Systolic hypertension in young adults: spurious definition of a genuine condition - Reply

Jean-Marie Krzesinski and Annie Saint-Remy

Nephrology Unit, CHU Sart Tilman, University of Liège, Liège, Belgium

We would like to thank McEniery et al. [1] for their criticisms about our editorial comment on spurious systolic hypertension [2]. Their remarks allow us to realize that a misunderstanding arose concerning the definition we used for the condition: 'spurious systolic hypertension is defined by a high brachial systolic blood pressure but a normal aortic systolic pressure estimated indirectly by a non-invasive technique using the SphygmoCor device'. The term 'normal' is probably unsuitable.

Hulsen et al. [3] defined their 'normal' central (ascending aortic) pressure on the basis of their own distribution of non-invasive blood pressure measurements using percentiles as a cut-off point. They considered that central systolic pressure was normal if its value was smaller than the 90th percentile. Thus, they defined a 'normal' value as a central systolic blood pressure < 124 mmHg for men and < 120 mmHg for women. Other authors, such as O'Rourke et al. [4], proposed that central systolic pressure was normal for values lower than 126 mmHg and Mahmoud and Freely [5] even proposed this parameter to be equal to or lower than 116 ± 1 mmHg.

We agree with McEniery et al. [1] that a definition of normal central blood pressure, without comparative invasive measurement, is difficult or even impossible. In such a situation, we also agree that any definition of 'normal' remains arbitrary.

When we spoke about a 'normal' value in the definition of aortic blood pressure of patients with spurious systolic hypertension, it was of course in relation to definition of Hulsen et al. [3].

As previously mentioned by Cockcroft et al. [6], it would be more useful to define 'normal' amplification. In their editorial, amplification for people aged 20-25 years was < 26 mmHg. In the present Dutch study, the difference between brachial and central pulse pressures was 28.5 mmHg in the spurious group, 24.6 mmHg in the hypertensive group and 21.2 mmHg in the normotensive group. Thus, in spurious systolic hypertensive patients, there is indeed an exaggerated amplification explaining the higher brachial systolic blood pressure, but this is associated with a significantly higher central pulse pressure compared to normotensive people.

However, the explanation of this phenomenon is not yet clear. Thus, it is mainly a problem of exaggerated amplification of the peripheral waveform, which could be analysed, as proposed in our editorial comment, by measuring cardiac output and stroke volume to eliminate the role of hyperkinetism. A white-coat hypertension phenomenon should also be eliminated by 24-h ambulatory blood pressure monitoring recording.

Of course, we fully agree that the spurious systolic hypertension group in the study Hulsen et al. [3] presented a significantly higher central blood pressure than the true normotensive group. In that way, they present at least an intermediate coronary risk. The latter must be evaluated by accurate long-term studies. The large population of young people included in the Dutch publication will certainly allow such an evaluation.

To conclude, this curious characteristic of some young 'hypertensive' (according to brachial blood pressure) cases is noted in 10-17% of individuals. It deserves further precise analysis to better characterize the reasons for such increased blood pressure amplification between central and peripheral sites.

However, great caution is warranted. We do not know whether such a condition will be free of complications in the future and we fully agree with the proposition of Pickering [7], who wisely stated: 'the best advice is to follow these individuals carefully, but not to start them precipitately on medication.'
References


