SHORT COMMUNICATION

Heart-rate changes on standing in elderly patients with orthostatic hypotension

N. J. WHITE
Nuffield Department of Medicine, John Radcliffe Hospital, Headington, Oxford, U.K.

(Received 30 October 1979; accepted 5 February 1980)

Summary

1. The heart-rate changes on standing were studied in 16 elderly patients with idiopathic orthostatic hypotension and compared with those of 20 controls. The mean age of both groups was 78 years, and all were in sinus rhythm.

2. The patients with orthostatic hypotension differed from the control patients in having no early peak in heart rate, and a significantly smaller and slower rise in heart rate within the first 40 s after standing ($P < 0.05$).

3. It is concluded that there is a failure of the autonomic reflex in elderly patients with orthostatic hypotension, which may contribute towards their symptoms.

Key words: geriatrics, heart rate, orthostatic hypotension.

Introduction

Orthostatic hypotension is common in the elderly (Norns, Shock & Yiengst, 1953; Caird, Andrews & Kennedy, 1973). Johnson, Smith, Spalding & Wollner (1965) reported an incidence of 17% in a geriatric inpatient population, significant orthostatic hypotension being defined as a fall in systolic blood pressure of $\geq 20$ mmHg.

Both inelasticity of the peripheral vasculature (Gross, 1970; Thulesius, 1976) and a defect in the autonomic nervous control of vascular tone (Johnson & Spalding, 1974) have been considered the pathogenic abnormality. As a measure of autonomic reflex function, the changes in heart rate that occur on standing were studied in elderly patients with symptomatic orthostatic hypotension.

Methods

Subjects

The subjects of this study were inpatients under the care of the geriatric department. They were aged between 67 and 93 years. All patients were in sinus rhythm. The 20 controls were patients of similar age range, who had complained of occasional giddiness on standing but no postural fall in blood pressure was found. The 16 patients all had significant orthostatic hypotension (systolic fall $\geq 20$ mmHg, diastolic fall $\geq 10$ mmHg repeatedly on ward testing). Patients with dehydration, diabetes or other disease associated with autonomic failure, or those who were taking drugs known to cause orthostatic hypotension, were specifically excluded.

Procedure

Pulse rates were recorded on a routine direct-writing ECG machine at a rate of 50 mm/s. Blood pressure was recorded with a sphygmomanometer. Recordings were initially made with the patients lying relaxed on their bed, and then continuously standing. Blood pressure was recorded at 1 min intervals. Instantaneous pulse rates were calculated from the ECG-derived R–R intervals. These
Fig. 1. Changes in normalized heart rate (instantaneous heart rate derived from ECG R–R interval/preceding lying heart rate) on standing upright. Mean ± 1 SEM values are shown: ●, 16 elderly patients with idiopathic orthostatic hypotension; ○, 20 controls. The patients with orthostatic hypotension have a significantly smaller ($P < 0.05$) increment in heart rate until 40 s has elapsed since standing.

were sequentially plotted against the time elapsed since the patient became upright. The pulse rates at fixed time intervals were derived from these individual graphs. The two groups were compared by Student’s $t$-test.

Results

There was a significant correlation between lying heart rate and subsequent increments at all times (controls: $r = 0.70$, $P < 0.01$; patients: $r = 0.79$, $P < 0.01$). Heart rates were therefore normalized by division of the instantaneous pulse rate by the preceding resting value. The resulting ratio was plotted against time (Fig. 1). There was no correlation between changes in pulse rate and the magnitude of orthostatic hypotension, which ranged from a fall of 20/10 to 80/35 mmHg.

Pattern of heart-rate changes on standing

The mean normalized heart rates are compared in Fig. 1. After standing the control patients showed a rapid rise to peak heart rate followed by decline within the first 2 min. In contrast the patients with orthostatic hypotension had significantly smaller increments in heart rate and had no early peak. There was a significant difference ($P < 0.05$) between the two groups until 40 s after standing.

Time to reach peak rate

Three of the 20 controls, and eight of the 16 patients, failed to reach peak heart rate within 1 min. Comparison of the remainder showed that the mean time to peak rate in controls ($n = 17$) was 12.06 s and in the patients ($n = 8$) it was 28.88 s ($P = < 0.005$).

Discussion

The results of this study show that elderly patients with symptomatic orthostatic hypotension have a reduced and delayed heart-rate response to standing. The process of standing upright in the unsteady elderly patient may take several seconds and with regard to timing of recordings is therefore less accurate than the more commonly employed tilt-table method of investigation. The latter procedure is, however, entirely passive, without any subjective sense of voluntary effort, and fails to produce the contraction of the leg muscles that aids venous return and reduces pooling in the legs.
In normal people there is a transient fall in blood pressure on standing (Hill, 1895). The initial pooling of blood in the legs is compensated by both redistribution of the intrathoracic circulation to maintain diastolic filling and by a reduction in end-systolic volume; nevertheless stroke output declines after the first few seconds (Clement, 1976). It has been suggested that the early fall in blood pressure in the elderly, and the exaggerated fall observed in patients with cerebrovascular disease, reflects rigidity and inelasticity of blood vessels as it occurs before baroreceptor-mediated circulatory reflexes have begun to act (i.e. before 8 s) (Gross, 1970). Hypotension persisting beyond this time, however, is due to failure of reflex vasoconstriction.

In young normal subjects the heart rate rises on standing to reach a peak approximately 15 beats after standing, followed by a relative bradycardia at 30 beats (Ewing, Campbell, Murray, Neilson & Clarke, 1978). The elderly patients in this study, in contrast to controls of similar age, did not show this immediate rate response. This points to a failure of the autonomic reflex, and presumably has the same pathophysiology as the orthostatic hypotension it accompanies. Baroreceptor function declines with age (Gribben, Pickering, Sleight & Peto, 1971), and other autonomic defects may occur in the elderly (Wollner & Spalding, 1978) suggesting both peripheral and central abnormalities.

The symptoms of orthostatic hypotension are due to impairment of cerebral perfusion. On standing the fall in stroke output will, if inadequately compensated by reflex tachycardia, reduce cardiac output. In these elderly patients the fall in both mean arterial pressure and cardiac output must diminish the blood supply to the brain. Thus this inadequate heart rate response to standing may contribute to the symptoms of orthostatic hypotension in the elderly.

Acknowledgments

My thanks to the physicians in the department of Geriatric Medicine for allowing me to study their patients.

References


