Vascular compliance is reduced in vascular dementia and not in Alzheimer’s disease

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Abstract

Objective: to determine whether functional changes in the vasculature differ between Alzheimer’s disease (AD) and vascular dementia (VAD).

Design: we determined vascular stiffness in patients with a clinical and radiological diagnosis of either AD or VAD and compared them to normal age- and sex-matched controls.

Methods: In all, 16 patients with late onset AD, 13 subjects with VAD and 16 age- and sex-matched controls were recruited to this study. Central arterial compliance (CAC), augmentation index (AI) and pulse wave velocity (PWV) (measures of arterial stiffness) were measured.

Results: the mean age was 77.7 ± 8.3 years (mean ± SD) in the AD group, 79.7 ± 8.9 years in the VAD group and 76.4 ± 6.9 in the controls (P = 0.44). CAC was significantly lower in subjects with VAD compared to both the AD and the control groups (0.57 ± 0.46 ml/mm Hg versus 1.12 ± 0.57 and 1.1 ± 0.47 ml/mm Hg respectively, P = 0.01). AI was significantly higher in the subjects with VAD compared to both the AD and the control groups (13.3 ± 9.0 versus 3.5 ± 11.4 and 4.2 ± 9.7% respectively, P = 0.03). PWV in the muscular and elastic arteries were not statistically different between the three groups but tended to be highest in the VAD group for carotid-radial measurements.

Conclusions: the reduced CAC and increased AI in VAD subjects indicate that the disease process is associated with less vascular compliance of the large elastic arteries in these patients, but not in patients with AD.

Keywords: aortic compliance, dementia, ageing, blood pressure, vascular, elderly

Introduction

Dementia is defined as a decline in cognitive function that may result in an impaired function in daily living [1]. The prevalence of dementia is 1% in 60-year-olds and rises to 30% in the 85-year-old age group [1]. Vascular disease and its principal risk factors have been found in population studies to be important associations of the two most common causes of age-associated dementia, Alzheimer’s disease (AD) and vascular dementia (VAD), respectively [1, 2]. A diagnosis of VAD is made if dementia results from ischaemic, haemorrhagic, or ischaemic-hypoxic brain lesions especially in the presence of a history of hypertension, ischaemic heart disease, transient cerebrovascular, and peripheral vascular disease [2–4].

AD is a gradually progressive dementia of insidious onset, which occurs in the absence of other diseases that could account for the cognitive deficits. Although certain diagnostic criteria emphasise that AD is a diagnosis of exclusion with regard to vascular disease; vascular factors may also have a role in late-onset AD [1, 2].

Decreased compliance of the central arterial vasculature changes the blood pressure and flow dynamics with significant impact on the cardiac contractility, coronary and cerebrovascular perfusion [5]. Other measures related to vascular compliance include pulse wave velocity (PWV) and augmentation index (AI).

Recent studies have shown that arterial stiffness is associated with cognitive decline, and that for each 2-m/s increase in PWV there is an increased risk of both types of dementia, the adjusted odds ratio (OR) being 1.73 (95% CI 1.27–2.47) for AD and 3.52 (95% CI 1.87–8.05) for VAD [6]. Scuteri et al. showed that worsening arterial stiffness as measured by PWV was more evident in subjects with cortical atrophy than in patients with subcortical microvascular lesions or controls (P < 0.05), and that higher PWV was associated with cognitive impairment, greater personal dependency, independent of other major modifiable
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Cardiovascular risk factors and medications use [7]. Both AD and VAD have significant vascular risk factors [8, 9]. A recent study by Bateman et al. showed that early VAD is characterised by normal cerebral blood flow and increased pulsation, while early AD is characterised by reduced blood flow and reduced compliance [10]. Nagai et al. showed a relationship between PWV and cognitive decline in non-VAD [11].

The objective of this study was to assess arterial compliance (AC) in subjects with AD and VAD compared to age- and sex-matched controls.

Methods

Subjects

Patients attending the Hammersmith Hospitals Trust Elderly Care Clinics were asked to volunteer for the study if they fulfilled the inclusion and exclusion criteria. All patients and their carers read the information sheet provided before giving verbal and written consent to participate in the study, which was approved by the Hammersmith Hospitals Ethics Committee. Healthy control volunteers were recruited from the community by advertisements placed in local newspapers and community centres. As many as 16 patients with dementia of the Alzheimer’s type (mean age 77.7 ± 8.3 years) and 13 patients with VAD (mean age 79.8 ± 5.3 years) fulfilled the criteria and were recruited to this study. They were compared to 16 age- and sex-matched controls (76.4 ± 6.9 years).

Inclusion criteria

The clinical diagnosis of AD was made in accordance with the DSM-III-R [12] and/or NINCDS-ADRDA criteria [13], and cranial computed tomography (CT) or magnetic resonance imaging (MRI) evidence excluded most other potential causes of dementia. VAD was diagnosed using DSM-III-R and/or NINCDS-ADRDA criteria, a score of seven points or more on Hachinski’s ischaemic score [14, 15] and cranial CT or MRI evidence of relevant cerebrovascular changes. Blood screen results at the time of diagnosis, including full blood count, vitamin B₁₂, folate levels and thyroid function tests, were reviewed to exclude metabolic causes of cognitive impairment. The control group was also clinically investigated to exclude the above diagnoses.

All the subjects were over 60 years of age, had no acute or chronic medical illness and were not on atrial fibrillation and not taking hormone replacement therapy. The control group had to fulfil the above criteria and had no evidence of cognitive impairment.

Procedures

Baseline characteristics were measured (Table 1). Initial cognitive testing was done using the Mini-Mental State Examination (MMSE) [16].

Central arterial compliance (CAC)

CAC was measured by simultaneous recording of carotid artery pressure wave, using a Miller Micro-tip pressure transducer (Miller Instruments, TX, USA); applanation tonometry and carotid flow recorded using Doppler probe placed in the suprasternal notch. Pressure waves recorded by applanation tonometry from carotid waveforms were calibrated against the diastolic blood pressure and mean brachial artery blood pressure. The average of three blood pressure readings at the brachial artery taken at 5-min intervals, in the right upper arm of the supine subject, was performed using an Omron HEM-705CP automatic blood pressure recording unit. The integral mean of the carotid pressure waveform using the area under the curve was assigned to measure the brachial mean pressure and the diastolic pressure was applied to the end-diastolic point on the carotid waveform to allow calculation of the carotid systolic pressure and central pulse pressure.

Pressure recordings and Doppler flow were measured for 30 s and analysed over 10 representative cardiac cycles, using a custom written programme [17, 18]. The average of these values was calculated to obtain a mean measurement for each subject. Central arterial compliance (CAC) was calculated using the area under the diastolic curve as described previously [18, 19].

Augmentation index (AI)

AI is an indirect measure of arterial stiffness which is known to increase with age. It was measured from the following formula: augmentation pressure (AP) divided by pulse pressure × 100. AP is a measure of the contribution that the wave reflection makes to the systolic arterial pressure, and is measured by the reflected wave coming from the periphery to the centre. Reduced compliance of the elastic arteries results in an earlier return of the wave reflection arriving in systole. AP was calculated from the difference between the first and second peaks of the aortic waveform. Applanation tonometry was performed using SphygmoCor (AtCor, Australia) over the right radial and carotid arteries, with the subject supine using the tonometer (Miller inc., Houston, TX, USA) to compress the carotid and radial arteries enough to record a pulse trace [20].

Pulse wave velocity (PWV)

PWV in the elastic arteries (carotid–femoral region) and the muscular arteries (carotid–radial and femoral–dorsalis pedis regions) was determined using the Complior (Colson Medicals, Paris) PWV recording unit [21]. Two sensors were positioned, one at the base of the right common carotid artery and one over the radial artery, and similarly over the right common carotid artery and the right femoral artery and the ipsilateral dorsalis pedis, thereby continuously measuring pulse wave signals. Distances between the two applanation sites were measured.
Dementia and vascular compliance

Table 1. Patient characteristics

<table>
<thead>
<tr>
<th></th>
<th>VAD (n = 13) (mean ± SD)</th>
<th>AD (n = 16) (mean ± SD)</th>
<th>Controls (n = 16) (mean ± SD)</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (years)</td>
<td>79.7 ± 5.3</td>
<td>77.7 ± 8.3</td>
<td>76.4 ± 6.9</td>
<td>0.35</td>
</tr>
<tr>
<td>Gender (male/female)</td>
<td>5/8</td>
<td>8/8</td>
<td>8/8</td>
<td>—</td>
</tr>
<tr>
<td>BMI (kg/m²)</td>
<td>23.7 ± 4.5</td>
<td>27.7 ± 5.0</td>
<td>25.6 ± 3.2</td>
<td>0.06</td>
</tr>
<tr>
<td>Cholesterol (mmol/l)</td>
<td>5.5 ± 0.7</td>
<td>5.0 ± 0.7</td>
<td>5.4 ± 0.9</td>
<td>0.27</td>
</tr>
<tr>
<td>Glucose (mmol/l)</td>
<td>5.5 ± 0.7</td>
<td>5.0 ± 0.7</td>
<td>5.4 ± 0.9</td>
<td>0.27</td>
</tr>
<tr>
<td>Systolic BP (mm Hg)</td>
<td>145.5 ± 20.2</td>
<td>127.1 ± 24.8**</td>
<td>136.8 ± 20.9</td>
<td>0.09</td>
</tr>
<tr>
<td>Diastolic BP (mm Hg)</td>
<td>75.2 ± 8.5</td>
<td>75.3 ± 11.2</td>
<td>78.6 ± 10.1</td>
<td>0.72</td>
</tr>
<tr>
<td>Mean BP (mm Hg)</td>
<td>98.5 ± 11.4</td>
<td>92.6 ± 15.0</td>
<td>98.1 ± 13.0</td>
<td>0.44</td>
</tr>
<tr>
<td>Pulse pressure (mm Hg)</td>
<td>70.2 ± 15.7</td>
<td>51.8 ± 16.9**</td>
<td>58.4 ± 15.0</td>
<td>0.01</td>
</tr>
<tr>
<td>Pulse rate (beats/min)</td>
<td>70.8 ± 13.7</td>
<td>67.7 ± 12.0</td>
<td>68 ± 8.6</td>
<td>0.77</td>
</tr>
<tr>
<td>MMSE (out of 30)</td>
<td>19.7 ± 6.1</td>
<td>19.7 ± 6.1</td>
<td>—</td>
<td>0.44</td>
</tr>
</tbody>
</table>

The baseline characteristics of the three groups. VAD, vascular dementia; AD, Alzheimer’s disease; Control, age- and sex-matched controls; BMI, body mass index; MMSE, Mini-Mental State Examination; BP, blood pressure.

* Significant difference vascular dementia subjects versus control group.
** Significant difference vascular dementia versus Alzheimer’s group.

as a straight line between these points on the surface above the artery, using a tape measure. The time interval between the feet of two simultaneously recorded waves at the two applanation sites was determined, and PWV was calculated as the ratio of the distance to transit time (distance/time) in metres/second (m/s).

Statistical analysis

The CAC and PWV (carotid–femoral) data sets were found to be skewed and log transformations were undertaken. Measurements of CAC, AI and PWV between the three groups were similarly compared using one-way analysis of variance (ANOVA). Post hoc analysis using least significant difference was used to compare the measurements between any two groups. Multiple regression, by stepwise elimination, was undertaken to adjust for significant covariates of each of the AC measurements. Type of dementia, gender, age, body mass index (BMI), systolic blood pressure, diastolic blood pressure, heart rate, smoking status and vasodilator therapy [β-blockers, calcium-channel blockers (CCB), glyceryl trinitrate (GTN) and angiotensin converting enzyme (ACE) inhibitors] were considered as covariates in performing these tests.

Results

The baseline characteristics of the three groups were very similar except for pulse pressure and systolic pressure (Table 1). Comparisons of baseline clinical characteristics between the three groups were made using ANOVA (Table 2). Appendix 1 in the supplementary data at Age and Aging online, shows the medications taken by the three groups included in the study. The three groups were closely matched for age, gender, BMI, pulse rate and diastolic blood pressure. However, pulse pressure, a surrogate measure of vascular stiffness, was higher in the VAD group (70.2 ± 15.7 mm Hg, mean ± SD) than in both the AD patients (51.8 ± 16.9 mm Hg, P < 0.01) and the controls (58.4 ± 15 mm Hg, P = 0.5) (Table 1). Table 2 gives the AC results in the three groups. The groups differed in CAC, with VAD (0.57 ± 0.46 ml/mm Hg) having lower compliance than both the AD (1.16 ± 0.57 ml/mm Hg) and the control (1.0 ± 0.47 ml/mm Hg) groups, P = 0.01. AI was significantly higher in the subjects with VAD compared to both the AD and the control groups [13.3 ± 9.0 versus 3.5 ± 11.4 and 4.2 ± 9.7 arbitrary compliance units (ACU) respectively, P = 0.03] (Figure 1).

The PWV was not statistically significantly different in the three groups. The VAD group had a PWV in the carotid–radial region of 11.03 ± 1.2 m/s compared to 9.6 ± 1.6 m/s in the AD group and 10.3 ± 1.6 m/s in the controls (P = 0.1). The subjects with VAD also had a PWV in the carotid–femoral region of 13.6 ± 2.6 m/s compared to the AD patients (12.3 ± 2.6 m/s) and the control group with a mean value of 12.6 ± 2.1 m/s (P = 0.41). Recordings of PWV in the femoral–dorsalis pedis region showed the VAD group to have a PWV of 9.5 ± 1.6 m/s, the AD group to have 8.7 ± 1.5 m/s and the controls to have 10.5 ± 1.85 m/s (P = 0.07).

Multiple regression analysis using CAC as the dependent variable and type of dementia (VAD, yes/no; AD, yes/no), gender, BMI, heart rate, smoking status, systolic blood pressure, diastolic blood pressure, smoking status and vasodilator therapy as independent variables, showed that a diagnosis of VAD was the main factor influencing CAC (P = 0.003). On repeating this with AI as the dependent variable, systolic blood pressure (P = 0.007), and diagnosis of VAD (P = 0.034) were the main factors influencing AI.

The regression equations were as follows:

1) \[ \text{CAC} = -0.54\text{VAD} + 1.12 \]
2) \[ \text{AI} = 6.99\text{VAD} + 0.18\text{SBP} - 19.99 \]

where VAD = 1 when present, 0 when absent and SBP was in mm Hg. Variables not included had P > 0.05.
Factors affecting arterial compliance

AC is defined as the change in arterial volume divided by the associated distending pressure. Previous studies have shown that ageing affects arteries differently, with the proximal vasculature being more adversely affected. The increase in systolic blood pressure with ageing resulting from the loss of compliance in large arteries is due to the reduction in elastic fibres and an increase in cross linkage of collagen. Passive AC decreases with increasing mean distending pressure. Therefore, it would be expected that the increase in mean systolic blood pressure in the VAD group compared to the AD subjects would in itself cause a decrease in measured CAC. However, our multiple regression analysis demonstrated that there is a decrease in CAC in VAD subjects above that explained by the associated increase in blood pressure. The percentage of variance is explained by the regression models for the systolic blood pressure and VAD 20%, and 30% for VAD only. Similarly AI was related both to the diagnosis of VAD and systolic blood pressure. The mean blood pressure was lowest in the AD group, consistent with observations that the decline in blood pressures over 75 years of age starts earlier and is more marked in subjects with AD [27, 28]. Although systolic blood pressure was not found to be an independent predictor of CAC, it is possible that the increased mean CAC in AD subjects compared to patients with VAD is partly due to the reduced mean arterial pressure in the former group. The lower pulse pressure in the AD group could account for the differences in the vascular compliance.

It is postulated that serum lipid levels affect endothelial function in resistance vessels, although the effect on conduit vessels remains controversial [29–31]. However, in the three groups, measures of total cholesterol were similar. A progressive increase in heart rate is accompanied by a progressive decrease in AC, due to shortening of the time available for vessel recoil [32]. Although the heart rate was highest in the VAD group, differences between the groups were not statistically significant. Vasoactive drugs that cause vasodilatation increase AC by transferring arterial wall stress from the collagen fibres to the more distensible elastic fibres [33]. However, although the percentage of subjects taking vasodilator therapy tended to be higher in the VAD disease group, six versus five in the AD group and two in the control group, it was not found to be a significant independent factor for AC when included in a multiple regression analysis. This could be explained by the fact

### Table 2. Vascular compliance measurements of the study patients

<table>
<thead>
<tr>
<th></th>
<th>VAD (n = 13)(mean ± SD)</th>
<th>AD (n = 16)(mean ± SD)</th>
<th>Controls (n = 16)(mean ± SD)</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td>CAC (ACU)</td>
<td>0.57 ± 0.5</td>
<td>1.16 ± 0.6**</td>
<td>1.08 ± 0.47**</td>
<td>0.01</td>
</tr>
<tr>
<td>Augmentation index (AI, %)</td>
<td>13.3 ± 8.9</td>
<td>3.5 ± 11.4**</td>
<td>3.5 ± 11.4*</td>
<td>0.03</td>
</tr>
<tr>
<td>PWV (C-R) (m/s)</td>
<td>11.0 ± 1.2</td>
<td>9.6 ± 1.6**</td>
<td>10.3 ± 1.6</td>
<td>0.10</td>
</tr>
<tr>
<td>PWV (C-F) (m/s)</td>
<td>13.6 ± 2.6</td>
<td>12.6 ± 2.6</td>
<td>12.6 ± 2.1</td>
<td>0.41</td>
</tr>
<tr>
<td>PWV (F-DP) (m/s)</td>
<td>9.5 ± 1.6</td>
<td>8.7 ± 1.5</td>
<td>10.45 ± 1.8</td>
<td>0.07</td>
</tr>
</tbody>
</table>

The large artery mechanical properties in the three groups.
VAD, vascular dementia; AD, Alzheimer’s disease; Control, age- and sex-matched controls.
* Significant difference vascular dementia subjects versus control group.
** Significant difference vascular dementia versus Alzheimer’s group.
† PWV in the femoral-dorsalis pedis region was recorded in seven VAD, nine AD patients and all controls.
CAC, central arterial compliance; ACU arbitrary compliance units; PWV (C-R), carotid-radial pulse wave velocity; PWV (C-F), carotid-femoral pulse wave velocity; PWV (F-DP), femoral-dorsalis pedis pulse wave velocity.

### Discussion

The study shows that subjects with VAD have significantly reduced CAC and an increased AI compared to patients with dementia of the Alzheimer’s type and age- and sex-matched controls. If proven in larger studies, these differences in vascular measurements between the two types of dementia can be used in the diagnostic criteria. Mean PWV over the trunk (carotid–femoral) and in the arm (carotid–radial) was also highest in the VAD group, but failed to reach statistical significance. CAC, AI and PWV are measures of arterial function, with AI and PWV increasing and CAC decreasing with increased arterial stiffness. Consistent with the findings of reduced AC is a higher mean systolic blood pressure and pulse pressure in the VAD group compared to the AD group. Arteries in the body stiffen with ageing and measures of arterial stiffness are closely related to age [22, 23]. As the arterial system becomes less compliant, a greater increase in systolic blood pressure must occur to drive the ejected stroke volume in systole. Also, a larger proportion of the stroke volume must flow to the periphery during systole and this, accompanied by diminished elastic recoil during diastole, results in lower diastolic blood pressure and a wider pulse pressure, which also has an impact on the left ventricular function [24, 25].

These findings suggest that the disease process in VAD adversely affects the large elastic arteries and that arterial stiffness in patients with AD is no different from that in controls. PWV has been shown to be a marker of cardiovascular disease in the elderly [26]. The mechanism for reduction in AC in VAD may possibly be more selective for the central elastic arteries than the medium-sized muscular arteries. However, the PWV in the muscular arteries was highest in VAD subjects but did not reach statistical significance.

VAD, vascular dementia; AD, Alzheimer’s disease; Control, age- and sex-matched controls.
* Significant difference vascular dementia subjects versus control group.
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CAC, central arterial compliance; ACU arbitrary compliance units; PWV (C-R), carotid-radial pulse wave velocity; PWV (C-F), carotid-femoral pulse wave velocity; PWV (F-DP), femoral-dorsalis pedis pulse wave velocity.
Dementia and vascular compliance

**Limitations**

The main limitation is that this study was not population based and there are therefore unknown bias in selection. The control population did not have their MMSE measured. The study was small and did not have sufficient power to detect small differences between the groups in PWV over the trunk (carotid–femoral) and in the arm (carotid–radial) and leg (femoral–dorsalis pedis). A shortcoming of the study was the lack of a full complement of PWV measurements in the femoral–dorsalis pedis segments in dementia patients. Only 16 of the 29 patients had this recording. This was primarily a consequence of behavioural disturbances (agitation/restlessness) in dementia subjects causing time constraints on procedures, and leading to difficulty in obtaining satisfactory dorsalis-pedis waveforms in VAD patients with peripheral vascular disease. Nevertheless, the data from this study suggest that AC is reduced in VAD patients. Whether reduced AC is an independent cardiovascular risk factor for VAD and whether treatment to reduce arterial stiffness can improve prognosis still has to be determined by therapeutic studies.

**Conclusions**

This study shows that CAC is significantly lower in patients with VAD. This will need to be confirmed in a larger population-based study. This finding may lead to a therapeutic target. In the heart, reduced AC reduces coronary perfusion [37, 38]. The same may well apply to the cerebral circulation. The pathophysiology of AD and VAD is not very clear. Post-mortem studies show vascular changes in all types of dementia [2]. Although the present study does not provide supporting evidence for vascular disease in AD, modifying vascular stiffness using drugs such as calcium antagonists or hormone replacement therapy or exercise may have a positive effect on VAD. Currently there are a number of anti-cholinesterase inhibitor drugs being used for the treatment of AD. They are also being used in clinical trials in patients with VAD. The vascular effects of these drugs are not clear. Do these drugs modify cerebral perfusion by altering vascular stiffness? The patients who have received calcium antagonists may have a reduced incidence of dementia [39] and it is well known that these drugs have a positive effect on vascular compliance [33]. Do these drugs show a neuroprotective effect due to alteration in vascular stiffness and an increase in cerebral perfusion?

Finally, the role of blood pressure in dementia needs to be addressed. Currently, blood pressure lowering has been shown to reduce cerebrovascular events.

**Key points**

- There are differences in the vascular compliance properties between patients with VAD and patients with AD.
- CAC is lower, and AI is higher in patients with VAD compared to patients with AD.
• The differences in vascular compliance seen in patients with VAD are independent of blood pressure and other cardiovascular risk factors.

Acknowledgements

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Conflicts of Interest

None.

Supplementary data

Supplementary data for this article are available at Age and Ageing online.

References

Optimising recruitment of older people into physical activity study


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Optimising recruitment into a study of physical activity in older people: a randomised controlled trial of different approaches

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Abstract

Background: physical activity studies in older people often have poor recruitment. Including a questionnaire with the invitation would provide information about non-participants and selection bias, but could reduce recruitment. Telephone contact might encourage participation.

Objective: to test the effects of different strategies for recruitment into a study of physical activity in older people.

Design: factorial randomised controlled trial. Randomisation by household into four groups: telephone contact plus questionnaire, telephone contact only, questionnaire only, neither.

Setting: primary care, Oxfordshire, United Kingdom.

Participants: 560 patients ≥ 65 years randomly selected after exclusions.

Interventions: questionnaire to assess health, functional ability and physical activity. Telephone contact by the research nurse a week after sending study information.

Main Outcome Measure: recruitment into physical activity study.

Results: telephone contact increased recruitment: contact 47.9% (134/280), no contact 37.9% (106/280), difference (adjusted for the clustering effect of household) 10.0% (95% CI 0.2–19.8). Questionnaire inclusion did not significantly reduce recruitment: no questionnaire 44.3% (124/280) questionnaire 41.4% (116/280) difference −2.9% (95% CI −12.7–7.0).

Conclusions: telephone contact significantly increased recruitment and should be considered in studies where recruitment may be low. While inclusion of a questionnaire provided valuable information on non-participants and did not significantly reduce recruitment, an adverse recruitment effect could not be excluded.

Keywords: recruitment, response rate, questionnaires, randomised controlled trial, physical activity, older people, elderly