Reduction of blood pressure already in the normal range further regresses left ventricular mass


Heart 2010 96: 1080 originally published online May 18, 2010
doi: 10.1136/hrt.2009.191619

Updated information and services can be found at:
http://heart.bmj.com/content/96/13/1080.1.full.html

These include:

References
This article cites 8 articles, 7 of which can be accessed free at:
http://heart.bmj.com/content/96/13/1080.1.full.html#ref-list-1

Article cited in:
http://heart.bmj.com/content/96/13/1080.1.full.html#related-urls

Email alerting service
Receive free email alerts when new articles cite this article. Sign up in the box at the top right corner of the online article.

Notes

To order reprints of this article go to:
http://heart.bmj.com/cgi/reprintform

To subscribe to Heart go to:
http://heart.bmj.com/subscriptions
Reduction of blood pressure already in the normal range further regresses left ventricular mass

To the Editor: We read with interest the paper by Simpson and colleagues describing a reduction in left ventricular (LV) mass among patients with hypertension recruited with left ventricular hypertrophy (LVH) despite blood pressure being within the normal range on entry.1 Further lowering of systolic blood pressure (SBP) by 9.3 (3.6) mmHg resulted in a reduction of LV mass index by 4.7 (3.7) g. We have recently demonstrated a comparable drop in LV mass in normotensive (<130/80 mmHg) patients with early chronic kidney disease and normal LV mass at baseline following treatment with spironolactone in addition to angiotensin blockade.2 Compared to placebo, treatment with spironolactone for 40 weeks altered LV mass by −14 (13) g versus +5 (11) g (p<0.01) in association with a mean drop in SBP of 11 mmHg. Our data confirm the findings of Simpson and colleagues that in normotensives, LV mass falls in response to further blood pressure lowering but also that this occurs even when baseline LV mass is within normal limits; thus, any reductions in LV mass occur independently of baseline LV mass.

The presence of LVH is consistently associated with increased cardiovascular and total mortality.3 Although LVH has traditionally been considered a dichotomous variable, there is good evidence of a continuous linear relationship between LV mass and risk of cardiovascular disease. This relationship extends to values of LV mass currently considered “within the normal range.”4 Regression of electrocardiographic markers of LVH has been associated with a reduction in events in those with hypertension.5 Although there are preliminary data suggesting that a reduction in LV mass irrespective of the presence of LVH at baseline is associated with fewer non-fatal cardiovascular events,6 large-scale clinical trials are necessary to confirm this and to demonstrate a significant mortality benefit.

Finally, we agree with Simpson and colleagues that it is clear that while blood pressure reduction is an effective method of reducing LV mass, hypertension is not the only cause of LVH. The variations in response to different anti-hypertensive agents suggest that blood pressure-independent mechanisms are involved in the regulation of LV mass.7 In particular, the influence of arterial stiffness and its modulation by the renin–angiotensin–aldosterone system is likely to be of high importance.8 Such a hypothesis was supported by our work, which found that treatment with spironolactone resulted in additional significant reductions in aortic pulse wave velocity, augmentation index and aortic distensibility, suggesting that the fall in LV mass in normotensive subjects was driven by a reduction in aortic stiffness.

C D Chun,1 N C Edwards,1 C J Farro,2 R P Steeds,1 J N Townsend2

1Departments of Cardiology, Queen Elizabeth Hospital, Birmingham and University of Birmingham, Birmingham, UK; 2Departments of Nephrology Queen Elizabeth Hospital, Birmingham and University of Birmingham, Birmingham, UK

Correspondence to Dr Jonathan N Townsend, Department of Cardiology, Queen Elizabeth Hospital, Birmingham B15 2TH, UK; john.townend@uwb.nhs.uk

REFERENCES


The Authors’ reply: We thank Chue et al1 for their letter in response to our paper.2 In general, we agree with them and are grateful to them for saying that our results are consistent with their paper where spironolactone reduced left ventricular (LV) mass in renal patients.3 There are some differences between the papers worth commenting on. In our paper, the baseline mean office systolic blood pressure (BP) was 122 mm Hg, whereas in their study, it was higher at 130 mm Hg, which is the target BP for patients with target organ damage. Therefore, on these grounds alone they would reduce the target BP. Second, our patients all had echocardiographic LV hypertrophy (LVH) at baseline, whereas baseline LVH was only present in 9% to 10% of their patients. In other words, our study population was normotensive patients with LVH, whereas their study population was primarily chronic kidney disease patients with reasonably good BP control. Given that we studied somewhat different populations, it is reassuring that we found basically the same result.

It is worth commenting on a unique feature of both our studies. That relates to the magnitude of the extra BP reduction we both produced, that is, we produced an active minus placebo office BP fall of −9.2 mm Hg, whereas their corresponding figure was −6 mm Hg. This contrasts with a fall of only −4 mm Hg in the Hypertension Optimal Treatment study, 4.8 mm Hg in the Comparison of Amlodipine Versus Enalapril to Limit Occurrences of Thrombosis study and 2.4 mm Hg in Ongoing Telmisartan Alone and in Combination With Ramipril Global Endpoint Trial. This greater BP fall in our studies may explain why we got positive results, whereas the results of the Hypertension Optimal Treatment study, the Comparison of Amlodipine Versus Enalapril to Limit Occurrences of Thrombosis study and Ongoing Telmisartan Alone and in Combination With Ramipril Global Endpoint Trial study were a bit disappointing. Perhaps, a lesson to learn from both of our studies is that studies of extra BP reduction within the normal BP range ought to ensure the extra BP fall is at least in the 6 to 10 mm Hg range.

H J Simpson,1 S J Gandy,2 J G Houston,2 A D Struthers3

1Division of Medical Sciences, University of Dundee, Dundee, UK; 2Department of Radiology, Ninewells Hospital and Medical School, Dundee, UK
3Correspondence to Professor Allan D Struthers, Division of Medical Sciences, Centre for Cardiovascular & Lung Biology, Ninewells Hospital and Medical School, Mail Box 2, Dundee DD1 9SY, UK; a.d.struthers@dundee.ac.uk

Funding Other funders: Chief Scientist Office.

Competing interests None.

Ethics approval This study was conducted with the approval of the Tayside Committee on Medical Research Ethics and the Tayside Research and Development Consortium. It was conducted at Ninewells Hospital, Dundee, UK.

Provenance and peer review Commissioned; not externally peer reviewed.

REFERENCES