Arterial pulse pressure increases according to diabetes duration, independently of age in patients with type 1 diabetes

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Background and aims:
Arterial pulse pressure (PP) reflects arterial stiffness and is considered as an independent cardiovascular risk factor. However, studies regarding PP in type 1 diabetic patients are scarce. The aim of the study was to investigate the influence of the duration of type 1 diabetes on PP, as well as to correlate PP with the quality of blood glucose control and the presence of microalumuninuria (µA) and/or drugs blocking the renin-angiotensin system.

Material and methods: A total of 159 type 1 diabetic patients (20-60 years) were evaluated with a continuous noninvasive arterial blood pressure monitoring (Finapres®). Recordings were performed in standing position (1 min), in squatting position (1 min), and again in standing position (1 min). Presented data correspond to average PP (systolic - diastolic arterial pressure) values calculated during the overall recording in the 3 positions. Blood glucose control was assessed by concomitant HbA1c levels. Thirty-one patients were treated with an ACE inhibitor or an angiotensin AT1 blocker (ARB) because of µA antecedent. Subjects were retrospectively separated according to: 1) diabetes duration: group 1: < 10 years (n = 39); group 2: 11-20 years (n = 45); group 3: 21-30 years (n = 57); and group 4: > 30 years (n = 18); 2) concomitant HbA1c levels (< or ≥ 8 %); 3) µA (< 30 or ≥ 30 mg/l); and 4) current treatment with ACE inhibitor or ARB (yes or no). In order to differentiate the effects of duration of diabetes from the effect of increasing age, healthy subjects were used as controls and matched for age, sex and body mass index (n = 30 in each subgroup).

Results: PP was higher in men than in women, in both diabetic (58 ± 15 vs 50 ± 14 mm Hg; p < 0.001) and non-diabetic (55 ± 14 vs 47 ± 12 mm Hg; p < 0.001) subjects. PP increased progressively throughout the 4 subgroups according to diabetes duration (47 ± 16 vs 51 ± 13 vs 59 ± 14 vs 62 ± 12 mm Hg, respectively). There was a marked difference between groups 1-2 and groups 3-4 (49 ± 14 vs 59 ± 14 mm Hg; p < 0.00002). Such a progressive PP increase was not observed in non-diabetic subjects in the same age range (mean of 35 years for groups 1-2 vs 46 years for groups 3-4): 51 ± 12 vs 50 ± 15 mm Hg; NS. Percentage of subjects with PP > 60 mm Hg was similar in diabetic and non-diabetic individuals in groups 1-2 (27 vs 25 %, NS); however, in groups 3-4, the prevalence of subjects with PP ≥ 60 mm Hg was significantly higher in diabetic patients than in controls (44 vs 27 %, p < 0.05). The difference in PP between the four groups of diabetic patients, already present in standing position, was amplified in squatting position: the coefficient of correlation between individual PP values and diabetes duration data increased from r = 0.289 (p < 0.01) while standing to r = 0.362 (p < 0.001) in squatting position. PP was similar in patients with HbA1c < 8 % (54 ± 14 mm Hg) or ≥ 8 % (55 ± 16 mm Hg). No significant differences were observed in patients with µA (57 ± 17 mm Hg) vs without µA (54 ± 14 mm Hg) and in patients receiving (56 ± 14 mm Hg) or not receiving (54 ± 15 mm Hg) an ACE inhibitor or an ARB.

Conclusion: Type 1 diabetes was associated with a progressive increase in PP according to the duration of the disease, in an age range where no significant influence of age was observed in a non-diabetic population. PP was not significantly influenced by concomitant HbA1c levels, and no variation in PP was associated with µA or blockade of the renin-angiotensin system.