Rebuttal

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Dear editor,

We thank Smulders *et al*. for commenting on several aspects of the recently published guideline on the management of hypertensive crisis.[1] We cannot but agree that not all changes incorporated in the 2010 revision are evidence-based. This is stated as such in the guideline along with the motivation for the recommendation (and the – grade D – level of evidence). Most such changes were incorporated to concur with international guidelines. In the recent summary we have tried to select the most important changes and motivations of the guideline.[2] We welcome the opportunity to respond to the issues raised by Smulders and colleagues, who have some reservations about definitions and recommendations in the revised guideline.

The first issue raised by Smulders *et al*. involves the definitions for hypertensive urgency and emergencies, which -in contrast to the previous guideline- conform to international guidelines and literature. Smulders *et al*. suggest that hypertensive urgencies cannot be considered a hypertensive crisis because acute target organ damage is lacking. As stated in the summary, a hypertensive urgency

is essentially a diagnosis of exclusion which can only be made after ruling out acute organ damage (e.g. by ECG, funduscopy). However, despite the lack of acute organ damage these patients are still considered urgent because of their severe blood pressure elevation. To prevent acute organ damage, treatment with oral blood pressure lowering medication is recommended along with a brief period of observation. In general further treatment and analysis can take place at the GP's office or outpatient clinic. In the guideline and summary, hypertensive emergencies are not defined by the promptness by which blood pressure should be lowered, but by the recommendation to start intravenous therapy (under haemodynamic monitoring) to lower blood pressure to safe levels and prevent progressive organ damage. This definition is in line with international literature and guidelines and also includes hypertensive crisis with advanced retinopathy (with or without microangiopathic haemolysis or acute renal failure).

The second issue concerns the choice for intravenous blood pressure lowering therapy in favour of oral blood pressure lowering medication. The disadvantages of oral medication for the treatment of a hypertensive emergency are discussed in the guideline and include the slower onset of action and unpredictable blood pressure lowering efficacy. As recognised by Smulders *et al.* blood pressure reductions exceeding a MAP of 25% should in general be avoided with the exception of acute aortic dissection. There are no studies showing that this can be reliably achieved by oral medication. The hazard of vigorous lowering of blood pressure in patients with hypertensive encephalopathy and grade III/IV retinopathy is not only theoretical since excess blood pressure lowering has been associated with incident stroke and death (see guideline for references).

The third issue concerns the therapeutic management of a hypertensive urgency, i.e. patients presenting with a severe blood pressure elevation (BP >220/120 mmHg) who are suspected of a hypertensive crisis and lack signs of acute target organ damage. This includes patients with both acute or chronic blood pressure elevations for different reasons (e.g. anxiety, chronic uncontrolled hypertension, substance abuse). These patients are difficult to compare with the average hypertensive patient receiving combination therapy in a controlled trial. The treatment of a hypertensive urgency is not aimed at reaching target blood pressure, but at reducing excess risk associated with severe blood pressure elevations within an acceptable time-frame. Combined with the knowledge that blood pressure may lower spontaneously in a number of these patients the goal is to lower blood pressure without 'doing harm'. The available evidence, summarised in the guideline, shows that nifedipine retard has the most predictable blood pressure lowering efficacy without risk of hypotension in these situations. Because of the heterogeneous causes of a hypertensive urgency and the spontaneous blood pressure changes that occur in these patients in an emergency setting there is reason to recommend a brief period (at least two to three hours) of observation. This will help in making a definitive diagnosis regarding the nature of the blood pressure elevation next to allowing appropriate observations of the initial BP-lowering effect. Finally, in our experience and that of others, most patients presenting with a hypertensive crisis have not received or taken their medication in the weeks prior to their presentation. This suggests that even patients who were prescribed calcium antagonists may respond well to nifedipine retard, although evidence for this is lacking.

To conclude, we fully agree with the general recommendations pointed out by Smulders *et al.* that form the basis of our guideline and clinical practice in general. We look forward to their experiences after using the guideline in their practice.

REFERENCES

1. Beutler JJ, van den Born BJ, Gaillard CA, de Gooijer A, Kroon AA, van den Meiracker AH. Richtlijn Hypertensieve Crisis 2010.
2. van den Born BJ, Beutler JJ, Gaillard CA, de Gooijer A, van den Meiracker AH, Kroon AA. Dutch guideline for the management of hypertensive crisis – 2010 revision. Neth J Med. 2011;69:248-55.