the size of individual parietal cells were found to be transient in nature, as following the cessation of the hormonal treatment the size of these cells returned to normal. On the other hand, the overall number of the parietal cells did not respond respectively, as the significant decrease in the number of these cells was sustained till the end of the recovery period. The present study, however, tends to indicate that the parietal cells are not involved solely in the process of glucocorticoid-induced gastric disorder. It became apparent that almost all the cell types composing the mucosal lining of the stomach are intimately involved. Hence, it would be reasonable to assume that the pathology resulting from triamcinolone is the result of a concomitant adverse effect upon the heterogenous cellular population responsible for the structural and metabolic integrity of the gastric mucosa.

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Active and inactive renin in dog plasma before and after bilateral nephrectomy

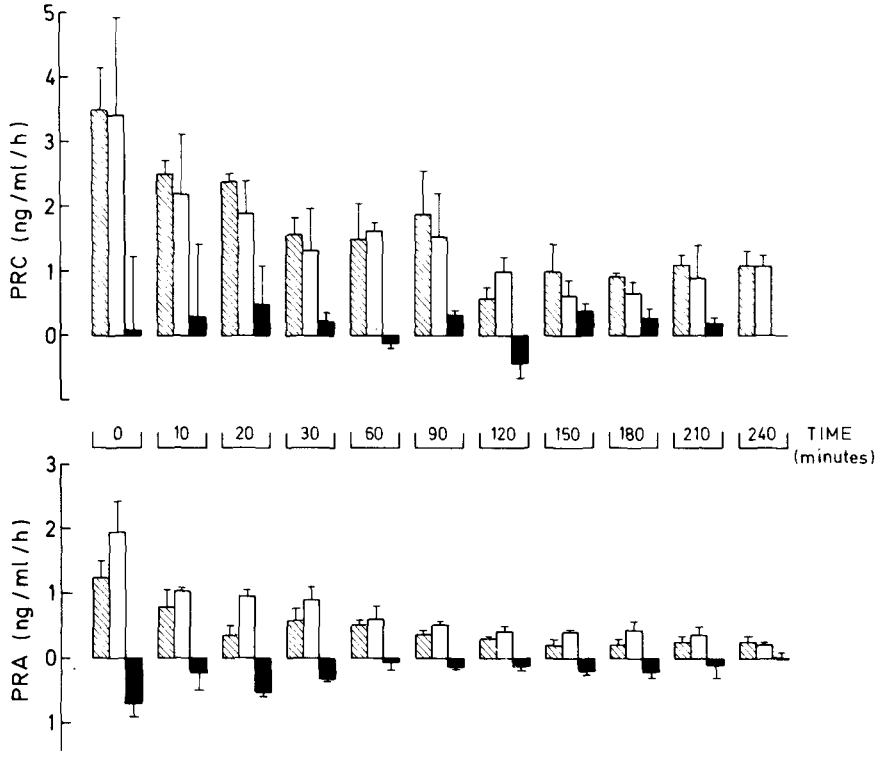
J. Staessen[1], P. Lijnen, R. Fagard and A. Amery[2]

Hypertension and Cardiovascular Rehabilitation Unit, Department of Pathophysiology, University of Leuven, B-3000 Leuven (Belgium), 13 October 1980

Summary. No significant amounts of inactive renin could be demonstrated by in vitro treatment (acidification or cryotreatment) of dog plasma obtained before and after bilateral nephrectomy. After bilateral nephrectomy, total and active renin were cleared from the plasma following similar disappearance curves, and dropped to half of their initial value within 30 min.

In a previous paper we reported on the disappearance of endogenous renin from the circulation of the dog after bilateral nephrectomy[3]. However, plasma renin was measured by a method involving acidification of the plasma to remove the endogenous renin substrate and an excess of exogenous renin substrate was then added for the generation of angiotensin I[4]. In the absence of a direct measurement, the plasma renin level is expressed as the amount of angiotensin I generated per ml of plasma/h. Meanwhile it has been recognized that the acidification of the plasma may activate an inactive form of renin[5]. The earlier described disappearance curve of renin after nephrectomy in dogs might represent a combination of the disappearance curves of active and inactive renin[3].

Inactive renin is not only activated by acidification but also by cold treatment of plasma[6]. In the present study we



Total (▨), active (□) and inactive (■) plasma renin concentration (PRC) and activity (PRA) before and after bilateral nephrectomy.

investigated in the dog the plasma levels of active renin, and of cryo- and acid-activated renin, before and after bilateral nephrectomy.

Material and methods. 3 male mongrel dogs with an average weight of 24.3±2.6 (SEM) kg were anaesthetized with 30 mg/kg sodium pentobarbitone i.v. A catheter was introduced into the femoral artery for blood sampling. The kidneys were exposed extraperitoneally by a flank incision, and dissected free from the surrounding tissues. After a recovery period of 60 min renal arteries and veins were clamped simultaneously on both sides, and the kidneys removed within 1 min. Arterial blood for renin estimation was withdrawn 5 min before and at 0, 10, 20, 30, 60, 90, 120, 150, 180, 210 and 240 min after removal of kidneys. Blood was collected into ice-cooled tubes containing EDTA, and o-phenanthroline. Plasma samples were stored, at −20 °C, until assay.

The plasma concentration of total, active and inactive renin was determined, as described previously[7]. For the estimation of total renin (TPRC) and active renin concentration (APRC) plasma was dialyzed against 0.05 M-glycine buffer (pH 3.3 and pH 4.5, respectively) containing 5 mM EDTA and 90 mM NaCl. This was followed by inactivation of the angiotensinases by heating at 32 °C for 1 h. The plasma was then incubated with excess sheep renin substrate at pH 7.4 for 1 h, as described by Skinner[4] and angiotensin I measured by radioimmunoassay (Lijnen et al.[8]). The difference between TPRC and APRC is described as the inactive plasma renin concentration (IPRC).

The cryoactivation of renin[6] was investigated by measuring the plasma reinin activity in plasma samples immediately after thawing and after storage of the plasma at −5 °C and at pH 7.4 for 4 days. The value of PRA before and after the exposure of the plasma to the cold is called respectively active (APRA) and total plasma renin activity (TPRA). The calculated difference between TPRA and APRA is considered as inactive plasma renin activity (IPRA). Plasma renin activity was measured by radioimmunoassay of the angiotensin I which was generated during incubation of the plasma samples for 1 h at 37 °C with the endogenous renin substrate at pH 6.0[9] according to the method of Fyhrquist and Puutula[10].

Results and discussion. As shown in the figure, the control values (average of time zero and 5 min before) were 3.5±0.65 (SEM) ng/ml/h for TPRC and 3.43±1.51 ng/ml/h for APRC. The concentration of acid-activated inactive renin was very low (0.07±1.14 ng/ml/h). In the control period TPRA averaged 1.25±0.26 ng/ml/h, and APRA 1.95±0.45 ng/ml/h. Cryoactivated inactive renin could not be demonstrated. Likewise, after nephrectomy IPRC and IPRA could not be demonstrated by acid activation or cryotreatment respectively. This is in contrast to the findings in human plasma where renin can be activated by various in vitro procedures such as acidification, exposure to cold or treatment with proteolytic enzymes[5]. Cryo- or acid activation of inactive renin has also been demonstrated in the plasma of several other species such as pigs[11], rabbits[12] and rats[13].

After bilateral nephrectomy the clearance of total and active renin from dog plasma is similar (fig.). Acidification and cryotreatment of dog plasma do not seem to interfere with the disappearance of renin.

On average, plasma renin (TPRC, APRC, TPRA, APRA) fell to half its initial value within 30 min; this is in agreement with previous studies[3,14].

1 Address for correspondence: Dr J. Staessen, Inwendige Geneeskunde, Cardiologie, A.Z. St. Rafaël, Kapucijnenvoer 35, B-3000 Leuven, Belgium.

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PRO EXPERIMENTIS

Demonstration of particulate accumulations in solid tissue[1,2]

M.E. LeFevre[3], H.M. Jablonski, J.A. Pile and D.D. Joel

Medical Research Center, Brookhaven National Laboratory, Upton (New York 11973, USA), 1 December 1980

Summary. We describe a histological clearing procedure involving partial tissue hydrolysis by pancreatin and demonstrate its application. Changes in distribution of 5.7-μm latex particles in mouse liver and heart muscle 6 months after i.v. latex injection were quantitated. The method is useful for studies of long-term redistribution of particles in vivo and for locating large, infrequent particles which are difficult to find by ordinary tissue-sectioning techniques.

Many satisfactory procedures are available for studying the disappearance of small, nondegradable particulates from blood and their accumulation in the tissues of the reticuloendothelial (RE) system[4]. Methods for following the subsequent fate of such particles, however, are not as well developed. The present report describes a clearing procedure which is useful for studies of the storage and redistribution of particles within tissues. The procedure is a modification of a KOH-glycerol clearing process previously applied to mouse Peyer's patches and mesenteric lymph nodes for the demonstration of accumulated 2-μm latex particles[5]. The earlier technique gave satisfactory results when applied to pale tissues, but was less successful with tissues such as liver and spleen. The new procedure, which utilizes partial tissue hydrolysis by pancreatic enzymes, successfully clears all types of soft tissue and is well suited