Reflex Control of Circulation in the Elderly

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Summary. Baroreceptor control of heart rate is markedly reduced in elderly subjects. However, the effects of aging on baroreceptor control of blood pressure and on the vascular and neurohumoral influences of volume cardiopulmonary receptors are unknown. In this paper we report evidence that in both conscious rats and humans aging is associated with a fall in the baroreceptor ability to rapidly cause blood pressure changes, but that the more long-term carotid baroreceptor control of blood pressure remains similar to that observed in younger individuals. Early and late cardiopulmonary receptor modulation of vascular resistance is impaired by aging, which also reduces the influence of this reflex on renin secretion. These dynamic and steady-state alterations in reflex cardiovascular control account for several hemodynamic abnormalities of the advanced age.

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Orthostatic hypotension, postprandial hypotension, and other unusual hypotensive events are more frequent in elderly than in young or middle-aged subjects [1-3]. This has generated the hypothesis that reflexes involved in blood pressure homostasis undergo an impairment with aging, failing to compensate with adequate vasoconstriction and cardiac stimulation to environmental disturbances or behavioral cardiovascular influences, leading to a reduction in cardiac output and/or vasodilatation [4].

Support for this hypothesis has been obtained by the observation that the bradycardia induced by increasing blood pressure and stimulating baroreceptors through IV administration of phenylephrine decreases progressively with aging and that this occurs also for the tachycardia induced by reducing blood pressure and deactivating baroreceptors through IV administration of a vasodilator [5-7]. However, this provides only limited information on the relationship between age and reflex cardiovascular control, because alterations of the baroreceptor-heart rate reflex may not reflect changes in the primary baroreflex function, i.e., blood pressure control [7-9].

Furthermore, no information exists as to whether aging affects the important reflex originating from volume receptors in the cardiopulmonary region. Like the baroreflex, this reflex is responsible for a tonic inhibition of sympathetic activity to peripheral circulation [10,11]. It is also responsible, however, for restraint of sympathetic tone to the kidney and the release of renin, thereby contributing to both blood pressure and blood volume homeostasis [12-14].

We have addressed these two points and this paper summarizes the results.

Aging and Baroreceptor Control of Blood Pressure

Baroreceptor control of blood pressure was studied by increasing or reducing carotid transmural pressure through application of 2 minutes of negative and positive pressures within a neck collar, thereby stimulating or unloading carotid baroreceptors [15]. The positive and negative neck pressures were corrected for the loss of pressure transmission through the neck tissues [15], and the reflex blood pressure responses (arterial catheter) were assessed by calculating mean values after 5-15 seconds and in the last 30 seconds of the stimulus (early and late or steady-state responses, respectively). The study was performed in four groups of subjects whose blood pressures were matched and whose ages ranged from less than 36 to more than 54 years [16]. As shown in Figure 1, carotid baroreceptor stimulation induced a fall in blood pressure that was equally pronounced in the early and steady-state phase. Conversely, carotid baroreceptor unloading induced a blood pressure rise that was smaller in the early as compared to the steady-state phase. The early and steady-state hypotensive responses were similar across the age spectrum. This was the case...
also for the steady-state pressor response. In contrast, the early pressor response showed a progressive decrease as age increased. In the oldest group, this response was only 47% of the response measured in the youngest group.

Thus, aging does not affect the ability of the carotid baroreflex to oppose a blood pressure rise. It also leaves unaltered its ability to ultimately correct for a blood pressure fall. However, the speed with which this correction is achieved is reduced, making the antihypotensive action of this mechanism more sluggish.

This was confirmed in conscious normotensive rats defined as young, adult, and old on the basis of an age of 5.8 ± 0.1 weeks, 15.0 ± 0.3 weeks, and 82.0 ± 2.0 weeks [17,18]. In line with the impairment of the baroreceptor control of the heart observed in aged humans [5–7], the reflex changes in heart rate obtained by stimulating and deactivating baroreceptors through the pressor response to IV phenylephrine and the depressor response to IV nitroprusside, respectively, were progressively less pronounced from young to adult and old rats (Figure 2). This was accompanied by an age-related reduction in the blood pressure rise measured a few seconds after the deactivation of the carotid baroreceptors induced by occlusion of the common carotid arteries. In contrast, the maximal and sustained blood pressure rise achieved during this maneuver showed no substantial difference in the three groups (Table 1). Similar results were obtained in rats in which one carotid sinus was denervated and common carotid occlusion was performed on the contralateral side only, to preserve brain perfusion and to avoid any dependence of the blood pressure rise on cerebral ischemia [18].