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LETTER TO THE EDITOR

Hypertension, Platelets, and Inflammatory Responses

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The exciting new basic studies reported in this journal show that A-II blocking agents can be expected to have an effect on the inflammatory responses mediated by platelets, with ultimate inhibition of AII-induced hypertension by the antiplatelet agent clopidogrel [1, 2].

Both the article and the Editorial focused on hypertension in the title, implying that the logical preferential therapy for clinical hypertension would be angiotensin-II (A-II)-blockade by an angiotensin converting enzyme inhibitor (ACEI) or an A-II receptor blocker (ARB). However, that conclusion would be misleading. Weighty bodies such as the European Hypertension Society and JNC 8 indicate that initial therapy of clinical hypertension can be by one of three classes of agents [3, 4]. These are diuretics that often indirectly stimulate A-II activity [5], or by calcium channel blockers (CCBs) that have no known effect on A-II, or by an ACEI or ARB. There is thus an important distinction between hypertension as known to clinicians and A-II induced hypertension as newly described by these basic workers [1, 2]. It is in patients with primary aldosteronism that increased oxidative stress is reported [6].

The Editorial also stresses the pro-inflammatory role of platelets [2]. What is the clinical evidence? There are substantial links between C-reactive protein (CRP), a marker of the inflammatory response and the global cardiovascular risk [7, 8]. Furthermore, C-reactive protein and cholesterol may now be regarded as equally strong predictors of cardiovascular risk and both are important for quality clinical care [9]. However, when it comes to clinical hypertension [10], "measures of CRP do not seem to add to the evaluation or management of hypertensives." Antihypertensive therapy, although improving arterial stiffness, has only small effects on markers of inflammation and endothelial activation [11].

Thus there are major differences between experimental angiotensin II-induced and clinical hypertension. From the point of view of clinical therapy of hypertension, the group of agents that might have anti-inflammatory effects are the ACEIs and ARBs which are ranked equally with diuretics that often increase inflammatory markers [12], while calcium channel blockers (CCBs) have no known anti-inflammatory effects. Nonetheless, there is a relevant potential clinical application of the basic science study by Jia et al [1]. Their studies lead to the suggestion that clopidogrel might influence clinical hypertension [1]. Thus in selected patients with adrenocortical-induced hypertension, clopidogrel therapy may have a dual role, both as a novel blood pressure reducing agent, besides also being an antiplatelet agent, thus indirectly also countering coronary heart disease. That would a major practical application of the basic work reported by Jia et al. [1].

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