Further Section

Nephron 1969;6:149-160

Summaries–Résumés

Major surgery in patients on maintenance hemodialysis
The hospital records of 33 patients with chronic renal failure undergoing 44 major operations and requiring pre- and postoperative hemodialysis were reviewed. The patients were evaluated as to preoperative preparation, intraoperative course, and the development of postoperative complications.
Dialysis was performed within 24 h prior to surgery in 30 instances. Most patients were hypertensive. Mean preoperative laboratory values included: blood urea nitrogen of 54 mg/100 ml, creatinine of 9.0 mg/100 ml, potassium of 4.8 mEq/1, and carbon dioxide of 24 mM/1. Hematocrit averaged 28%.
Preoperative anesthetic medications consisted for the most part of short acting barbiturate and atropine. Halothane was the general anesthetic most commonly used in conjunction with nitrous oxide.
Postoperative fever was present in 26 patients, atelectasis in 20, wound infection in six, bleeding in four, and pericarditis appeared after surgery in 11. In six patients, thrombosis of the arteriovenous cannula developed within one week of operation. All postoperative complications were minor and most patients demonstrated excellent wound healing. Twenty-three patients had dialysis within 24 h of surgery. The most common indication for dialysis was a rapidly rising serum potassium level.
From the authors experience, they recommend that the following conditions prevail in patients on dialysis who are to undergo major surgery: (1) dialytic treatment should be carried out the day prior to surgery; (2) there should be adequate control of hypertension, either by ultrafiltration or antihypertensive medication; (3) serum potassium concentrations should be below 5.5 mEq/1 on the day of surgery.
With careful attention to fluid balance and awareness of the propensity of these patients to the development of electrolyte-induced cardiac abnormalities, and institution of early post-surgical dialysis, the outlook for recovery from operation in the patient on maintenance dialysis would appear to be excellent.
Author’s address: Dr. C. L. Hampers, Peter Bent Brigham Hospital, Boston, Mass. 02115 (USA).
Effects of renal impairment, peritoneal dialysis and hemodialysis on serum sodium colistimethate levels
A single 75 mg dose of sodium colistimethate was administered intramuscularly to 39 subjects with varying degrees of renal function; 17 of these subjects were undergoing regular peritoneal dialysis or hemodialysis therapy. Sequential concentrations of the drug in blood and dialysate were determined. When the endogenous creatinine clearance was below 20 ml/min, sodium colistimethate blood levels and their duration of elevation were inversely proportional to the degree of renal impairment.
Approximately 1 mg of sodium colistimethate was removed from the blood per hour during peritoneal dialysis, and the drug was cleared 30% as effectively as urea. Detectable quantities of sodium colistimethate were not removed from the blood by Kiil or Kolff hemodialysis.

The authors recommend the following dosages of sodium colistimethate in patients with renal insufficiency: (1) when endogenous creatinine clearance exceeds 20 ml/min, 75 to 100% of the recommended daily dose, divided into doses every 12 h; (2) when endogenous creatinine clearance is between five and 20 ml/min, 50% of the usual daily dose, divided into doses every 12 h; and (3) when endogenous creatinine clearance is less than 5 ml/min, 30 to 35% of the normally recommended dose divided into doses every 12 to 18 h.

Author’s address: Dr. Norm a J. Goodwin, Downstate Medical Center, 450 Clark-son Avenue, Brooklyn, N.Y. 11203 (USA).

Platelet factor 3 in normal subjects and patients with renal failure

Two tests were used to differentiate abnormalities in release of platelet factor 3 (PF3) from quantitative deficiencies of this factor in normal subjects and in patients with renal failure. The first test was an assay which determined availability of PF3 (PF3-A time) and involved the use of a mixture of patient’s platelet rich plasma (PRP) and normal platelet poor plasma (PPP) in a fixed ratio (1:8). The second test was similar but used ‘frozen and thawed’ platelets to obtain a quantitative estimate of PF3 (PF3-F time). Abnormal PF3-A time was found in approximately three-quarters of 55 patients with renal insufficiency; 43 of those had chronic and 12 had acute renal failure. This abnormality was present both in patients with and without hemorrhagic manifestations, although it was slightly more common in bleeders. The PF3-F test was abnormal in approximately 1/3 of the bleeding patients and ⅛ of the non-bleeders. The PF3-A time returned to normal or was significantly shortened 24-48 h after peritoneal or hemodialysis. Studies on patients who were not dialysed, showed no statistically significant correlation between the PF3-A time and either the serum urea nitrogen, creatinine, calcium, or phosphorous. Furthermore, the PF3-A time was not effected by guanidinosuccinic or guanidinoacetic acid. It was, therefore, concluded that the demonstrable platelet abnormality in patients with uremia is produced by an unknown dialysable material.

The mean plasma clot retraction of uremic patients was also significantly different from the mean plasma clot retraction of normal controls. However, unlike the PF3-A time, the abnormality of plasma clot retraction was not immediately effected by dialysis. Correction of the plasma clot retraction did occur after repeated dialyses.

Author’s address: Dr. S. Frederick Rabiner, Department of Medicine, Michael Reese Hospital, Chicago, Ill. 60616 (USA).

Shunt de l’artère fémorale profonde. Une contribution au problème des shunts j Der Femoralis-profund-Shunt. Ein Beitrag zum Shunt-Problem

Le résultat de la dialyse répétée de malades en insuffisance rénale chronique depend dans une large mesure du shunt artério-veineux. L’auteur propose d’im-
planter le shunt entre l’artère et la veine circonflexe externe. Dans la technique proposée il n’est pas fait usage d’embout vasculaire en teflon mais le tube en silastic est directement introduit jusqu’au niveau de l’abouchement de l’artère circonflexe externe dans l’artère fémorale profonde. Cette technique présente comme avantage un moindre risque de thrombose et un meilleur débit de sang lors de la dialyse. De plus le shunt est entièrement cache par les vêtements et sa localisation à la face externe de la cuisse l’expose moins à des traumatismes accidentels.
P.S. On attend avec intérêt de savoir si l’expérience confirmera la première impression favorable de l’auteur (N.D.L.R.).
Adresse de l’auteur: Dr. J. Hoeltzenbein, Chirurgische Abteilung, St.Franzis-kus-Hospital, MünsteriWestf. (Allemagne).
Inhibition of renin release by vasopressin and angiotensin
Vasopressin and angiotensin inhibited release of renin induced by lowered renal perfusion pressure. This action could not be related to a measurable haemodynamic change produced by either peptide; in some experiments with vasopressin there was no change in either systemic pressure or renal blood flow. The angiotensin analogues, angiotensin diamide and alanine-7-angiotensin II, had no effects on arterial pressure, renal blood flow, or renin release; neither did mixtures of all of the amino acids present in the angiotensin molecule. Oxytocin did not affect renin release when infused in the same doses found to be effective for vasopressin.
Authors’ address: Dr. R.D. Bunag, Dr. I.H. Page and Dr. J.W. McCubbin, Research Division, Cleveland Clinic Foundation, Cleveland, Ohio (USA).
The progressive pressor response to angiotensin in the rabbit
(1) The threshold for any detectable rise of systemic arterial pressure during the prolonged intravenous administration of angiotensin to conscious rabbits was observed to be an infusion rate of 0.003-0.005 µg kg-1 min-1.
At infusion rates between threshold and 0.04 µg kg-1 min-1 the systemic arterial pressure rose progressively over a 3- to 7-day period to a plateau, as was first reported by Dickinson and Lawrence (Lancet i: 1354-1356 [1963]). On stopping the angiotensin infusion the blood pressure fell rapidly back to its base line much faster than it rose during the infusion. The time taken to reach control values was approximately related to the duration of the infusion. At infusion rates of about 0.05 µg kg-1 min-1 the full rise of blood pressure developed within a few minutes, and could be sustained without change for many days. At higher rates the blood pressure diminished with time. Diurnal fluctuations of blood pressure were often seen during prolonged infusions of angiotensin at low rates: and more rapid fluctuations of blood pressure over an hour or two were frequently encountered immediately after an infusion was turned off.
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(6) The possible role of angiotensin in producing chronic renal hypertension is discussed in the light of these observations.
Authors’ address: Dr. C.J. Dickinson and Dr. R. Yu, Medical Unit, University College Hospital Medical School, University Street, London, W.C. 1 (England).
Effects of angiotensin and noradrenalin on flow and composition of the renal lymph
In the anaesthetized dog, intravenously infused angiotensin significantly reduced the urine flow and the amount of Na and K excreted with the urine and left flow and composition of the renal lymph unaffected. Intravenous infusion of Nor-adrenaline® exerted no effect on renal lymph flow.

Author’s address: Dr. M. Papp, P.O.B. 67, Budapest 9 (Hungary).

The effect of angiotensin II infusion, renal hypertension and nephrectomy on salt appetite of sodium-deficient sheep

In sodium-replete and sodium-deplete rats, hypertension affects salt intake. Aldo-sterone secretion rises in sodium deficiency, and experiments have demonstrated that renin and/or angiotensin levels also increase in sodium deficiency. In sheep, sodium deficiency can be studied by causing loss of saliva through a parotid fistula. If the renin-angiotensin system contributed to the activation of sodium appetite in sodium deficiency, it is possible that the superimposition of renal hypertension, which could acutely augment renin levels, might augment appetite additional to that attributable to sodium deficiency caused by saliva loss from a parotid fistula. In sheep made sodium-deficient by this method, intravenous infusion of pressor doses of angiotensin II (60-120 µg/h) caused a small, but significant reduction in salt appetite. In three hypertensive sheep, angiotensin II infusion did not significantly change sodium appetite. The induction of renal hypertension by partial occlusion of one renal artery did not modify salt appetite in response to sodium deficiency. Nephrectomy did not eliminate salt appetite in three of six sheep studied. The results do not indicate that the renin-angiotensin system directly stimulates salt appetite.

Author’s address: Dr. S. Weller, Howard Florey Laboratories of Experimental Physiology, University of Melbourne, Parkville 3052 (Australia).

Changes of plasma renin concentration during pressor infusions of renin in the conscious dog: the influence of dietary sodium intake

Intravenous infusion of renin in conscious dogs raised blood pressure and increased plasma renin concentration. Changes of blood pressure during and after infusion

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of renin were related to changes of plasma renin concentration. A ten-fold reduction of dietary sodium intake led to an increase of plasma renin concentration, a decrease in the pressor response of infused renin and angiotensin, and an increase in the level of plasma renin concentration during standard pressor infusions of renin.

These effects of exogenous renin suggest that the amount of endogenous renin normally present in blood influences the level of arterial pressure and that changes of sodium balance influence the pressor effect of endogenous renin.

Author’s address: Dr. A.F. Lever, MRC, Blood Pressure Research Unit, Western Infirmary, Glasgow, W. 1 (Scotland).

Metabolism of aldosterone in patients with renal artery stenosis/Aldosteron-stoffwechsel bei Nierenarterienstenose
Renal Excretion (E), Secretion Rate (SR), Metabolic Clearance Rate (MCR) and Mean Plasma Concentration of Aldosterone (MPC = SR/MCR) were determined by double isotope derivative assay in eight patients with renovascular hypertension due to severe renal artery stenosis. Mean plasma concentration was found to be elevated (1) either by increase of Aldosterone-Secretion and decrease of Metabolic Clearance Rate or (2) in cases with normal Aldosterone-Secretion by decrease of Metabolic Clearance Rate alone. Evidence exist that (3) exclusive increase of Aldosterone-Secretion can occur as well. No correlation was found between renal Excretion, Secretion Rate and Mean Plasma Concentration. Normalisation of Aldosteron metabolism could be observed after nephrectomy in three patients and was associated with a reduction of systemic blood pressure. Whereas the increase of Aldosteron Secretion can be explained by a stimulatory action of angiotensin on the adrenal cortex, an impaired hepatic blood flow is considered to be the cause of the reduced Metabolic Clearance Rate in renal artery stenosis.

Authors’ address: W. Kaufmann, B. Steiner, F. Dürr, H. Nieth and C. Behn, Medizinische Universitäts-Klinik, Otfried-Müller-Strasse. 7400 Tubingen (Germany)

Metabolism of plasma glucose and lipids following diazoxide administration in dogs

The mechanism by which diazoxide influences carbohydrate and lipid metabolism has been investigated in healthy, anesthetized dogs, using isotope-dilution techniques. Results have shown that single intravenous injections of diazoxide in the dose of 15 or 20 mg/kg body weight produce the following alterations: Firstly, diazoxide increases plasma concentration and intermixing mass of glucose due in part to a prompt increase in rate of glucose appearance (hepatic production) into the circulation. This is in keeping with findings of others in mice and rats that diazoxide decreases hepatic glycogen content. Secondly, diazoxide produces no significant alterations in the rate of glucose utilization by tissues despite the marked increase in plasma glucose concentration. This is most likely related to the reported inhibition of insulin secretion and increased catecholamines and plasma free fatty acid (FFA). Thirdly, diazoxide increases the turnover rates of plasma FFA, indicating an increase in flux of FFA from fat depots into the circulation, probably induced by catecholamines. Fourthly, diazoxide had no immediate effect on plasma triglyceride or cholesterol concentration, but the incorporation of labeled FFA into plasma triglyceride was increased by diazoxide despite the fall in plasma FFA specific activity which presumably would dilute the hepatic pools of FFA that provide precursors for triglyceride synthesis. These findings suggest that diazoxide accelerates triglyceride synthesis without change in plasma triglyceride concentration. In conclusion, diazoxide induces hyperglycemia due to increased hepatic glucose production and relative inhibition of glucose utilization by tissue, and it increases plasma FFA concentration by enhancing the influx of FFA into plasma.

Author’s address: Dr. S.S. Sanbar, Department of Internal Medicine, The University of Michigan, Ann Arbor, Mich. 48104 (USA).

The renal accumulation of urate and />-aminohippurate in the rabbit

The concentration of urate and PAH in cortex kidney tissue was examined during mannitol diuresis in rabbits anaesthetized with pentobarbital. After administration of 2,4-dinitrophenol, fumarate, succinate, probenecid, and salicylate accumulation and renal net secretion of urate
were abolished. The secretion and accumulation of PAH were only partially inhibited during infusion of these substances. Accumulation of urate was depressed in relation to the reduction of urate secretion during infusion of PAH. The data show a correlation between renal accumulation and excretion of urate and PAH. The renal accumulation of urate and PAH in rabbits is in all likelihood predominantly a consequence of tubular secretion of these two substances.

Author’s address: Dr. J.V. Møller, Department of Biochemistry, University of Aarhus, Aarhus (Denmark).

Etudes histochimiques sur le role des mucopolysaccharides acides de la médullaire rénale dans les processus de la concentration urinaire

La médullaire du rein, siege des processus ultimes de la concentration urinaire, est particulièrement riche en mucopolysaccharides acides. La densité de leurs charges negatives libres s’accroît avec la capacité de la concentration des espèces animales étudiées. En outre, chez le rat et le lapin, elle est nettement plus forte dans l’antidiurèse qu’au cours de la diurèse aqueuse. L’hypothèse est envisagée, selon laquelle ces phénomènes sont directement liées aux mécanismes d’élaboration de l’urine terminale.

Adresse de l’auteur: Dr. J.C. Morard, Laboratoire du Pr. Halpern, Hôpital Broussais, Paris XIV-e (France).

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Renal blood flow, sodium excretion, and concentrating ability during saline infusion

During rapid infusion of isotonic saline (1500 ml in 30 min), p-aminohippurate clearance (Cpah) increased 20% and PAH extraction (Epah) decreased from 83 to 71 %. Estimated noncortical plasma flow increased strikingly, from 49 to 129 ml/min. When the same volume of saline was given more slowly (1500 ml in 2 h), Cpah increased 9% whereas Epah decreased only from 81 to 78%. Estimated noncortical plasma flow increased only slightly, from 48 to 65 ml/min. The differing responses of total and noncortical plasma flow to rapid and slow infusion seem most closely related to the rapidity of initial expansion, although the duration of saline infusion may also be a factor. Despite greater noncortical plasma flow, medullary sodium content and negative free-water clearance (TỈ20) were higher during rapid saline infusions. This suggests that sodium transport in the loop of Henle was greater during rapid infusion. In another series of experiments, it was found that sodium excretion increases during slow saline infusion, despite reduction of total and noncortical plasma flow, as well as filtered sodium, by aortic clamping. These studies demonstrate that the natriuresis of saline loading is not caused by increased total or noncortical renal plasma flow.

Authors’ address: Dr. A. Shuster, Dr. E.A. Alexander, Dr. R.C. Lalone and Dr. N.G. Levinsky, 80 East Concord St., Boston, Mass. 02118 (USA).

Impaired renal conservation of chloride and the acid-base changes associated with potassium depletion in the rat

In the rat, the renal conservation of chloride during the ingestion of a low chloride diet, when sodium was given as the neutral phosphate, was very efficient, but became markedly impaired during severe potassium depletion, induced by des-oxycorticosterone acetate injections and dietary potassium restriction. This impairment was restored rapidly to normal by even partial
repair of the potassium deficit. This chloride leak is tentatively attributed to a defect in active chloride transport in the collecting duct. In severe potassium depletion with alkalosis, the accompanying hypochloraemia resulted from a negative chloride balance, secondary to renal chloride loss. In less severe potassium depletion, marked alkalosis and hypochloraemia could exist without chloride depletion, the hypochloraemia then being due to extracellular fluid expansion. Recovery from severe alkalosis due to combined potassium and chloride depletion was studied. Replacement of either ion independently of the other (with sodium chloride or potassium bicarbonate) resulted in an approximately 50% correction of the elevated serum bicarbonate. After sodium chloride, the fall in serum bicarbonate was associated with a marked reduction in renal acid excretion. After potassium bicarbonate, the fall in serum bicarbonate and the neutralization of ingested bicarbonate was attributed to the correction of intracellular acidosis since there was no marked change in acid output. Replacement of both potassium and chloride, as potassium chloride, restored the serum bicarbonate completely to normal by the combined effect of moderate suppression of urinary acid excretion and correction of intracellular acidosis.

Author’s address: Dr. R. G. Luke, Renal Division, Department of Medicine, University of Kentucky Medical Center, Lexington, Ky. 40506 (USA).

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Renal contribution to thoracic duct lymph in dogs
The renal contribution to thoracic duct lymph in anaesthetised dogs was estimated from the decrease in thoracic duct flow which followed acute renal arterial occlusion. The operation to occlude the renal artery was carried out in two stages –the first to expose the artery and the second, an hour later, to clamp the artery. Control thoracic duct flow measurements and renal plasma flow and glomerular filtration rate studies indicated that these parameters were not significantly affected of the first stage of the operation. In animals which were not infused during experiment the average values obtained for control thoracic duct flow, left renal contribution and right renal contribution were 1.4, 0.4 and 0.35 ml/h/kg body weight: in animals infused with isotonic saline or dextrose (approx. 1 ml/min) these values were 2.0, 0.7 and 0.3 ml/h/kg body weight respectively. In three experiments occlusion of the renal vein, after release of arterial occlusion, resulted in an abrupt 3 to 4 fold increase in thoracic duct flow. Close correlation was found between control thoracic duct flow and body weight and between renal lymph flow and control thoracic duct flow.

Author’s address: C.C.C. O’Morchoe, Department of Physiology, Trinity College, Dublin University, Dublin 2 (Ireland).
The effect of urine osmolality and pH on the bactericidal activity of plasma
(1) The ability of normal human plasma to inhibit the growth of Escherichia coli and Proteus mirabilis in urine has been studied.

1200
300  600  900
Urine (m-osmole/kg)
Fig. 1. Effect of increasing osmolality on the bactericidal activity of plasma in urine at pH 7.4. Rough strain of E. coli.
Bacterial growth (%) × 100.
Organisms/ml in urine with plasma
Organisms/ml in urine without plasma

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200
½; 150–
□ 100
50

1 1 1 1 _
900 1200
300 600

Urine (m-osmole/kg)

Fig. 2. Effect of increasing osmolality on the bactericidal activity of plasma in urine at pH 5.3.
Rough strain of E. coli.
At pH 7.4 bacterial growth was inhibited by plasma in isotonic and hypo-tonic urine.
Plasma did not inhibit bacterial growth in hypertonic urine.
It is suggested that this effect may contribute to the susceptibility of the renal medulla to infection.

Author’s address: Dr. H. Acquatella, Department of Medicine, Charing Cross Hospital Medical School, Fulham Hospital, London. W. 6 (England).

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Author’s address: Dr. H. Acquatella, Department of Medicine, Charing Cross Hospital Medical School, Fulham Hospital, London. W. 6 (England).

Hyperuricemia and urate excretion in chronic renal disease
Standard renal clearances were performed in 92 subjects, comparing uric acid excretion, glomerular filtration and renal plasma flow in normal people and people with proven chronic renal disease with normal and sub-normal glomerular filtration. Despite comparable filtered urate loads, patients with chronic renal disease, and normal GFR demonstrate decreased uric acid-clearance and hyperuricemia. So too, do patients with decreased GFR, but the latter also manifest enhancement of fractional uric acid excretion. Comparison of true creatinine excretion and maximum rates of glucose reabsorption in normal and abnormal subjects show similar adaptive alterations. Although uric acid retention occurs early in the course of chronic renal disease, compensatory tubular function of a nonspecific nature tends to minimize it as renal disease progresses.

Author’s address: John J. McPhaul, Jr., M.D., Department of Experimental Pathology, Scripps Clinic and Research Foundation, Lajolla, Calif. 92037 (USA).

Une néphropathie glomérulaire apparemment bénigne: les depots fibrinoïdes intercapillaires
A l’examen de biopsies rénales, des depots fibrinoïdes intercapillaires constituent parfois l’unique lesion glomérulaire, les autres structures du rein étant indemnes. L’individualité de cette alteration est certaine car elle ne semble pas exister en association avec d’autres lesions glomérulaires. A cette atteinte morphologique correspond un syndrome urinaire, variable mais
indépendant de tout déficit fonctionnel renal ou de toute tendance hypertensive. L’étiologie en est encore totalement inconnue. Tout suggère que révolution en est bénigne.
Adresse de l’auteur: L. Morel-Maroger, Chaire de Néphrologie Clinique et Expérimentale, Hôpital Tenon, 4, rue de la Chine, Paris 20e (France).

Degradation of 131I-labelled bovine insulin by kidney homogenates of the rabbit in presence of serum from normal and uremic rabbits/Untersuchun-gen zum Abbau von Rinderinsulin-131I durch Kaninchennierenhomogenat in Anwesenheit normaler und urämischer Seren von Kaninchen

In rabbits with artificial uraemia produced by operative ligature of both ureters the degradation of intravenously administered 131I-labelled bovine insulin was found to be slower than in sham-operated controls. When dilutions of the soluble supernatant of kidney homogenates were incubated with serum from uraemic rabbits the degradation of 131I-labelled bovine insulin was protected as compared with control sera. The Michaelis constant in presence of control and uraemic serum was the same Km = 23.5 ×10-8 Mol/l, Km = 24.6 ×10-8 respectively. It is concluded that in the serum of uraemic rabbits a non-competitive inhibition of the insulinase takes place.

Author’s address: Dr. J.W. Woenckhaus, Medizinische Universitäts-Klinik Freiburg, Hugstetter Strasse 55, 7800 Freiburg (Germany).

The renal response of children to acute ammonium chloride acidosis
Observations of the renal response of children aged 4-13 years to administration of ammonium chloride support the hypothesis that the concentration of bicarbonate in glomerular filtrate, together with the level of the bicarbonate threshold, plays a major role in the regulation of renal excretion of hydrogen ion. An inverse relation was demonstrated between the total CO2 content of blood and the concentration of hydrogen ion in urine. Rates of excretion of titratable acid and ammonium increased as concentration of total CC½ in blood fell below normal levels; both rates reached a plateau, however, when total CC½ reached concentrations a few millimoles below the renal bicarbonate threshold. All children achieved a value of urinary pH of 5.5 or less. The mean rates of excretion of titratable acid and ammonium were 52 µEq/min/1.73 m2 (range ± 2 S.D., 33-71) and 73 µEq/min/1.73 m2 (47-100), respectively. These results indicate that the necessary condition for assessing maximal acidifying capacity during metabolic acidosis is that the blood bicarbonate concentration be several millimoles below the renal bicarbonate threshold; the use of a "standardized" dose of ammonium chloride does not assure a maximum response. Evidence is presented that calculation of the hydrogen ion clearance index does not provide a physiologically meaningful assessment of urinary acidification.

Author’s address: Dr. CM. Edelmann, Jr., Department of Pediatrics, Albert Einstein College of Medicine, Bronx, N. Y. (USA).

Concentration of blood pyruvate and maximal rate of tubular excretion of PAH in infectious hepatitis/Blut-Pyruvatspiegel und tubuläres Transport-maximum für para-Aminohippursäure (PAH) bei Hepatitis epidemic
In normals as well as in patients with infectious hepatitis a positive correlation between concentration of blood pyruvate and the maximal rate of tubular excretion of \( >^pah \) was found. It is suggested that the increased concentration of blood pyruvate might result in an increased \( 7\Pi^pah \) in these patients.

Author’s address: Dr. P. Dittrich, Medizinische Universität-Klinik, Α 6020 Innsbruck (Austria).


Comparative histometrical measurement of total area of glomerulus, capillary tuft and free capsular space were carried out in 12 kidneys after sudden death (right heart failure mostly) and in ten kidneys after two to three days of acute renal failure with the following results:

Total glomerular area is equal in both groups.
Capillary tuft area is 26% smaller in acute renal failure than after sudden death (this means a 52% reduction in volume).
Free capsular space increases by 52% in area and by more than 100% in volume after acute renal failure in comparison to sudden death.

These results show that not only wide tubular lumina, but wide capsular spaces as well are commonly found in patients dying with acute renal failure.
This increase in capsular space is brought about by osmotically induced water transport from tubular cells into lumen after death has occurred. Thus evidence is brought about of the importance of passive water movement in the morphogenesis and possibly in the pathogenesis of acute renal failure.

Author’s address: Dr. Hans Christian Burck, Medizinische Universität-Klinik, 7400 Tubingen, Schnarrenberg (Germany).

Summaries – Resumes
Sodium excretion was studied in a group of patients with chronic renal disease, (a) on constant salt intakes of varying amounts with and without mineralocorticoid hormone administration, and (b) after acute extracellular fluid volume expansion. The lower the steady state glomerular filtration rate (GFR), the greater was the fraction of filtered sodium excreted on both 3.5 to 7.0 g salt diet; and the lower the GFR, the greater was the change in fractional excretion in the transition from the 3.5 to 7.0 g salt diet. This regulatory capacity did not appear to be influenced by mineralocorticoid hormone administration. After acute expansion of extracellular fluid (ECF) volume, the increment in sodium excretion exceeded the concomitant increment in filtered sodium in six of nine studies and in the remaining three studies, the increment in excretion averaged 59% of the \( \Delta \) filtered loads (i.e., only 41% of the increase in filtered sodium was reabsorbed). During saline loading, the decrease in fractional reabsorption of sodium tended to vary inversely with a steady-state GFR, although all patients received approximately the same loading volume. When an edema-forming stimulus was applied during saline infusion, the natriuretic response was aborted and the lag time was relatively short. When GFR and the filtered load of sodium was increased without volume expansion, the \( \Delta \) sodium excretion averaged on 19% of the \( \Delta \) filtered load; more over, change in fractional sodium reabsorption were consistently smaller than those observed during saline loading. The authors concluded that the
presence of a factor other than GFR and mineralocorticoid changes are present in the modulation of sodium excretion in uremic man.

Author’s address: Dr. Eduardo Slatopolsky, Renal Division, Department of Internal Medicine, Washington University School of Medicine, St. Louis, Mo. (USA).

Book Review – Livre Nouveau

Handbuch der inneren Medizin

Important traité de Néphrologie auquel ont contribué les plus grands spécialistes de langue allemande, chacun traitant un sujet auquel il s’est lui-même consacré. Le premier tome, porte sur la morphologie et la physiologie normale et pathologique, les méthodes d’exploration, l’insuffisance rénale et l’immunologie des néphropathies. Dans le deuxième tome, l’analyse des grands syndromes est complètée tandis qu’est abordée celle des principales néphropathies, primitives d’apparence, secondaires à une cause précise ou associées à divers états morbides, qui se poursuit dans le 3ème tome, complétée par l’étude des confins de la Néphrologie et de l’Urologie.

Deux qualités dominent cet ouvrage: la richesse de la documentation, évidente dans le texte comme dans la bibliographie, et la perfection de la présentation, tout particulièrement des reproductions microscopiques, et radiologiques.

G. Richet