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Envoi

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As most readers know, the weight of scientific and clinical opinion around the world is that approximately one in ten patients with hypertension has primary aldosteronism. What is understandable, given two determinants of how we approach this issue, is that our focus has been very narrow. The first is that aldosterone is 'disputed territory', living in the marches rather than the mainstream—discovered by a physiologist and biophysicist, characterized by nephrologists, embraced by endocrinologists (and much more recently cardiologists), and on occasion by clinical pharmacologists. The second is that the realization that this ~10% of hypertension reflects autonomous aldosterone secretion is relatively recent, and on occasion turning round medical opinion and clinical practice is like turning round the Queen Mary.

One upshot of this is that the focus among the informed and expert practitioners has been squarely on diagnosis and management, an approach that might seem to reflect the historic teaching that primary aldosteronism is both a rare and relatively benign form of hypertension. Neither of these is palpably the case, the contribution of my good friend Norman Kaplan notwithstanding, and what this does in my opinion is underscore the need to refocus our attention and energy.

If 30% of the population in many countries with highly developed health care systems are hypertensive, and 10% of these have primary aldosteronism, we are faced with hundreds of thousands in lightly populated countries such as Australia, and millions with primary aldosteronism elsewhere. It is currently inconceivable that such numbers can be screened for autonomous aldosterone secretion, at least until the development of simple, accurate and cheap point-of-care methodology. Subsequent exclusion/confirmatory testing, not to mention adrenal venous sampling, are even more inconceivable, whether privately or publically funded, given the numbers involved.

While current opinion is that laparoscopic surgery is optimal for a unilateral adrenal producing adenoma, there are also reports that after 5 years the results of medical and surgical treatment of such patients are indistinguishable. To date we have focused on the tip of the iceberg: tomorrow needs to be different, in many ways along the lines suggested both by Norman Kaplan and another good friend from Dallas, Richard Auchus. What I would suggest as a blueprint for future attention and action is:

1. A campaign to bring to the notice of primary care-givers the magnitude of the issue, the gravity of aldosterone excess induced hypertension, and the logistic and financial impossibility of addressing it by our current approaches;
2. Given the impossibility of optimally managing all but a tiny proportion of cases in even advanced countries, and the utility of mineralocorticoid receptor blockade in essential hypertension, to advocate low dose spironolactone or eplerenone as first-line treatment, in combination with standard anti-hypertensives, for all hypertensive patients;
3. Remaining alert to the occurrence of a constellation of symptoms (hypokalemia/youth/resistant hypertension/atrial fibrillation/etc.) which might point to investigation and intervention;
4. Refining measurement of aldosterone to renin ratio to a simple point-of-care procedure, to allow focus on

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the ~10% of patients with primary aldosteronism: and refining non-invasive distinction of unilateral and bilateral disease.

In public health terms, the first two of these are of overwhelming importance, and essentially the realm of medical education and politics. As such, they may be more difficult, and less interesting, than the latter two suggestions: this should not however, detract from what needs to become the main game for primary aldosteronism in the second and subsequent decades of the 21st century. It's now primarily a problem of public health.