New problems raised by increased pulse pressure

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This editorial refers to 'Importance of arterial pulse pressure as a predictor of coronary heart disease risk in PROCAM'† by G. Assmann et al., on page 2120

There is now ample evidence that brachial artery pulse pressure (PP) is an independent predictor of cardiovascular (CV) outcomes, principally in subjects aged >59. This finding has been observed not only in European but also in American and Asiatic populations. The results of PROCAM, which involves a 10-year follow-up of more than 20,000 subjects, allow new prospective views on this finding.1

Initially, studies on PP were focussed on two principal aspects. First, as the incidence of aortic insufficiency is nowadays consistently reduced in patients with CV diseases, the meaning of increased PP remained almost unknown till the recent years, particularly in the elderly. Thus, the finding that increased PP, a parameter strongly linked to arterial rigidly in old people, was a predictor of CV risk was an interesting advance in medical semiology. Second, the ability to distinguish two different components of blood pressure (BP), a steady component, represented by mean arterial pressure (MAP) and a pulsatile component, represented by PP, was quite important to consider in the pathophysiology of hypertension. Indeed, although MAP is practically unchanged along the totality of the arterial tree, systolic BP (SBP) and PP are consistently higher in peripheral than in central arteries. This physiological difference, which relates to arterial stiffness and mainly to wave reflections, is close from 14 mmHg but tends to be reduced with age. So defined, the presence of systolic and PP amplification is susceptible to greatly modify the diagnosis and the clinical management of hypertension.

Surprisingly, these two different aspects of the understanding of PP and mostly PP amplification, did not raise major questions in the medical literature. The interest on PP was focussed on a single question: was brachial PP susceptible to take the place of brachial SBP to determine CV risk and even to discriminate hypertension. Some studies did not confirm that PP might give distinct information from those provided by increased SBP.2 For instance, it was almost completely forgotten that increased PP could be widely observed even in non-hypertensive populations and did not relate to peripheral vascular resistance. Indeed, PP points to pulsatile stress, aortic rigidity, and finally to coronary risk. In addition, at this same period, when SBP and PP were compared in large populations in order to determine CV risk assessment, several methodological problems were still difficult to resolve. First, from a pathophysiological view point, it is important to define statistical methods enabling to determine MAP and PP independently. For this, the most adequate was in fact the principal component analysis.3 Second, for any clinician, it seems obvious that increased PP mainly relates to the older portion of the population. This situation requires that long-term follow-up could be obtained. For such reasons, meta-analysis seemed easier to establish. The curves relating BP to CV risk were frequently extrapolated at their terminal portions, i.e. in younger and older subjects. In such conditions, the long-term follow-up provided by PROCAM and the focus of this study on coronary risk enable to discriminate the most modern questions raised by increased PP.

First, increased PP influences CV risk, but the finding is observed only in old populations, in which increased aortic rigidity is the principal determinant of PP, i.e. in subjects without congestive heart failure and with preserved ejection fraction. The recent findings on the validity of PP as a CV risk factor in subjects with congestive heart failure and mostly, the evidence that increased aortic stiffness is an independent predictor of CV risk more powerful than PP itself clarify such questions nowadays.

Second, in subjects submitted to cardiac catheterization and mostly to coronaryography, it has been suggested that increased central PP is superior to brachial PP for the prediction of coronary risk.4 A clear cut response to this interesting proposition has not yet been ruled out.

Finally, in subjects with preserved ejection fractions, it has been reported that the level of central PP is proportional to the severity of coronary atherosclerotic alterations, as judged from the number of coronary stenosis in each individual.5 In contrast, increased PP, and not increased SBP or DBP, is highly linked to the severity of the coronary ischaemic disease. This finding is of primary importance as, in patients with coronary insufficiency, the coronary reserve is markedly reduced, indicating an impaired ability to cause arteriolar dilatation. Under this condition, the exclusive haemodynamic factor susceptible to maintain the coronary perfusion is aortic DBP. Because aortic PP is widely increased in this population as a consequence of increased aortic
rigidity, and because increased PP involves both an increase of SBP and a decrease of DBP, aortic elasticity becomes the critical parameter maintaining coronary perfusion. Thus, it is easy to understand that a further DBP reduction due to an associated lowering of vascular resistance (possibly due to antihypertensive agents) may be detrimental for the heart.

The maintenance of an adequate coronary perfusion is a critical problem in any subject treated for hypertension. Indeed, antihypertensive drug treatment is able to reduce markedly DBP (<90 mmHg) in more than 80% cases, whereas SBP remains frequently elevated (>140 mmHg) resulting in an increase in PP, further accentuated by age and resulting in increased aortic rigidity. Thus, only drug treatment acting selectively on large vessels may contribute in the long-run to improve CV risk in hypertensive subjects through selective reduction of SBP and maintenance or even increase of DBP.5

In conclusion, increased PP raises major problems in patients with atherosclerosis and hypertension. It seems relevant nowadays that, during the haemodynamic procedure involving coronarography, dilatation, and stent indications, several associated investigations could be proposed: (i) to measure intra-aortic BP, and mainly PP, and to compare the validity of such determinations with non-invasive brachial BP measurements, (ii) to relate the PP levels to the presence of structural changes of coronary arteries, and mainly to the number of arterial stenosis, and (iii) to determine whether intra-aortic or non-invasive measurements or both are the long-term significant predictors of CV risk. It is evident that such questions require multicentric studies. It is reasonable to wish that the response to these questions may be obtained in European countries, where the concept of increased PP as well as its predictive value was primarily described.

References