Vitamin D and in-patient falls

SIR—Burleigh and colleagues [1] have shown that vitamin D and calcium in the acute care setting does not reduce in-patient falls. There were clearly however a number of major limitations within the study which the authors discuss in detail and I applaud they recognise these.

A recent BMJ systematic review found no consistent evidence for single or multiple interventions to prevent in-patient falls [2]. Whilst there is better evidence for falls prevention in the community-dwelling elderly [3–5], these findings are unlikely to be generalisable to the acute hospital setting. Up to 30% of in-patient falls occur during the first 48 h of the patients’ stay, and in addition, 75% of in-patient falls occur in the first 2 weeks [6, 7]. Therefore, patients need to be supported during this early phase as the effects of medical treatment, rehabilitation and multifaceted falls assessment measures are undertaken, and it is these measures that are most likely to reduce in-patient falls.

Our hospital audit data has shown that over half the number of in-patient falls occur at the patient’s bedside during transfers or whilst getting up to go to the toilet. In the majority of cases, these falls are unwitnessed. When patients are advised to call for assistance, they are often reluctant to do so, with ‘not wanting to bother the nurses’ or unable to do so because of cognitive impairment/delirium. Continuously observing patients at their bedside may be one method in reducing falls in the early admission period, but this is impracticable in most health care settings. One potential option may be the use of monitoring systems.

A number of monitoring systems are available which alert nearby staff that a person is attempting to leave his or her bed or chair to enable staff in an ordinary ward setting to respond quickly enough to reduce the risk of falling. We conducted a 12-month uncontrolled before (n = 209) and-after (n = 153) feasibility study of a monitoring system, undertaken on an orthogeriatric rehabilitation ward, in patients recovering from an acute hip fracture. The system comprised a bedside chair and bed pressure sensor, incorporating a paging alert mode to alert the ward auxiliary nursing staff. The pre-phase consisted of a 12-month observational phase and the intervention, 12 months. The intervention phase consisted of a bed and bedside chair sensor linked to a pager, wired up to all 18 beds in the orthogeriatric ward. All patients admitted to the ward were included in the study. Bedside falls (defined as the area around the patient’s bedside) were recorded pre-and post-intervention on the ward incident forms, which is a mandatory requirement for all falls incidents that occur in the ward. These forms were then collated by an independent researcher. Mann–Whitney non-parametric tests were used to compare the number of falls and length of stay before and after the intervention phase.

The percentage of falls during the hospital stay was reduced from 14.9 to 8.2% [OR 0.55 (95% CI 0.32, 0.94)], adjusted for age. The mean number of falls per patient (including recurrent fallers) was reduced from 0.14 (0.38) to 0.09 (0.33) (P = 0.032). The mean difference in the length of stay between ‘fallers’ and ‘non-fallers’ was 3.6 days [95% CI 2.1, 9.2 (P = 0.048)]. There was no significant difference in the mean length of stay before and after implementation of the programme. The technology was found to be reliable, with few false alarms, and well received by patients and staff. The limitations of this study and the practicalities of sensors and choice of patients generally [8] are recognised; however, the potential for this type of technology in supporting what is recognised as a major clinical governance issue in most trusts in the UK must not be underestimated. The next phase is a 3-year randomised controlled trial, with both qualitative and quantitative outcomes, which has just been funded by the NIHR-Patient Benefit Programme.

Key points

- Over half the number of in-patient falls occur at the patient’s bedside and the majority are unwitnessed.
- Monitoring systems using bed and bedside chair pressure sensors may reduce bedside falls.
- Reducing bedside falls will reduce the overall length of stay in hospital.
- Further RCTs are necessary before these systems are implemented nationally.

Conflicts of interest

There is no conflict of interest to declare.

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Research letters


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Early parental death and late-life dementia risk: findings from the Cache County Study

SIR—Dementia is a major public health problem. Alzheimer’s disease (AD) comprises the majority of dementia cases, and while the causes are still largely unknown, epidemiological studies are investigating a broad array of both genetic and environmental risk factors. Plausible aetiological mechanisms include lipid metabolism, inflammation and glucose regulation. Emerging biological evidence suggests that another potential mechanism for increased AD risk is neuronal death through the lifelong cumulative effect of stress reactivity and recovery [1]. Repeated stress causes damage to the CA3 region of the hippocampus via glucocorticoids and excitatory amino acid neurotransmitters released during and immediately after stress. Long-term, chronic stress over many years appears to continue the process and result in neuronal death in the hippocampus [2]. Physiological stress responses may also affect health by modulating the rate of cellular ageing via higher oxidative stress, lower telomerase activity and shorter telomere length, all indicators of cell longevity [3] with evidence that neurodegenerative changes may begin decades before clinical manifestation [4, 5].

These potential biological mechanisms, viewed from a lifecourse perspective, are consistent with observations that poor growth and development and adverse environmental conditions in early childhood are associated with increased disease risk in late life [6]. Socioeconomic adversity in the early years of life, specifically father’s occupation of unskilled manual labourer, has been associated with a 2-fold increase in offspring AD risk [7] and faster rate of late-life cognitive decline in offspring [8]. Early-life parental death is an unexpected and traumatic event which usually poses the greatest adaptive challenges [9] thus introducing a host of potential stressors. These are not only economic but also psychosocial and may have a lifelong impact, especially for individuals with inadequate access to social and emotional resources. Though few studies directly address this question, in a sample of elderly Swedes, a 6-fold increase in dementia risk was demonstrated among participants experiencing a parental death before age 16 [10]. Although early-life adversity is not easily modifiable, establishing a clear link between psychosocial adversity and increased vulnerability to AD and other dementias will aid in the identification of at-risk individuals for more targeted interventions. Effective interventions may reduce the intensity of the body’s physiological stress response and vulnerability to neurodegenerative diseases such as AD and other dementias.

We report here analyses of the relationship between early parental death and dementia risk, in a large population-based epidemiological study. The relative effect of parental death at different stages in the lifecourse was examined. We hypothesised that experiencing early parental death would significantly increase dementia risk, with stronger effects posited for parental deaths occurring earlier in the course of development. Analyses were conducted before and after controlling for gender, age, education and apolipoprotein E (APOE) genotype.

Materials and methods

The Cache County Study on Memory Health and Aging is a population-based epidemiological study of dementia and other cognitive impairments, and the genetic and environmental factors that affect risk for these disorders. The original cohort consisted of 5,092 permanent residents of the county (90% participation rate) aged 65 or older in January 1995. Described in detail elsewhere, four successive data collection waves spaced 3–4 years apart implemented a multi-stage dementia ascertainment protocol to identify prevalent [11] then incident [12] cases of dementia. The diagnostic protocol included a neuropsychological test battery, brief neurological examination, neuropsychiatric inventory and semi-structured clinical history interview with informant. Subjects with a provisional diagnosis of dementia, determined by a geropsychiatrist and the clinical team, also completed MRI brain imaging, lab work, and were re-examined by a study physician. Expert panel consensus conferences generated final diagnoses of dementia, according to DSM-III-R criteria. The published sensitivity of the screening protocol to detect prevalent dementia was 0.99 for the 90/91 cut-off score used in the third wave [13]. Informed consent was obtained for each interview, and all procedures were approved by the Institutional Review Boards of Utah State University, Duke University, and the Johns Hopkins University.