LETTER TO THE EDITOR

Comment on summary of the updated Dutch guidelines for the management of hypertensive crisis

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Dear editor,

In the May issue of this journal, Van den Born and colleagues present the essentials of the updated Dutch guideline for the management of hypertensive crisis.[1] The guideline committee is to be commended for their work, as several aspects of the guideline have clearly been improved, compared with the previous 2003 version.[2] In a number of respects, however, the new guideline is unclear, and important changes in recommendations are neither evidence-based nor appropriately motivated. Our main concerns are as follows:

- In the 2003 guideline, hypertensive crises were distinguished in *emergencies* and *urgencies*. Both were characterised by acute organ damage, the difference between an *emergency* and an *urgency* being the time available for intervention (minutes in an *emergency*, hours in an *urgency*). In the updated guideline, a crisis is still defined by the presence of 'acute target organ damage', but the definition of urgency is 'severe hypertension *without* evidence of acute hypertensive organ damage'.[1] This is confusing at least, if not impossible. It suggests that *urgencies* are basically non-existent as part of hypertensive crises.
  Strange as the implicate abolishment of the hypertensive *urgency* may be, another choice that was made is actually compatible with this: all forms of acute organ damage now suddenly require intravenous therapy. This is advocated not only for retinopathy, but also for microangiopathy and acute renal failure. Nonetheless, the updated guidelines state that effective therapy in these conditions may take up to a few hours. Hence, as opposed to the 2003 guidelines, we now appear to define two types of *emergencies*: 'emergency *emergencies*' and 'urgent *emergencies*'.
- We fail to understand why intravenous therapy is required in all situations where at least several hours are available for blood pressure lowering. Some argue that intravenous therapy is safer in terms of sudden excessive blood pressure drops. Although this may be true theoretically, unstable blood pressure on intravenous drugs is unfortunately a frequent reality. We think that evidence showing that intravenous therapy provides both a safer time course and degree of blood pressure lowering in daily practice is lacking. Also, intravenous therapy necessitates a subsequent transition period to oral drugs, for which no proven safe algorithms are available. Again, unstable blood pressure during this transition period is not infrequent in our clinical experience. Finally, a crucial point to be made is that short-term lowering of blood pressure by >25% should *never* be considered safe. Only in theory is this particularly unsafe in situations of acute organ damage, especially grade III-IV retinopathy and/or encephalopathy.
- What is now defined as a hypertensive *urgency* is basically, as acknowledged by the committee, severe hypertension without acute organ damage. International guidelines on the management of hypertension advocate the use of long-acting antihypertensives in these patients. In fact, in severe hypertension, starting with long-acting combination tablets is recommended.[3] What is the reason for stepping away from these recommendations? Why propose the use of nifedipine-retard tablets? As a result of their shorter half-life, blood pressure instability, particularly after discharge, is a real danger. Also, clinical experience is that moderate doses of long-acting antihypertensives, even as combination tablets, rarely if

ever cause an excessive drop in blood pressure, which is why international guidelines actually recommend this strategy.[3]

Also, observation for a few hours is advocated in patients with severe hypertension without signs of acute organ damage (i.e. an *urgency*). Although this may be harmless in itself, we do not see the justification for this, let alone for defining an absolute maximum level of blood pressure allowing discharge.

- Finally, the guideline would have been more complete if recommendations had been made for patients who are already on antihypertensives, which is the case for a large proportion of those presenting with severe hypertension. Is nifedipine for example still treatment of choice in those already taking calcium blockers?

In conclusion, the updated guidelines are a step forward in some respects, but a step backward in several others. We contemplated on this when we had to decide what to teach our residents. We decided to recommend they read the guideline carefully, but take terminology and classification with a couple of grains of salt. We teach them that a common mistake is to overestimate the benefit and underestimate the risk of acute lowering of blood pressure. Blood pressure should be lowered as acutely as the associated clinical condition may reasonably be expected to deteriorate. When the acute benefit is less clear, such as in the case of grade III/IV retinopathy without visual disturbance, they should proceed with the attitude of 'first, do no harm'. We thus teach that central to the approach to patients with severe hypertension is the question of whether there are signs of acute organ damage and, if so, what degree of hurry is dictated by common sense and epidemiological evidence is leading. We do not encourage routine use of intravenous drugs when acute blood pressure lowering is not called for, nor do we support the use of short-to-medium long acting drugs for severe hypertension without acute organ damage.

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