Conflicting Results on Peripheral Adrenoceptor Function in Hypotension of Hemodialyzed Patients

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Dear Sir,

It is with great interest that we read the contribution by Lübbecke and co-workers entitled 'Thrombocyte Alpha-2-Adrenoceptors and Hypotension in Hemodialyzed Patients' published as Letter to the Editor, in this edition of Nephron. They discuss their data taking into account our previous paper on the same topic published last year in Nephron [1]. The results by Lübbecke on platelet alpha-2-adrenoceptors do not agree with our findings. In addition, they raised some doubts on our results, mainly on the methodology we used. We do not agree with the analysis expressed by Dr. Lübbecke and respond to his criticisms as follows:

The clinical characteristics of our patients (mean blood pressure 85.7 ± 12.1 mm Hg) are different from those of Lübbecke (mean blood pressure 102 ± 18 mm Hg). Hypotension is a frequent occurrence in chronic hemodialysis patients, and uremic hemodialyzed patients with transient hypotension during dialysis must be distinguished from those with stable hypotension [2]. A high number of platelet alpha-2-adrenoceptors is detectable only in patients with stable hypotension, as shown in table 1 which summarizes our experience.

It is always very difficult to compare results derived from receptor-binding studies performed with different ligands. In particular, 3,4-rauwolscine, a high-affinity alpha-2-adrenoceptor antagonist, binds to both highland low-affinity alpha-2-adrenoceptor sites in the platelet membrane, while 3,4-yohim-bine binds to only one class of binding sites on human platelet membranes [3].

In order to avoid binding site loss during membrane preparation, we used intact platelets in our study. We agree with Lübbecke that the expression of binding values in terms of milligrams of membrane protein may be somewhat inaccurate. However, as data obtained previously in our laboratory have shown, a close linear relationship exists between the maximum number of platelet 3H-yohimbine binding sites expressed as sites/ platelet and those as fmol/mg protein.

(4) Conflicting data have been reported on plasma levels of catecholamines in patients on maintenance hemodialysis. Recently, it has been shown that the concentration of plasma noradrenaline increased significantly with increasing duration of dialysis treatment [4]. The absence of receptor down-regulation in the presence of a high concentration of the physiological agonist could be the consequence of an altered receptor function and/or defective postreceptor events. We are aware that a possible defect in the adrenoceptor coupling mechanism is only an interesting hypothesis that requires further confirmation. Nevertheless, to state that the basic

receptor mechanism seems to be unchanged in uremic patients on the basis of adenylate cyclase activity assessed in only 2 patients, seems to us much less substantiated.

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