone reduced renal function in these patients since ethanol and spironolactone did not completely overcome the orthostatic reduction of water and Na excretion respectively. GFR was well maintained upright when leg movements were encouraged (Creatinine excretion unchanged).

Authors’ address: Dr. D. H. P. Streeten and Dr. P. J. Speller, Department of Medicine, State University of New York, Upstate Medical Center, Syracuse, N.Y. (USA).

Studies of the mechanism through which sodium depletion increases aldosterone biosynthesis in man


Six normal subjects were studied under standard metabolic conditions. It was found that approximately 90% of corticosterone is ACTH dependent and for this reason, the subjects received dexamethasone during salt restriction. Salt restriction (sodium intake less than 15 mEq/day) resulted in rises in both aldosterone and corticosterone secretion (but not in cortisol secretion), whereas physiologic infusions of ACTH caused rises in cortisol and corticosterone secretion but not in aldosterone secretion. It is concluded that increased aldosterone biosynthesis under conditions of salt restriction results from a fairly specific pathway which increases the availability of aldosterone precursors.

Author’s address: Dr. G. W. Liddle, Department of Medicine, Vanderbilt University School of Medicine, Nashville, Term. 37203 (USA).

Relation of plasma renin to sodium balance and arterial pressure in experimental renal hypertension

The relationship of peripheral plasma renin activity and sodium balance was evaluated in a large number of dogs in which hypertension was produced by renal constriction following unilateral nephrectomy. All animals were on constant intake of salt and complete urine and fecal collections were made.

In a series of 18 dogs subjected to renal arterial constriction, 11 developed hypertension. Of these 11, 5 developed malignant hypertension with the remaining 6 exhibiting benign hypertension. In those with malignant hypertension, plasma renin levels were markedly elevated in most cases whereas in those with benign (chronic) hypertension only an initial transient elevation of renin was seen.

Sixteen dogs with thoracic caval constriction also underwent renal arterial constriction with 4 then developing hypertension. Two of these 4 hypertensive animals had malignant hypertension and increases in plasma renin levels were noted. In one of the two animals developing benign hypertension, no further rise in the already elevated plasma renin level was observed. Marked sodium retention was present in all animals in this group. Another series of 6 dogs underwent sodium depletion (dietary restriction plus diuretics) followed by renal arterial constriction. Two of these animals developed chronic hypertension despite no further rise in the already elevated plasma renin levels. An additional 5 dogs with chronic hypertension were subjected to sodium depletion with the result that plasma renin levels increased but arterial pressure measurements remained unchanged.

It was concluded that in chronic hypertension there exists an excellent correlation between plasma renin activity and sodium balance, but neither plasma renin levels nor sodium balance bore a discernible relation to arterial blood pressure.

Author’s address: Dr. J.O. Davis, Section on Experimental Cardiovascular Disease, Laboratory of Kidney and Electrolyte Metabolism, National Heart Institute, U.S. Public Health Service, Bethesda, Md, (USA).

Studies on the binding of aldosterone in the toad bladder


The binding of aldosterone in toad bladder was studied by a technique which distinguished that hormone which was adsorbed or bound in the tissue from that in a free or soluble state. Physiological activity was associated with only the former which comprised only 33 to 2% of the hormone accumulated by the tissue when exposed to physiologically active concentrations of aldosterone, \(3 \times 10^{-10}\) to \(10^{-7}\) M. The bound or adsorbed hormone was resolvable into two sets: one with high affinity, \(K_{assoc} = 1.4 \times 10^{14}\) and a maximum binding capacity of some \(9 \times 10^{-14}\) moles of aldosterone per g of tissue, and another of lower affinity, \(K_{assoc} = 4 \times 10^{12}\), binding \(3 \times 10^{-12}\) moles of hormone per g of tissue. The bound hormone was displaced readily from both sets of sites by d-aldosterone, desoxycorticosterone, cortisol, progesterone and spironolactone (SC 14266) but not by cholesterol. Because the binding sites of the large set were saturated at concentrations of hormone corresponding to maximum physiological activity, whereas those of the small set were essentially saturated at concentrations too low to elicit a physiological response, it is concluded that the large set contains the receptors responsible for the hormonal effect. The characteristic latent period prior to onset of the hormonal effect is not attributable to a delay in the binding of the hormone with the tissue.

Author’s address: Dr. G. Sharp, Massachusetts’ General Hospital, Boston, Mass. (USA).

The direct renal action of angiotensin in the rabbit
In conscious rabbits, synthetic Val-5 angiotensin, in a dose of 1 µg/kg/min, was infused into an ear vein and into one renal artery through a catheter previously inserted into a branch of the adrenolumbar artery. Urine was collected from each ureter.

Ear vein infusions caused an increase in urine flow, and in the excretion of sodium and chloride. This response was roughly equal on the two sides.

Renal artery infusions caused similar changes in urine flow and composition, but the response was roughly five times greater on the infused side. There was no evidence that these infusions caused a greater change of inulin or PAH clearances by the infused kidney, as compared with the opposite kidney. This indicates a direct effect of angiotensin upon the function of the kidney which may be mediated by direct tubular action or through haemodynamic changes.

Infusion of angiotensin into a renal artery caused a much smaller blood pressure rise than infusion of the same dose into an ear vein.

Pressor effects of angiotensin infusions into different vascular beds in the rabbit

Angiotensin was infused into systemic or mesenteric veins and into femoral, renal, or mesenteric arteries of anaesthetised or conscious rabbits; the dose usually employed was 1 µg/kg/min. The rise in systemic arterial pressure in a steady state was measured.

Infusions into the renal artery, mesenteric artery, or mesenteric veins caused smaller pressor responses than infusions into systemic veins or arteries. Noradrenaline infused into a renal artery caused a pressor response similar to that following intravenous infusion.

The reduced pressor response to renal arterial infusion of angiotensin was unaffected by previous ureteric ligation, and it is probable that the kidney can inactivate large amounts of circulating angiotensin.

The effects of starvation, high fat diets, and ketone infusions on uric acid balance

Studies of factors arising during fasting which could influence uric acid excretion were carried out in 15 patients. Potassium supplementation and alkalinization did not influence the decreased urinary excretion of uric acid found during fasting, whereas probenecid and glucose administration effected a prompt uricosuria. Glomerular filtration rate was not altered by 7 days of fasting, but changes in uric balance appeared to be related to changes in ketone concentrations. The effect of ketosis in the absence of fasting was studied by feeding high fat diets in 6 subjects and ketone infusions in 5 others. In both groups, uric acid clearance was depressed. It is postulated that ketones compete with uric acid for a common tubular secretory site, the magnitude of the changes in ketones precluding normal uric acid secretion.

Renal Angiography
L’angiographie rénale est devenu un procédé d’exploration irremplaçable en néphrologie. Ce livre est un exposé clair et systématique, par une équipe très expérimentée, de la méthode, des différentes techniques et des résultats dans les diverses affections rénales, médicales ou chirurgicales.

A juste titre, le principal effort est consacré à l’hypertension artérielle vasculorénale, la plus belle acquisition actuelle de l’artériographie. Les autres chapitres sont traités avec autant de soin que le précédent. C’est ainsi que sont données de parfaites images accompagnées de commentaires précis des malformations rénales, des tumeurs et des kystes, des hydronéphroses, des diverses néphropathies et des contusions rénales.

Le seul regret que l’on puisse exprimer est un certain dédain de la phlébographie, particulièrement de la phlébographie occlusive qui, en Europe, est de plus en plus répandue. Il est vrai que la bibliographie autre que de langue anglaise ne semble pas avoir été très explorée.

G. Richet
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