Sodium Retention in Experimental Glomerulonephritis and Natriuretic Factor

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Dear Sir,

In a recent issue of this journal, Van Liew et al. [1] demonstrated the absence of sodium retention in rats with nephrotic syndrome (serum sickness and Heyman nephritis). Their conclusions were based on the absence of ascites and the absence of a significant difference in absolute sodium excretion between the nephrotic animals and the controls, both placed in metabolic boxes and starved for 16 h. Such findings are in agreement with those of Allison et al. [2], who also failed to find sodium retention in these animals. In contrast, in 1972 we observed in antglomerular basement membrane glomerulonephritis [3], and in 1978, Bernard et al. [4] found in Heyman nephritis an impaired sodium excretion in the glomerulonephritic animals when compared to controls. Such discrepancies in the published results can be explained by the different natriuretic stimuli: important saline expansion in the study of Bernard et al. [4] and our [3] studies, as compared to the moderate or absent saline expansion in the experiments of Allison et al. [2] and Van Liew et al. [1]. As we recently demonstrated [5] in experimental antglomerular basement membrane glomerulonephritis, an impairment in sodium excretion can be observed only with a sufficient natriuretic stimulus. In response to 2 types of volume expansion in the same animals, with however a similar amount of absolute sodium perfused in each, only the rapid volume expansion was sufficient to unmask the inability to excrete sodium in the nephritic rats (fig. 1). Therefore, the absence of a disturbance in sodium excretion in the nephrotic animals of Van Liew et al. [1] can be explained by an insufficient natriuretic stimulus. Moreover, as already shown, the glomerulonephritic rats present hypertension [2,3,5] and are probably hypervolemic [6]. Therefore, in the absence of a sodium or volume challenge, their baseline sodium excretion is comparable if not greater to the control rats.

We wonder what was the sodium intake and the blood pressure level of the animals in the above mentioned study [1].

Finally, Van Liew et al. [1] support in their paper the hypothesis that sodium retention in glomerulonephritis might be due to some intrarenal mechanisms. We wish to remind your readers that we had already demonstrated in 1972 that the inability of glomerulonephritic rats to excrete a sodium load was due to such intrarenal mecha-

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Fig. 1. Comparison of absolute and fractional excretion of sodium during slow volume expansion (SVE) and rapid volume expansion (RVE). Rats received a similar amount of sodium (598 μ mol) during each period, but different rates and volumes of perfusions. * $p < 0.001$; glomerulonephritic (GN) animals compared to normal ones (N) within the same period. Values are given as means $\pm$ SEM.

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nisms [3] which was identified as being the disappearance of a natriuretic factor [7] of renal origin [8,9]; its administration to the glomerulonephritic kidney restored the ability to excrete a saline load [10, 11]. This renal natriuretic factor was also absent in human glomerulonephritis with or without nephrotic syndrome [12]. We think that such a mechanism should certainly be considered as an explanation for the sodium disturbance observed in other models of experimental glomerulonephritis at a time when an increasing number of publications is confirming the importance of such intrarenal mechanisms.

References

- Van Liew, J.B.; Noble, B.; Brentjens, J.R.: Absence of sodium and water retention in rats with severe proteinuria. *Nephron* 40: 476–481 (1985).
- Allison, M.E.M.; Wilson, C.B.; Gottschalk, C.W.: Pathophysiology of experimental glomerulonephritis in rats. *J. clin. Invest.* 53:1402–1423 (1974).
- Godon, J.P.: Sodium and water retention in experimental glomerulonephritis. *Kidney int.* 2/271–278 (1972).
- Bernard, D.B.; Alexander, E.A.; Couser, W.G.; Levinsky, N.G.: Renal sodium retention during volume expansion in experimental nephrotic syndrome. *Kidney int.* 74:478–485 (1978).
- Chachati, A.; Godon, J.P.: Impaired sodium excretion in experimental glomerulonephritis: an explanation for the controversies in the literature. *Renal physiol.* (in press, 1985).

Mees, E.J.D.; Geers, A.B.; Koomans, G.H.A.: Blood volume and sodium retention in the nephrotic syndrome: a controversial pathophysiological concept. *Nephron* 56:201–211 (1984).

- Godon, J.P.: Sodium and water retention in experimental glomerulonephritis: the urinary natriuretic material. *Nephron* 14: 382–389 (1975).
- Godon, J.P.; Nizet, A.: Release by isolated dog kidney of a natriuretic material following saline loading. *Archs int. Physiol. Biochim.* 54:309–311 (1974).
- Godon, J.P.; Dechenne, C: In vitro production of a natriuretic material of renal origin. *Renal physiol.* 7:201–210 (1978).
- Godon, J.P.; Chachati, A.: Edema and third factor in experimental nephritis; Kiyoshi Seki, Casley-Smith, Andreoli, *Edema: recent advances*, pp. 245–252 (Tokyo 1981).
- Godon, J.P.: Evidence of increased proximal sodium and water reabsorption in experimental glomerulonephritis: role of a natriuretic factor of renal origin. *Nephron* 27:146–154 (1978).